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Gait Analysis in Cerebral Palsy

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Martin Švehlík

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THEORETICAL PART

Cerebral Palsy

Cerebral palsy (CP) is a well-recognized and common neurodevelopmental disorder beginning in early childhood. It is a lifelong condition that challenges the individual child, their family and the individual as an adult. The term of cerebral palsy was first used by William J. Little, English orthopedic surgeon working in London, in a series of lectures in 1843 entitled "Deformities of Human Frame". At about the same time, a German orthopedic surgeon Henoch described the hemiplegia in children. Regardless, cerebral palsy was known for many years as "Little Disease' ". In his best known work in 1862 Little differentiated between the congenital deformities, such as talipes equinovarus, and the limb deformities that developed subsequent to preterm, difficult or traumatic births, due to what he termed rigid spasticity [1]. Another famous name connected with research of cerebral palsy is Sigmund Freud. He advocated for classification of cerebral palsy using only clinical findings and was the first one to use the term "diplegia" for all bilateral disorders. Regarding the etiology he identified three groups of causal factors: (1) maternal and idiopathic congenital; (2) perinatal; (3) postnatal causes [2]. In the 1920's Winthrop Phelps, an American orthopaedic surgeon, pioneered modern aproaches to the management of children with cerebral palsy advocating physical therapy, orthoses and nerve blocks [1]. He was one of the founders of the American Academy for Cerebral Palsy in 1947. The first to describe the cerebral palsy in the Czech literature were Havernoch (1897) and Haškovec (1898) [3]. There has been a long history of treatment of children with cerebral palsy at Orthopaedic department for children and adults, 2nd Faculty of Medicine, Charles University, Prague. I would like to mention former chiefs of our department, Professor Otakar Hněvkovský and Assistent Professor Václav Smetana, who dedicated their careers to work with cerebral palsy and handicapped children.

There have been numerous attempts at defining the cerebral palsy. One of the older but still useful definition is the one reported by Bax [4] : "Cerebral palsy is a disorder of movement and posture due to a defect or lesion of immature brain.". This defnition is helpful in descriptive terms, in order to diferentiate cerebral palsy from progressive neurological diseases. However, it may contribute to a misunderstanding of the secondary musculoskeletal pathology, which is not static but is most definitely progressive [5]. The heterogenity of disorders covered by term „Cerebral Palsy", as well as advances in the understanding of infants with early brain demage led Mutch and colleagues to a more recent definition [6] : „Cerebral palsy is an umbrella term covering a group of non-progresive, but often changing, motor impairmant syndromes secondary to lesions or anomalies of the brain arising in the

early stages of its development.“. This definition ephasizes that cerebral palsy is not a disease entity. The cerebral palsy syndromes are heterogenous in terms of primary ethiology and timing of central nervous system (CNS) lesion, primary CNS pathology, clinical features and associated impairments and secondary musculoskeletal pathology. To underline the idea that a comprehensive aproach to cerebral palsy needs to be multidimensional and that management of CP patients requires multidisciplinary setting, disorders commonly accompanying the motor aspects of CP have been identified in the refined definition.

The definition of cerebral palsy [7]

Cerebral palsy describes a group of disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occured in the developing fetal 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.

Incidence of Cerebral Palsy

Cerebral palsy is one of the most common neurodevelopmental disabilities. According to last population studies the world-wide prevalence of cerebral palsy is 2-4 patients per 1000 live births, varying according to the amount and type of prenatal care, the socioeconomic conditions of the parents, the environment, and the type of obstetrical and pediatric care the mother and child receive [8]. In Europe [9] it affects 2 to 3 per 1000 live birhts, as is usuall in developed countries. Low birthweight infants now comprise about 50% of all cases of cerebral palsy; in the early years of the study they comprised about 32% of all cases [9]. Infants with a birth weight of less than 1500 grams are more likely to have cerebral palsy, with a prevalence of 60 per 1000 compared with an overall prevalence of 3 per 1000 infants with normal birth weight [10]. The proportion of cerebral palsy by clinical type has changed among low birthweight babies, with relatively fewer cases with diplegia and a concomitant increase in the proportion with hemiplegia [11]. In developed countries the overall frequency of congenital cerebral palsy has changed little during the last decades. However this masks a dramatic increase in the frequency in the infants born most preterm, a decline in those born moderately preterm and little change in those born at term, but the severity of impairments of those born very preterm is decreasing while for those born at term severity in increasing [12]. These changes may be the result of the increasing ability of perinatal care to rescue very vulnerable infants.

Etiology of Cerebral Palsy

The lesion responsible for cerebral palsy may have its origin in the prenatal, natal, or postnatal period. The prenatal period lasts from conception until the onset of labor, the natal period from the onset of labor until the actual time of delivery, and the postnatal period from the time of delivery until about 2 years of age [13]. According to the European Cerebral Palsy Study [14] brain MRI scans showed that white-matter damage of immaturity, including periventricular leukomalacia (PVL), was the most common finding (42.5%), followed by basal ganglia lesions (12.8%), cortical/subcortical lesions (9.4%), malformations (9.1%), focal infarcts (7.4%), and miscellaneous lesions (7.1%). Only 11.7% of these children had normal MRI findings. CNS pathology associated with cerebral palsy includes: CNS haemorrhage; mechanical spinal-cord or brainstem damage; deep CNS hypoxia; cerebral cortex hypoxia; and transient or irreversible ischaemia resulting in cell necrosis secondary to free-radical formation or hypoxia-related metabolic cellular death [8].

Prenatal causes

Several prenatal causes have been implicated in the development of cerebral palsy, including maternal and pregnancy-specific problems. Intrauterine and TORCH (toxoplasmosis, rubella, cytomegalovirus, and herpes) infections may lead to cerebral palsy, which can be severe in some cases, especially if the infections occurred in the mother during the first and second trimesters of pregnancy. The European Cerebral Palsy Study [14] reported high rate of infections during pregnancy (39.5%). Hypoxia, another cause of cerebral palsy, results in the prenatal period from various causes, including a ruptured placenta or placental infarction. Chemical or alcohol dependency in a mother during pregnancy has been shown to increase the incidence of cerebral palsy [10].

Natal causes

Natal causes for cerebral palsy are trauma or asphyxia occurring during labor. Current medical evidence, however, indicates that labor and delivery events account for a relatively small portion of patients with cerebral palsy . Nelson [15] reported that asphyxia alone accounted for less than 10% of patients with cerebral palsy and that most patients with cerebral palsy had no signs of asphyxia in the perinatal period. Prematurity is the most common natal cause of cerebral palsy. Low birth weight (less than 2500 g) and cerebral palsy have been known to be causally related. Approximately 10% of cerebral palsy patients weigh less than 1500 g at birth. In this low birth weight group, the risk of having cerebral palsy is 60 in 1000 compared with 3 in 1000 in infants weighing more than2400 g or appropriate-for-gestational age

[16]. Even with the increased risk of cerebral palsy in premature infants, prematurity cannot solely be blamed for most of the cases, since 54% of affected children are born at full term [14].

Postnatal causes

Encephalitis and meningitis can lead to a permanent brain injury, resulting in cerebral palsy. Traumatic head injuries, caused by motor vehicle accidents and child abuse, account for a significant number of cases of cerebral palsy that develop in the postnatal period. Cerebral palsy resulting from trauma or associated hemorrhage usually is spastic [10]. In a study of children who suffered brain trauma, Brink and Hoffer related the prognosis for recovery directly to the level and length of unconsciousness after the initial insult [17]. Deep coma for longer than 1 week results in a poor prognosis for any significant recovery. Anoxic encephalopathy from near-drowning creates a hypertonic pattern seen with extreme rigidity [10].

Classification of Cerebral Palsy

Cerebral palsy is historicaly subclassified by nature of the motor disorder and its distribution. Howevwer, classification by distribution applies mainly on the spastic types of CP beause the other motor disorders have mostly total body involvement. Part of the children have mixed pattern. Percentages of the main toporaphical subtypes of CP are listed in Table 1.

	Mutch (1992) (N=502)	European Study (2003) (N=381)
Spastic		
Monoplegia	2	-
Hemiplegia	21	27
Diplegie	22	36
Tetra/quadruplegia	33	21
Ataxic		
Truncal	6	4
Dyskinetic/dystonic	3	12
Mixed/unclassified	13	-

Table 1. Comparison of the percentages of the main topographical cerebral palsy subtypes from two large surveys. Reproduced from Srutton D., Damiano D. Management of the motor disorders of children with cerebral palsy. London: Mac Keith Press; 2004. [18]

Spastic cerebral palsy

Hemiplegia

In hemiplegia the arm appears to be much more involved than the leg. The majority of the children with spastic hemiplegia develop flexion/pronation/adduction deformities of the shoulder, flexion deformity of the elbow and the „thumb in palm“ deformity is very common and is associated with significant functional impairment and equinus in the lower limb. Muscle imbalance is important in genesis of upper-limb deformities [18] p.119. The stronger flexors and pronators tend to overcome less powerful extensors and supinators. All children with hemiplegia walk, although their onset of walking may be delayed. In the lower limbs, spasticity and contractures are more pronounced distally than proximally. Therefore equinus deformity is very common and hip is mostly unaffected. There is a classification of gait patterns in spastic hemiplegia by Winters et al. [19].

Diplegia

The child described as having classical diplegic cerebral palsy has both legs affected. However, partial involvement might be found also in upper-limbs [20] p.78. The position of the upper extremity is usually with internal rotation in shoulders, flexed elbows, wrist and fingers. Children with spastic diplegia walk with slightly flexed and internally rotated hips, semi-flexed knees, extended plantar-flexed ankles. However, the gait pattern of children with cerebral palsy may be highly variable because of mixture of spasticity, contractures and muscle weakness. During the growth some of these dynamic contractures may develop to fixed, what has important clinical consequences. The classification of gait patterns based on kinematics of gait in spastic diplegia was introduced by Rodda et al. [21].

Quadriplegia

This is the most severe form of spastic cerebral palsy. It is characterised by bilateral spasticity with upper-limbs more involved than lower-extremities. Quadruplegia is frequently associated with seizure, cognitive impairment and severe mental retardation and microcephaly [20] p.79. The children develop no (or very limited) functional movement and they are at great risk of developing contractures and deformities. These children are unable to walk.

Dystonic cerebral palsy

Formely also called extrapyramidal or athetoid cerebral palsy. Dystonic cerebral palsy is defined to be a motor disorder associated with abnormal movements or posture based on the dysregulation of muscle tone and coordination [22]. It has few signs in the early months of life and the abnormal movements or muscle tone develop at the end of the first year of life. This form often develops from hypotonic

syndrom [20] p.81. There are unwanted movements around the mouth and of the arms and legs, and these become particularly prominent when attempting fine or gross motor movements. Speech is always problem and bulbar disorders (swallowing difficulties, nutrition and drooling) may be a major problem [18] p.14.

Ataxia

This is a much less common diorder. The infant with ataxic cerebral palsy presents as floppy baby, opposite to spasticity. There are increased ranges of motion at all joints. Volitional movements are affected usually all over the body. Postural development is delayed, as is walking. Children with ataxic cerebral palsy has unsteady, wide-base stamping gait and often gross intentional tremor in the arms and hands [18] p.14.

Mechanisms of deformity

At birth, children with cerebral palsy do not have muscle contractures, bony torsion or hip dislocation. These deformities are aquired during childhood. When looking at the pathological gait, it is important to remember that what we see is always combination of cause and effect. We can distinguish primary, secondary and tertiary abnormalities of gait [23] pp.180-204. Primary abnormalities are those which occur at the moment of the brain injury and are a direct efect of this injury. They are permanent and they can not be corrected. They are represented by loss of selective control of muscles, balance difficulties and abnormal muscle tone (mostly spasticity). In normally developing children, muscles and bones growth proportionally. In 1984 Ziv et al. [24] showed that muscle growth takes place at the musculotendinous junction, which they called muscle growth-plate and that the stimulus to longitudinal growth of muscle is stretch. In spastic mice they found that the rate of growth was reduced by 45%, which resulted in contractures. It has been also reported that muscle belly becomes smaller in proportion to the muscle tendon over the time [25]. In simple words, growing bones and muscles follow the „Star Wars Principle“ popularized by professor Gage: „May the force be with you!“ [23] p.180. Because the primary effects of the brain injury impose abnormal forces on the skeleton, neither bone nor muscle grows normally. These changes we refer as the secondary abnormalities [23] p.180. Because muscles and bones grow gradually, these seconday abnormalities do not occur immediatelly after brain injury but in a direct proportion to skeletal growth. Even if we have certain mixture of primary and secondary abnormalities there are allways several possibilities how to cope with them. And any of these coping mechanisms may interfere with normal walking and therefore we refer to these „coping mechanisms“ as tertiary gait abnormalities. Pathological gait is then a mixture of primary, secondary and

tertiary abnormalities. It is highly important to distinguish between these different abnormalities, because we are not able to change the first ones and the third ones dissapear spontaneously if we treat secondary abnormalities correctly. And the gait analysis my help us to discriminate one from the other. So the basic general principal to treat the gait problems in children with cerebral palsy is best expressed by Reinhold Neibuhr:

„God, give us grace to accept with serenity the things that cannot be changed, courage to change the things which should be changed and the wisdom to distinguish the one from the other."

Spasticity

Spasticity is one of the most serious problems in patients who have upper motor lesion in the brain or spinal cord. It was defined by Lance in 1980: „Spasticity is a motor disorder characterized by a velocity increase in tonic stretch reflexes, with exagerated tendon jerks resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome". As the definition says, spasticity is only one of the positive features of the upper motoneuron syndrome. Other positive features are clonus, hyperreflexia and co-contraction. Clinicians tend to concentrate to these positive features and omit the negative ones (weakness, loss of selective motor control, sensory and balance deficits). Nevertheless, these negative features of the upper motoneuron syndrome are important for prognosis of a child with cerebral palsy. Amongst these factors, muscle weakness is significant in limiting walking ability and motor function [26]. In the past, spasticity has been considered the major obstacle to motor function. However, advances in the management of spasticity have demonstrated that muscle weakness limits functional improvement [27]. Furthermore, orthopaedic surgery involving muscle-tendon lengthening procedures is known to reduce muscle strength [28], particularly in the presence of pre-existing weakness. Nevertheless, strength gains in children with spastic cerebral palsy can be achieved through strength training [29]. In contrast to healthy subjects it was shown in children with cerebral palsy [30;31] that major reason for increased energy cost of locomotion is ineffective gait due to simultaneous activation of agonist and antagonist muscles, i.e. co-contraction, which is therefore another important positive feature of the upper motoneuron syndrome. Both positive and negative features of upper motor neuron syndrome are displayed in Figure 1.

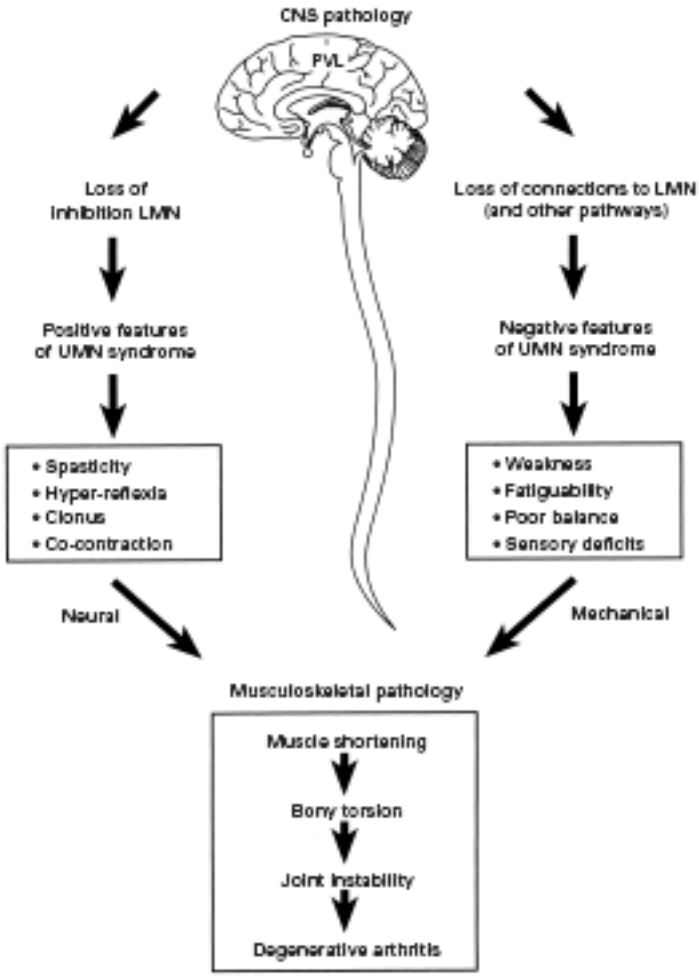

Figure 1. Diagram showing the neuromusculoskeletal pathology in cerebral palsy. Both positive and negative features of upper motor neuron syndrome together with their influence on musculoskeletal pahtology are displayed. Reproduced from Graham and Selber: Musculoskeletal aspects of cerebral palsy, J Bone Joint Surg [Br] 2003;85-B:157-66 [32].

Treatment options of children with cerebral palsy

When treating a child with cerebral palsy the goal is not to treat the cerebral palsy itself, but to improve some area of function and/or the quality of life. These goals should be clearly stated before the treatment. The ability to walk still remains the most significant goal for most parents with respect to their child with cerebral palsy [13]. However, going beyond this to help the child improve, develop and acquire new skills in all areas of development, is also important. Because of the complexity of the problems in children with cerebral palsy, the team taking care of these children should be

interdisciplinary to determine the best treatment and optimize outcomes. Usually, the team consists of the neurologist, physiotherapist, orthopaedic surgeon, pediatrician, orthotist to name just the most important subspetialities. The treatment option is influenced by several factors, like the patient's age, size, functional status, present or risk of future musculoskeletal deformities, developmental potential and mental status. All these have an impact on treatment decision-making process. Although children with cerebral palsy have altered development because of their neurologic abnormality, they will still make developmental gains due to the brain maturation. Rosenbaum et al. described 5 distinct motor development curves [33]. These describe important and significant differences in the rates and limits of gross motor development among children with cerebral palsy by severity. However, even if a certain gross motor development status was achieved, the functional status may be adversely affected by growth. Adolescents are particular susceptible to these changes during pubescent growth and fuctional deterioration si common at that time [34;35]. Most children with cerebral palsy do not have just one specific treatment modality for their spasticity, but rather benefit from variety of treatments. Traditionally, treatment adopted „pyramid" aproach (Figure 2) in which the most conservative measures were initiated first and more aggresive measures used when the more conservative treatment failed [23] p.251.

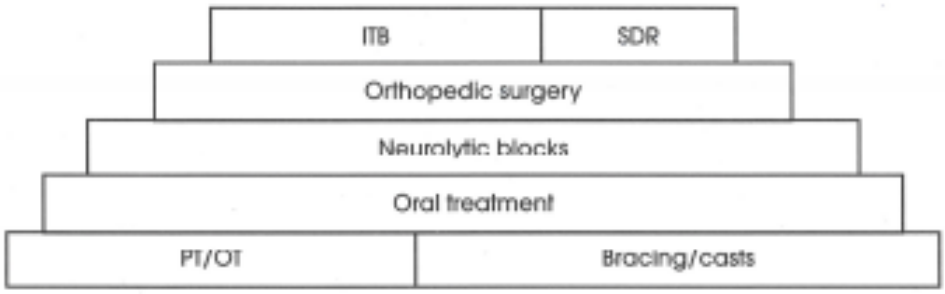

Figure 2 Ilustration of the traditional „pyramid" approach to the treatment of cerebral palsy.

A variety of treatment modalities have been used and investigated for management of cerebral palsy and spasticity. They include traditional physiotherapy (with a range of motion-stretching exercises and splints or orthotics), oral medications, neurolytic blocking agents (such as phenol or botulinum toxin A (BTX-A)), orthopaedic surgery (in particular tendon-lengthening procedures), intrathecal baclofen (ITB) pumps and selective dorsal rhizotomy (SDR). Intervention and rehabilitation for spasticity and related musculoskeletal effects in cerebral palsy should commence early and onces started it is a lifelong lasting process. As I mentioned before in the text, the goals of the therapy should be stated before the

treatment and re-evaluated whenever the goal is reached or the child does not progress futher. The overview of the treatment modalities in children with cerebral palsy is to be found in Table 2.

	Type of tone problem	Age	Advantage	Disadvantage	Cost
Physiotherapy	All types	Any	Improves development	Expensive	$$$
Oral medication	Diffuse	>1 year	Works systematically	Sedating	$
Bracing/Casts	All types	Any	Improves joint position and range of motion	Fails to reduce deformity	$
Orthopaedic surgery	All types	5 years to adolescent	Corrects alignment	Temporarily reduces deformity	$$$
Neurolytic blocks	Focal	Any	Reduces spasticity	Temporary	$$
SDR	Diplegia	4-8 years	Eliminates spasticity	Irreversible	$$$$
ITB	Lower extremity	>17kg of bodyweight	Adjustable tone reduction	20% of adverse effects	$$$$

Table 2: Treatment options for cerebral palsy: indications, advantages/disadvantages, and relative costs. Reproduced from Gage JR.: The treatment problems in cerebral palsy. London: Mac Keith Press, 2004, p.250 [23].

Physiotherapy

Physiotherapy plays an essential role in the management of cerebral palsy. It is the first therapy to be descibed. The intervention ranges from passive muscle stretching to increase the range of motion to specific neurodevelopmental concepts based on understanding of neuroscience and child motor development. The most common aproaches are Neurodevelopmental treatment introduced by Karel and Berta Bobath and the Method of Vojta's reflexed locomotion. To maintain the functional level of a child with cerebral palsy, muscle strengthening is an important issue. It is a well documented fact that children with cerebral palsy have a significant weakness of lower extremity muscles in comparisson to normally developing children [26]. Damiano and Abel found an improvement in gait and gross motor function following 6 weeks of isotonic muscle strengthening in ambulant children with cerebral palsy [29].

Moreover, the effect of 6 weeks of strength training may be as high 140% in children with cerebral palsy [36]. Orthoses are designed to provide joint stability, to hold a joint in a functional position and to keep tight muscles stretched. The biomechanical prerequisites of the orthotic design need to be considered in order to address the gait problem. Serial casting is used to lengthen already shortened muscles and soft tissues and to prevent or correct contractures. Although some of the studies showed positive effect of 6 weeks casting comaprable with BTX-A [37], it might have deletorious weakening effect on agonist as well as antagonist muscles. However, serial casting might prolong the possitive effect of BTX-A injections [38].

Oral medication and neurolytic blocks

Oral antispasticity agents are usually of relevance to individuals with generalized or diffuse muscle spasticity, such as spastic quadriplegia, rather than to individuals with focal spasticity. These medication exert their effect by inhibiting excitatory neurotransmiters or augmenting inhibitory neurotransmiters at the level of spinal cord. Unfortunately, these medications are not selective for the spine, and the neurotransmiters changes in the brain may produce sedation prior to spasticity modulation [39]. For oral distribution Baclofen (presinaptically inhibits release of excitatory neurotransmiters in the spinal cord), benzodiazepines (augment GABA-mediated inhibition in the spinal cord and supraspinally), Dantrolene (inhibits release of calcium from sarcoplasmatic reticulum in the muscle) and alpha2adrenergic agonists (presynaptically inhibits release of excitatory neurotransmiters in the spinal cord and supraspinally).

Botulinum toxin type A (BTX-A) injections has been used clinically for more than 20 years. BTX-A induces muscle weakness by preventing the release of acetylcholine from the presynaptic axon at the motor endplate [40]. The degree of weakening depends on the dose of BTX-A and on the number of synapses affected [41]. Spasticity reduction due to the effects of BTX-A injections typically lasts from 12 to 16 weeks. Re-innervation takes place by sprouting of new nerve terminals, a process that peaks at 60 days in humans [42]. Functional benefits may last for up to 6 months or even longer [43]. Beside the effect of muscle weakening, BTX-A was proved to influence longitudinal growth of an injected muscle in an animal model [24]. Based on computer simulation and modeling the same effect was confirmed in humans 4 weeks after the BTX-A application [44]. Similarily to orthopaedic procedures, there is a trend for multilevel BTX-A application [38].Moreover, the BTX-A treatment was proved to delay and reduce the frequency of surgical procedures [45].

Surgical treatment

Selective dorsal rhizotomy (SDR) has been used to reduce spasticity and improve function in ambulant children with spastic diplegia [46] and to ease care of children with spastic quadriplegia [47]. The rationale of SDR is consistent with neurophysiological evidence that spasticity is the result of decreased inhibition from upper motor neuron corticospinal tracts and interneuron inputs. The procedure is most safely performed by using intraoperative nerve root stimulation and EMG with the cutting of the least number of nerve rootlets which will bring about an effective reduction of spasticity. Outcomes of SDR are generally favorable. Children with cerebral palsy sustain permanent reduction in muscle tone of lower extremity, increased range of motion and also gait improvement [48]. However, the proper indication is crutial for a good outcome.

Baclofen has been used extensively as an oral agent, but problems with the blood–brain barrier and side effects at the amounts necessary for efficacy have resulted in limited clinical utility. Using a programmable implanted pump, baclofen can be delivered intrathecally to the target tissue at reasonable dosages, avoiding systemic side effects [49]. Intrathecal baclofen is both adjustable and reversible. Appropriate candidate for intrathecal baclofen are those with severe generalized spasticity that interferes with function or impairs the ability of caregivers to help the patient [50]. Continuous intrathecal baclofen infusion decreases spasticity in the upper and lower extremities, and reduces clonus and muscle spasms [49]. The high cost of the pump remains problematic and therefore its use in the Czech Republic is very limited.

Orthopaedic surgery

Orthopaedic interventions remain a critical element in the management of children with cerebral palsy. Because of the spasticity, certain muscle groups function in an abnormal fashion including prolongation of their phase of activity into a portion of the gait cycle when they should otherwise be silent (dysphasic activity) and an increase in the strength of contraction during their active phase, thereby overpowering their antagonists (excessive activity) [51]. If such a muscle imbalance persists, spastic muscles may develop contractures over time, and limitation of joint motion together with bony deformities is the results. Therefore reduction of muscle strength of these dysphasic and overactive muscles by surgical release of their origins or re-establishing of balance by lengthening of tendons provides functional improvement during gait. Overall, the aims of surgery are to reduce established deformity, improve cosmesis, improve gait pattern and reduce the energy cost of walking. The muscles which are most frequently addressed surgically tend to be those muscles which cross two joints and work for a

significant portion of their activity in an eccentric mode [52]. Those muscles are hip adductors, hip flexors, hamstrings, rectus femoris and triceps surae. The selection, timing and procedural use of orthopaedic surgery has changed significantly in recent years. In the lower limb there is a definite trend to avoid, or at least to delay, surgery until a child's gait has matured in late childhood. With introduction of multilevel application of BTX-A, the age of the first surgery has been proved to be postponed [45]. Another important change over the pas 20 years is a steady trend towards single-event multilevel surgery (SEMLS) in place of the 'birthday syndrome' when children come in for their annual single-staged surgical procedure [52]. The introduction of gait analysis into the pre-operative planning of surgical interventions in children with cerebral palsy means a great step forward to evidence-based medicine and better understanding of pathobiomechanics of gait and muscles' function. Thanks to gait analysis a new surgical method, rectus femoris transfer to treat the stiff-knee gait and improve the knee function, was introduced and widely accepted [53]. It has been proved that surgical intervention, guided by preoperative gait analysis, is effective and safe for children with cerebral palsy [54]. It is beyond the scope of the present work to discuss all the indications, limitations and timing of different surgical procedures. The reader may find the clinical and gait analysis criteria of the most frequent surgical procedures in children with cerebral palsy in the Table 3 or more detailed information might be found in the paper of Davids et al. [55].

Procedure	Clinical criteria	Gait analysis criteria
Intrapelvic psoas lengthening	Fixed flexion deformity of >15°	Double bump pattern seen on sagittal plane pelvis kinematics
Adductor lengthening	Passive hip abduction after hamstring lengthening of less than 30°	-
Medial hamstring lengthening	Increased popliteal angle/fixed flexion deformity under anesthesia	Decreased knee flexion at initial contact/terminal swing
Lateral hamstring lengthening	Increased popliteal angle/fixed flexion deformity under anesthesia persisting	Decreased knee flexion at initial contact/terminal swing
Distal rectus transfer	Positive Duncan-Ely Test	Inadequate knee flexion in swing
Gastro-soleus lengthening	Equinus deformity not correctable under anesthesia	Equinus at initial contact and in stance, reversal of slope of ankle moments, energy generation in mid-stance
Foot tendon lengthening/transfers	Varus/valgus deformity seen during observational gait analysis	-
Tibia derotation osteotomy	Bony rotational deformity of more than 10°	Persistent internal/external rotation throughout the gait cycle
Femur derotation osteotomy	Bony rotational deformity of more than 10°	Persistent internal/external rotation throughout the gait cycle

Table 3. Indication criteria for the most frequent surgical procedures in management of children with cerebral palsy, based on the clinical and gait analysis examination. Reproduced from Saraph V. et al.: Multilevel Surgery in Spastic Diplegia: Evaluation by Physical Examination and Gait Analysis in 25 Children. Journal of Pediatric Orthopaedics (2002) 22:150–157

Gait

Locomotion is a feature of all animals. Quadrupeds are inherently fast and stable. Their centre of mass is located inside of base of support. A long stride is possible, because the body is interposed between the front and back limbs. Humans employ bipedal gait that is less stable, because the centre of mass is located above the base of support, just in front of the S2 vertebra. However, bipedal gait has the significant advantage of freeing the upper extremities.

Gait can be defined as a method of locomotion characterized by periods of loading and unloading of the limbs [56]. Another definition of gait is that of Perry: „Walking uses repetitious sequence of limbs motion to move the body forward while simultaneously maintaining the stance stability.“ [57]. Gait is a very complex activity. It requieres a coordination of central nervous and musculoskeletal system, therefore many disorders of these systems result in significant interference with gait which make it difficult to participate in normal human activities. One of the basic purposes of clinical gait analysis is to define these difficulties and to suggest remedial intervention.

Normal gait has four basic prerequisities that were defined by Perry [58] and the fifth one was added by Gage [23]. These are, in order of importance:

1) Stability in stance – is a big challenge because the centre of mass is situated high above the base of support. This means that body must constantly alter the position of the segments in space in order to maintatin ballance over the base of support which is changing while walking. This is also called dynamic stability.

2) Sufficient foot clearance during swing – this is a function of balance on the stance side and sufficient ankle dorsiflexion on the swing side

3) Appropriate swing phase pre-positioning of the foot – very important for the weight acceptance

4) Adequate step length – demands sufficient stability on the stance side and adequate hip flexion and knee extension on the swing side

5) Energy conservation - is a typical aspect of walking. Oxygen consumption gives an objective view of the overall efficacy of the patient’s gait. Many factors influence energy consumption, including spasticity, bony deformity, strength and selective motor control. Thus energy efficiency reflects the cumulative effect of many factors [59]. It is a functional tool because its interpretation provides an

indication of endurance, fatigue and ability to accomplish the routine daily task of locomotion [23]. Children with cerebral palsy have been shown to expend greater energy during walking than their typically developing peers when walking at self-selected economical [60] as well as at a given speed [61]. Energy conservation is also accomplished by minimizing the displacement of the centre of mass while progressing forward in a steady, minimal, wavelike pattern [59].

The Gait Cycle

Walking invovles repetitious patterns of movement resulting in each foot periodically moving from one position of support to the next. Because of its cyclic nature, human gait is conventionally described in terms of gait cycle (Figure 3). A complete gait cycle or stride begins when foot strikes the ground and ends when the same foot strikes the ground again and consists of two steps.

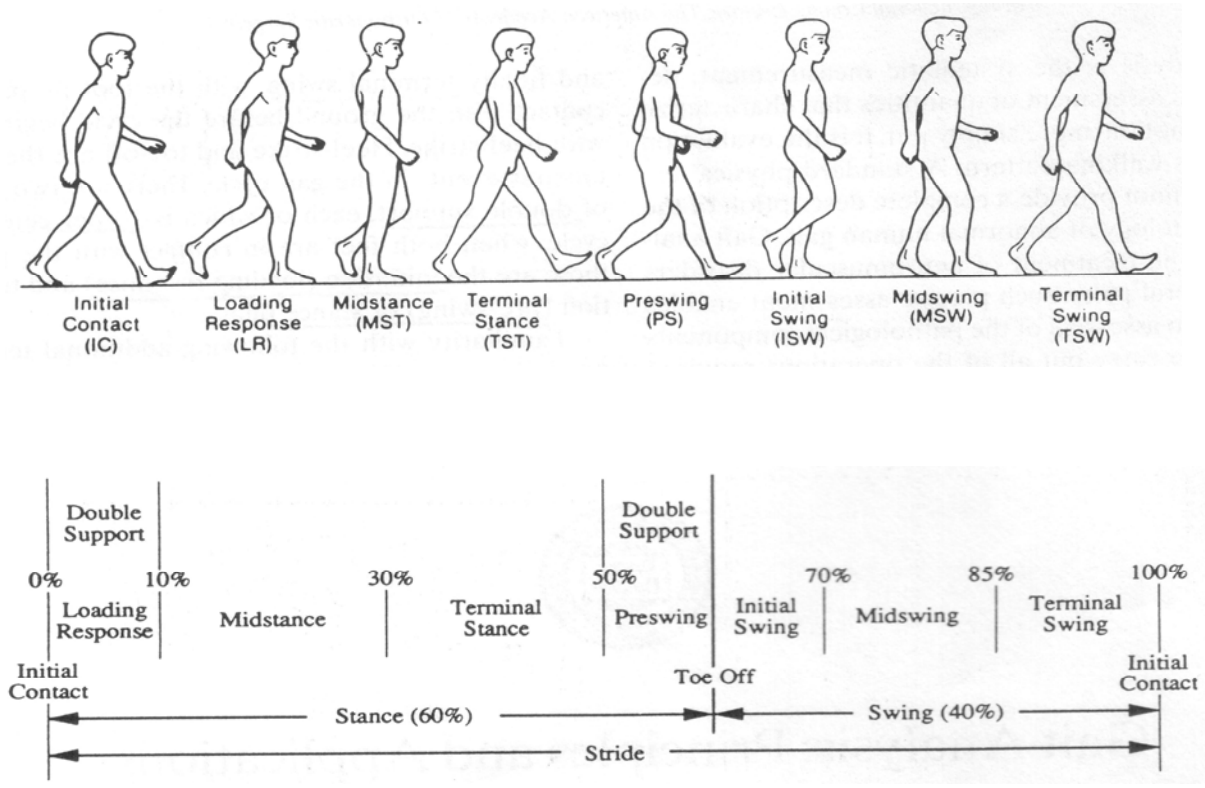

Figure 3. Graphical presentation and timing of gait cycle events and phases. Reprinted from Rehabilitation medicine: Principles and practice. 1998; pp. 167-187, Philadelphia: Lippincott-Raven Publishers

The gait cycle is devided into two major phases, stance and swing phase. Stance phase is the period of time when the foot is in contact with the ground and begins with Initial contact (IC). Swing phase is defined as the period of time when the limb is advancing forward and the foot is not in contact with the ground. It begins with the Toe-off (TO). Usually, we normalize the period of time for a complete gait cycle as 100%. Initial contact occurs at 0% and Toe-off at approximatelly 60%. Therefore, stance phase represents 60% and swing phase 40% of the gait cycle. If we take a gait cycle of a particular lower limb, then during that gait cycle contralateral toe-off occurs at 10% and contralateral initial contact occurs at 50%. This means that during stance phase there are two double support phases each having 10% of gait cycle. The first one is often reffered as loading response (LR) and occurs just after the initial contact. The second one is situated at the end of the stance phase and is called preswing (PSw). In between those two double support pahses, there is a single support phase which can be subdevided into the midstance (MSt) and terminal stance (TSt). Swing is generally devided into three phases: initial swing (ISw), midswing (MSw) and terminal swing (TSw). Three important tasks must be accomplished during the gait cycle: weight acceptance, single limb support and limb advancement (Figure 4). Weight aceptance occurs during the first two periods (IC and LR), single limb support during the second ones (MSt and TSt) and limb advancement during the final four (PSw, ISw, MSw and TSw). Running is differentiated from walking by the fact that two periods of double support are replaced by periods of „double float“ when neither foot is on the ground. The terminology and description of the gait cycle is based on the work of Perry [57](pp.1-19).

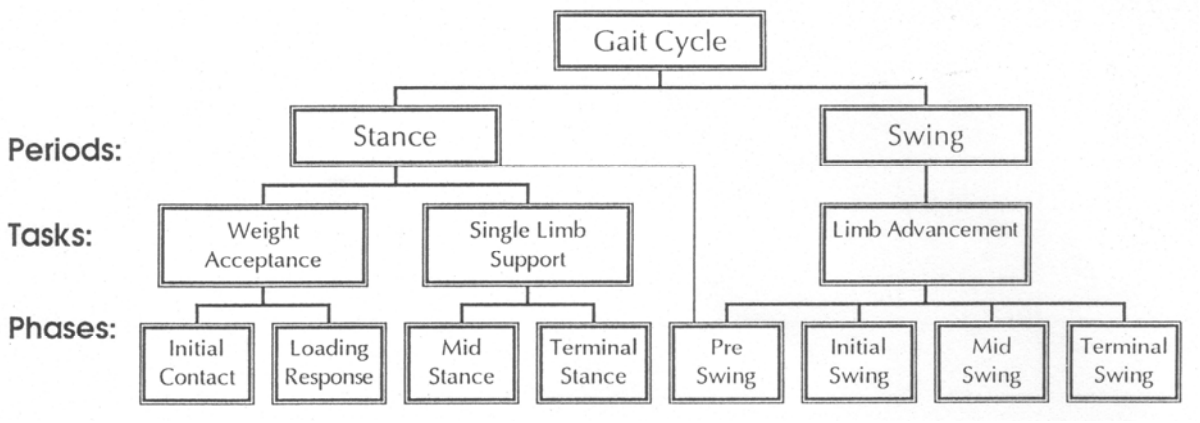

Figure 4. Diagram showing tasks and related phases of the gait cycle. Reproduced from: DeLisa A., Rehabilitation medicine: Principles and practice. 1998; pp.167-187, Philadelphia: Lippincott-Raven Publishers

Initial Contact

The heel is the first part of the lower limb to come into contact with the floor. The hip is in maximal flexion, about 30°, knee is extended and ankle in neutral position. The principal function of the IC is to absorb and accept the weight of the body and insure the stability while the body continuous to move forward. Concentric activity of musculus gluteus maximus and musculi ischiocrurales is important to maintain the stability and the excentric activity of tibialis anterior to slow down the body (Figure 6).

Loading Response

Weight acceptance and stability insurence is a common feature of the IC and LR. The LR is equal to the first double support phase of the gait cycle. The hip is slowly extending and ground reaction force (GRF) is situated in front of the hip a behind the knee. Therefore the hip extensors and musculus quadriceps have to be active to maintain the upright posture. Gradual plantarflexion controled by excentric activity of tiabialis anterior takes pace at the ankle. This movement is called the „first rocker“ and toether with slight knee flexion are important to cushion the body weight during the loading response (Figure 5).

Mid-Stance

During the Mid Stance the stance lower limb is fully loaded because the contralateral limb is swinging. The pelvis is shifted to the stance limb thanks to the concentric activity of hip abductors. The hip flexion decreases and reaches the neutral position. The activity of hip extensors follow the same pattern. The GRF is situated in front of the knee which makes it extend and stretches the posterior part of the knee capsule. The ankle is dorsiflexing while the foot is plantigrade. The shank is rolling over the stationary foot. This ankle motion is called the „second rocker“.

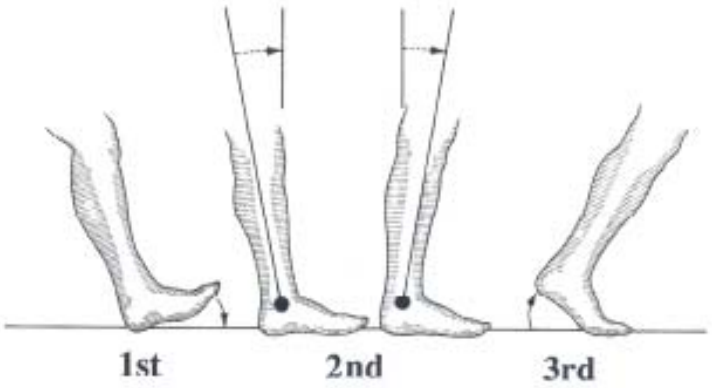

Figure 5. Foot rockers during the stance phase of the gait cycle. Reproduced from Gage, J.R. *The treatement of gait problems in cerebral palsy*. London: Mac Keith Press; 2004. [23]

Terminal Stance

In the Terminal stance, the body is propelling forward over the stance leg. The hip extension is controlled by iliofemoral ligament. The maximal extension is reached when the opposite foot is in IC. Here begins the second double support. The knee is in extension and the ankle continues to dorsiflex until the heel leaves the floor. At this phase musculus gastrocnemius is active to stabilize the knee against the hyperextending forces. This is known as „plantar flexion/knee extension couple“ [62].

Pre-Swing

The aim of this phase is to prepare the lower extremity to swing. The concentric activity of the musculus adductor longus, iliopsoas and rectus femoris makes the hip joint to start flecting. The passive flexion of the knee joint is controled by excentric activity of the musculus rectus femoris. The rapid plantarflexion of the ankle joint is called „push-off“ and this is the most important propulsive power for forward motion of the whole body [63].

Initial Swing

The role of the initial swing is to propel the swinging limb forward. The hip joint carry on flecting and reaches its maximal acceleration, mainly due to the hip flexors concentric activity. The knee joint is acting as a pendulum. The ankle extensors, namely the musculus tibialis anterior, are contracting and the foot goes back to neutral position.

Mid-Swing

The lower limb advances in flexion. The hip joint reaches its maximal flexion of about 20° thanks to the iliopsoas concentric activity. The knee acts still as compound pendulum and reaches its maximal flexion when passing by the opposite stance leg. The ankle is kept in neutral position.

Terminal Swing

During the Terminal Swing it is imporant to ensure the stability of the hip and knee joints before the Initial Contact. Therefore the hamstrings are contracting to terminate the hip flexion and controle the knee extension. The tibialis anterior muscle is active to maintain the neutral position of the foot during the Initial Contact.

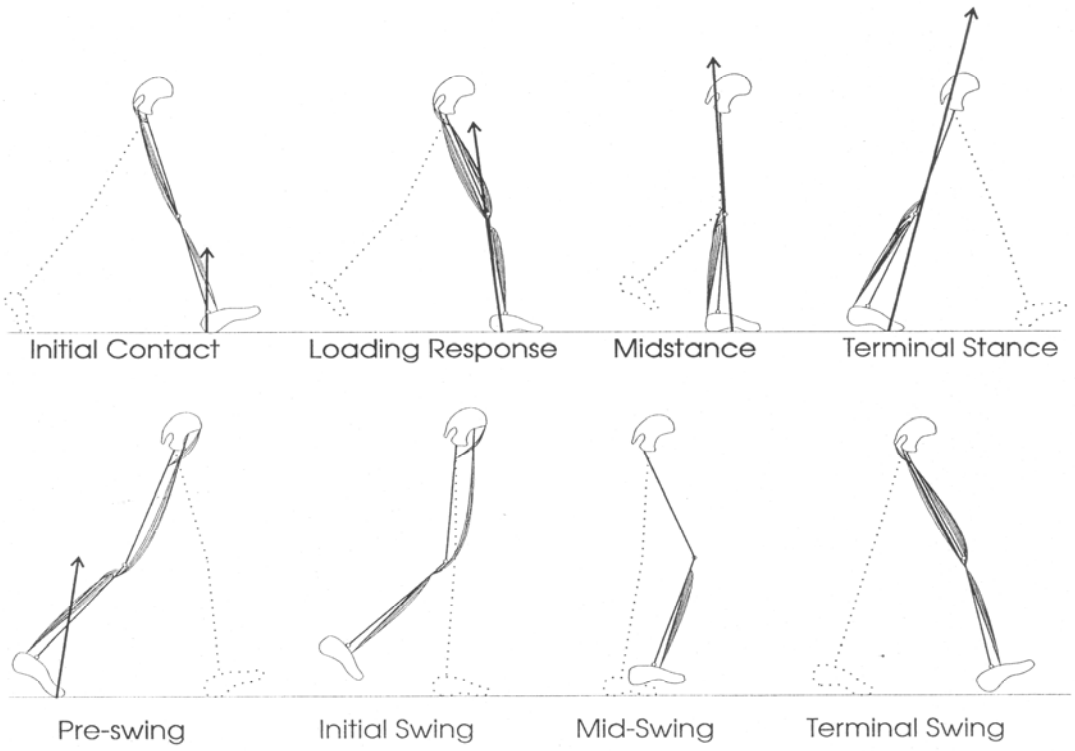

Figure 6. Graphical representation of muscle activity in particular phases of the gait cycle. Reprinted from DeLisa A., Rehabilitation medicine: Principles and practice. 1998;167-187, Philadelphia: Lippincott-Raven Publishers

Gait analysis

Human movement analysis aims at gathering quantitative information about the mechanics of musculoskeletal system during the execution of motor tasks [64], more simply put it is the evaluation of a subject's walking pattern. A standard physical examination cannot provide a complete description of abnormal human gait. Gait analysis can [65]. Gait analysis data cannot be sufficiently predicted by a combination of clinical measurements. This fact supports also the study by Deslovere et al. which has proved only moderate correlation between the physical examination and gait analysis [66]. There are several important components of gait analysis: kinematics, kinetics, electromyography, energy expenditure and clinical observation. The summary scheme of gait analysis is to be seen in Figure 7.

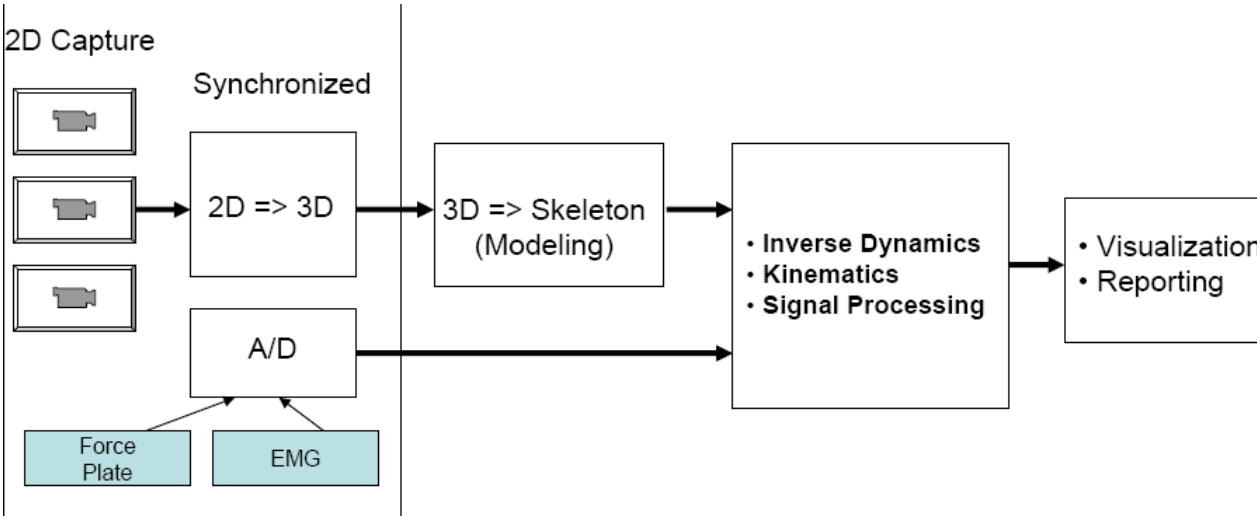

Figure 7. Scheme of the gait analysis methodology.

Kinematics

Gait is a dynamic activity. In order to understand gait we need to understand dynamics, which is the study of objects in motion and factors that affects these motions. These factors include force, mass, momentum and energy. Kinematics describes spatial movement of the body without the regards to its cause. The movements are linear and angular displacements, velocities and accelerations [65].

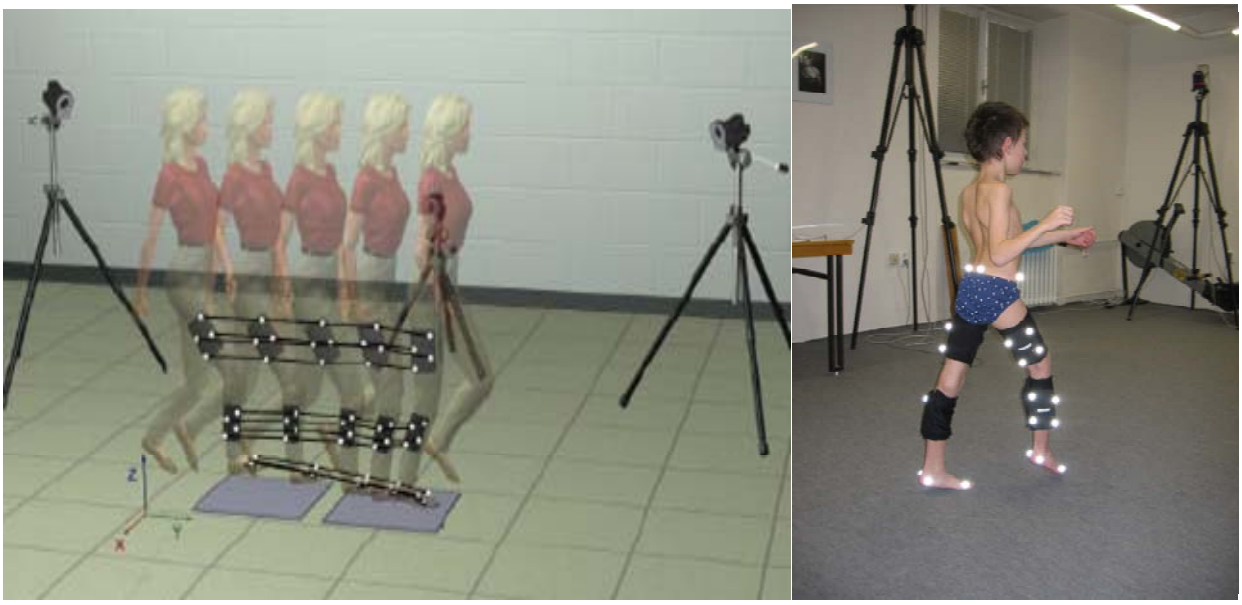

Figure 8. A drawing and photograph of a child walking through the calibrated volume during the gait analysis. Three dimensional marker tracking is graphically demonstrated.

Passive reflective markers are placed on the surface of the patient's lower extremities and aligned with specific bony landmarks. As the patient walks in the laboratory, the location of the markers are monitored with a three-dimensional motion data capturing system comprising of several cameras emitting and receiving infra-red light. These systems work in a similar way to how our eyes provide a 3D binocular vision. If the marker is visible for 2 or more cameras, it is possible to reconstruct its position in the room. The mathematics involved in the reconstruction of 3D trajectory is highly sophisticated and fundamentally dependent on the calibration of the volume [67]. Accuracy of tracing markers is typically around ±0,1% of the capture volume. Since the length of the capture volume in most laboratories is around 5m, this is an equivalent to about ±5mm of measurement error [68]. In practice, however, the major limitation on accuracy is model used for deriving joint motion from skin mounted markers [69]. Defining the position of a point in volume requires three co-ordinates system (x,y,z). Any individual point on a segment may be defined this way. Human movement analysis begins by dividing the body into a series of segments which are assumed to behave as a rigid body. In order to locate the position and orientation of a whole segment three individual points (markers) are required. Two points define axis of the segment and its orientation. The third point is required to describe rotations [69]. This set of three markers has not to be collinear.

There are three different types of coordinate systems used to derive kinematics (Figure 9). The global coordinate system is fixed to the lab space and generally orientated along the walking path. A technical

coordinate system is fixed to a body segment but is not aligned with anatomy. Technical coordinate systems are derived from observed marker positions, and as such they are sometimes referred to as marker-based coordinate systems. Anatomical coordinate system are attached to body segments, and aligned with the principal anatomical directions (sagittal, coronal and transverse) [23] (p.100).

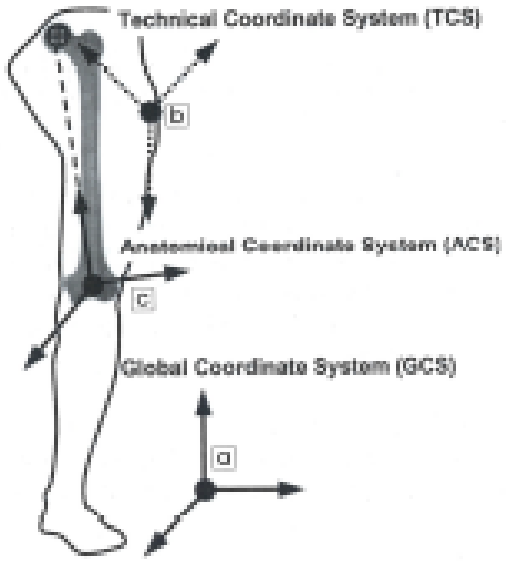

Figure 9. Graphical illustration of the three coordinate systems. Reproduced from **Gage, J.R. *The treatement of gait problems in cerebral palsy*. London: Mac Keith Press; 2004. [23]**

We have determined that 3 markers are required to fix the position and orientation of a segment in the space. A joint, in terms of mechanics, can be defined between any two segments, and it is the interrelationship of these segments which results in the kinematic graphs. It is fundamental that joints do not move. Body segments adjacent to joints move and the relative motion of these segments is termed joint rotation. In clinical gait analysis, it is common to report this relative motion in terms of Euler angles. Euler angles are one method to describe the position of one segment relative to adjacent body segment. An important aspect is the numerical value of the angles depending on the sequence of rotations. The sequence arbitrary used in gait analysis is sagittal plane, followed by coronal plane followed by transversal plane [23] (p.102). The graphical illustration of normal joint kinematics over the gait cycle at the pelvis, hip, knee and ankle in all three planes for the pediatric population may be found on the Figure 10.

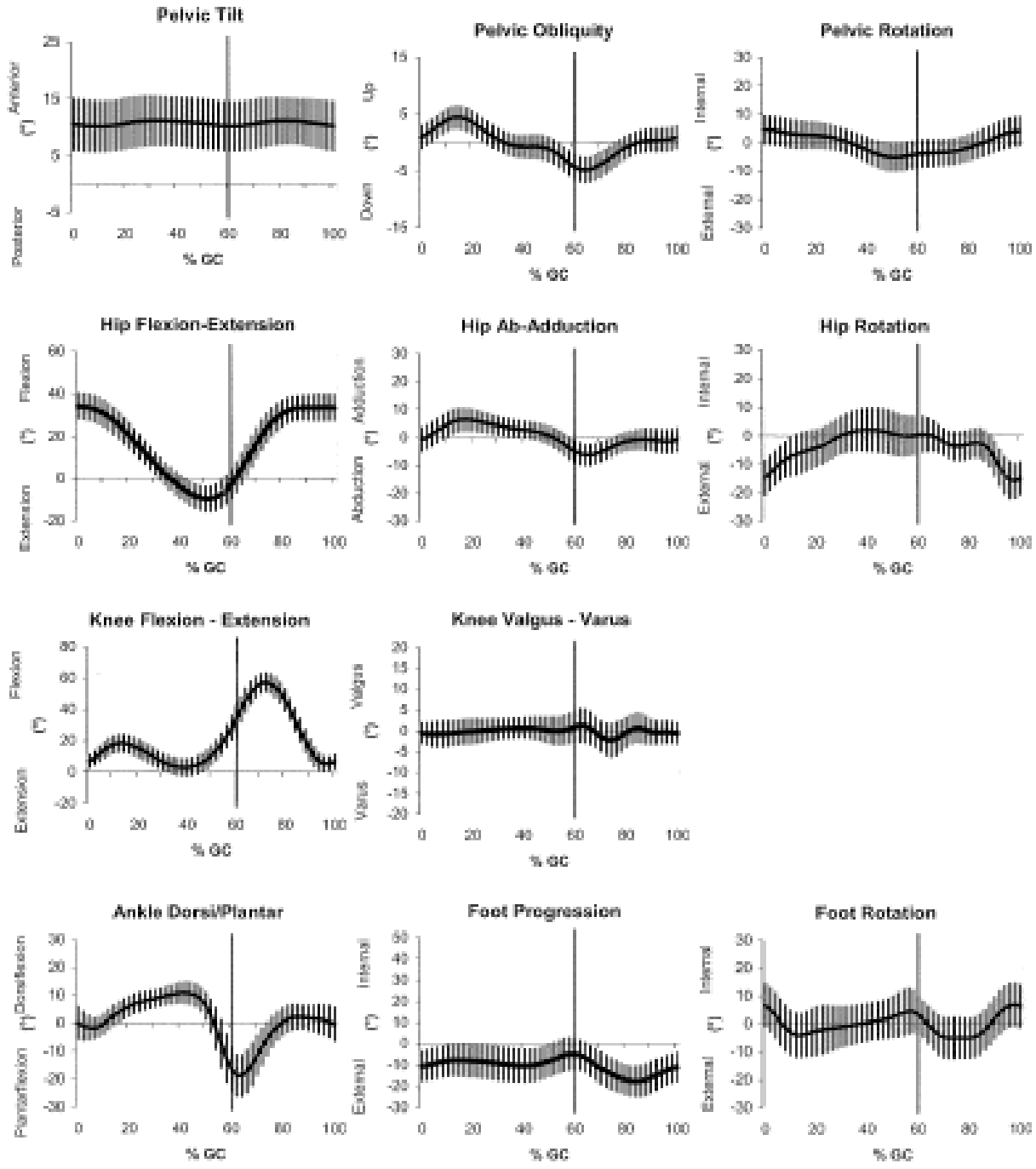

Figure 10 Pelvis, hip, knee and ankle kinamtics of healthy paediatric population.

Biomechanical model

A biomechanical model is a simplified representation of a biological system. The term „model“ refers to a set of assumptions and idealizations that allow to calculate important clinical information [23] (p.99). There are plenty of different models motion analysis of lower extremities. The first one we used is an antrhropometric model. This model is defined by markers attached over the bony landmarks (spina iliaca anterior superior, lateral malleolus etc.) and was developed in the pioneering years of 3D gait analysis, when the technology had difficulty tracking more than few markers. The most popular model is variously called Modified Helen Hayes, Kadaba or Vicon Clinical Manager. The mathematic equations describing the model may be found elsewhere [63;69]. As mentioned earlier in the text, the major limitation of these models is the accuracy of marker placement which is fundamental. Another source of artifacts is the skin movement and marker wobbling. However, this might be low-pass filtered [70]. Important limitation to be remembered is the definition and calculation of hip joint centre which is based on the location of the pelvis markers and the height of the patient [63]. These equations were developed from measurements of normal pelvic x-rays, making the model to be dependent on anthropometry. Indeed, the error of hip joint centre calculation may be as high as ±1cm [71].

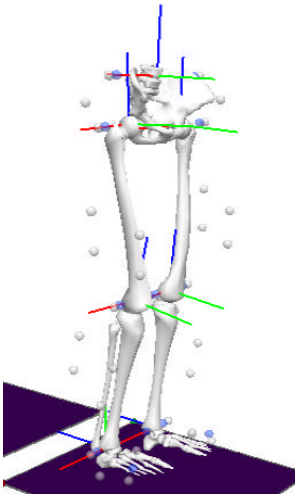

Figure 11 Ilustration of the six-degree-of-freedom model used in our study.

A second model used for our research (Figure 11) is so-called six-degree-of-freedom (6DoF) model. This model is not dependent on the anthropometry and marker placement is not as fundamental as in the previous model. In this aproach, the segments are tracked separately with no assumptions being made about joint constraints. It resolves the problematic hip joint centre calculation in Helen Hayes model and uncovers hidden artefacts[72] (p.59). To define the thigh and shank marker clusters, described by

Cappozzo et. al. [73], were used. This is simply set of four orthogonal markers. Although such clusters are attached to soft tissue, they are less sensitive to placement errors and appear to track the underlying bone more faithfully [74]. Despite all the advantages of the 6DoF models using marker clusters, the Vicon Clinical Manager model remains most routinely used for 3D clinical gait analysis.

Kinetics

As described earlier in the text, kinematic analysis is the study of the movement, joint angles and angular velocities. It describes the position of the body in the space. Kinetics examines why these movements happen and what are the consequences for the muscluloskeletal system. Speaking about kinetics, we speak about forces, joint moments and powers. Kinematics is an important part of clinical gait analysis because it allows to examine the mechanisms that either produce or control motion, thus developing a better understanding of gait. It brings additional information to kinematics. Moreover, unlike kinematics, moments and powers can not be directly observed.

When a person is standing still, the ground produces a reaction force equal and opposite to their body weight (based on the third Newton's law) [75]. This is called Ground Reaction Force (GRF) and acts on the sole in the centre of pressure (aproximatelly 5cm anterior to the ankle joint). In quiet standing, the GRF is constant, being equal and opposite to body weight (). Whenever a force is applied some distance away from a joint centre, it will tend to rotate the joint in the direction of the force. This effect is called the moment of force or simply joint moment (Figure 12).

Joint Moment = Force x Moment Arm (the distance from center of rotation)

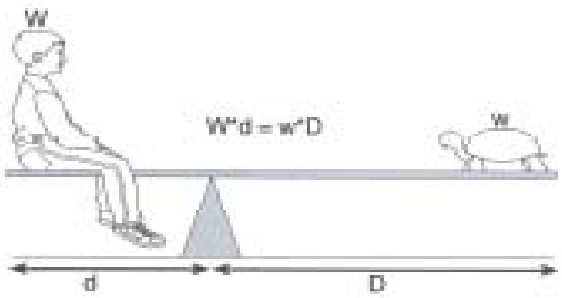

Figure 12. To produce a moment of the same magnitude, the individual on the right hand side needs a longer lever arm. Reproduced from Gage, J.R. *The treatement of gait problems in cerebral palsy*. London: Mac Keith Press; 2004. [23]

To balance the external dorsiflexing load caused by the GRF while standing, active tension has to counteract on the Achilles tendon. This internal plantarflexing moment is produced by calf muslces (Figure 13). More generally, this simple example helps us in interpreting the joint moments during the gait because it shows that the active muscle group is always the one on the opposite side of the joint to the GRF [75].

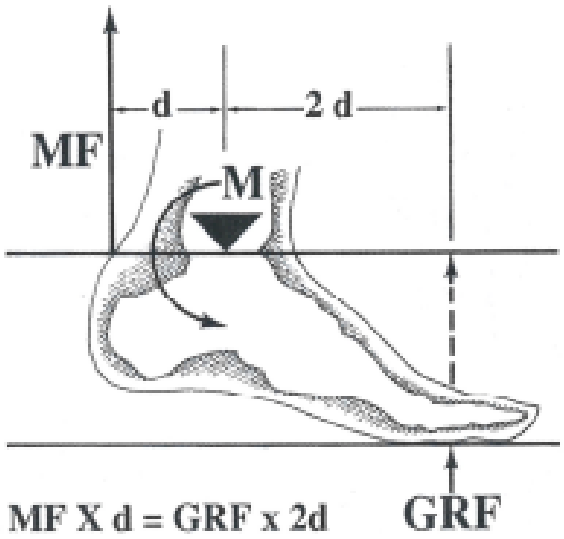

Figure 13. The relationship between the external moment produced by GRF and internal moment produced by plantarflexing muscles. Since the lever arm of the GRF is twice as long as that of the plantarflexor muscles, GRF magnitude would be only half of the one produced by plantarflexors. Reproduced from **Gage, J.R. *The treatement of gait problems in cerebral palsy*. London: Mac Keith Press; 2004. [23]**

During normal gait, GRF changes with the gait cycle, resembling the shape of a letter „M“ in sagital plane. These dynamic changes of GRF vector during gait oscilate above or below the body weight. It has been described by Pedotti [76] and is often reffered to as „Butterfly diagram“ (Figure 14). The GRF oscilation is caused by accelerating and decelerating phases during the gait cycle. Even if the vertical component of the GRF vector is the biggest one, there are another two shear components of this vector. Antero-posterior or fore-aft shear is directed posteriorly (braking) in the first 50% of the stance but anteriorly (propulsive) in late stance. Medio-lateral shear is almost always directed medially in response to lateral motion of the body [77].

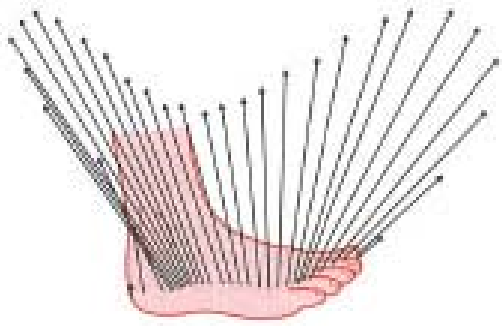

Figure 14. Buterfly or Pedotti diagram. Each arrow represents the GRF at each point of the gait cycle. Reproduced from Kirtley, C. *Clinical gait analysis: Theory and praxis*. Washington DC: Elsevier; 2006. [72]

The individual load and the shear components of the GRF vector can be measured using a device called force plate. This instrument uses either strain gauges or piezo-electric crystals to convert force into electric signals. Unfortunately, there is no practical and noninvasive method for estimating individual internal muscle forces or moments. Therefore external forces are measured instead and based on the mathematical and the second Newton's law of motion we relate these external forces and moments to internal ones. This approach is called „inverse dynamics" because it involves working back from kinematics to deduce muscle activity that must be responsible for them [78] (p.29-43). As mentioned before, we are unable to measure or calculate internal moments and forces for every individual muscle, thus reffering to muscle groups. When speaking about net internal plantarflexor moment or force it inlcudes all of the moments and forces produced actively or pasively by all structures (muscles, tendons, ligaments) crossing the dorsal aspect of the ankle joint (soleus, gastrocnemius, tibialis posterior, Achilles tendon etc.). To be able to mathematically model and understand such a complex system as human gait, certain assumptions and simplifications must be inevitably made. However, in vivo measurements using electromyography found good agreement of estimated joint moments with those calculated by inverse dynamics [79].

In addition to the GRF and joint moments, powers arise in response to changes in motion. Power measures the rate of change of energy. It describes its generation or absorbtion. For human joints, which predominantly function by rotating, the the power is a product of the joint moment and joint angular velocity and is measured in watts per kilogram [75].

Power = Joint Moment x Angular Velocity

The flow of power through the limb provides insight into the source and destination of the power responsible for driving the gait pattern. Passive flows of power take place across the joint surfaces, while active flows transfer power through the muscles and tendons. Of these, only the latter are generally reported by commercial gait analysis systems [56] (p.177). During the gait, muscle is capable of three basic functions: concentric contraction (power generation), excentric contraction (power absorbtion) and isometric contraction. When the moment and angular velocity act in the same direction, the muscle is performing concentric contraction. If they are in opposite direction, an excentric contraction takes place. In isometric contraction there is no power generation or production. Additional to power generation and absorbtion muslces redistribute power between segments [80].

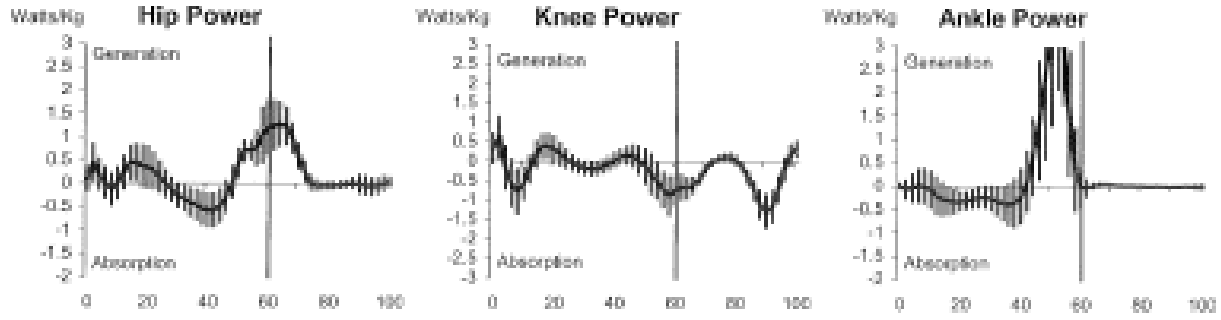

Figure 15 Mean Hip, Knee and Ankle powers of a group of healthy children generated over the gait cycle

Energy expenditure

Normal gait depends on an efficient use of energy something that quantitative gait data cannot directly measure. Conservation of energy is a typical aspect of walking. Oxygen consumption gives an objective view of the overall efficacy of the patient's gait. Many factors influence energy consumption, including spasticity, bony deformity, strength and selective motor control. Thus energy efficiency reflects the cumulative effect of many factors [59]. It is a functional tool because its interpretation provides an indication of endurance, fatigue and ability to accomplish the routine daily task of locomotion [23]. Children with cerebral palsy have been shown to expend greater energy during walking than their typically developing peers when walking at self-selected economical [60] as well as at a given speed [61].

Energy conservation is also accomplished by minimizing the displacement of the centre of mass (COM) while progressing forward in a steady, minimal, wavelike pattern [59]. COM trajectory is often compared to inverted pendulum, a model of gait showing how COM rises during single support and falls during

double support. Energy transfers have been identified as power-conserving mechanisms in walking. During the gait cycle, forward kinetic energy is converted to potential energy. The recovery rate during walking is about 65%, so only 35% of the energy needs to be replenished each cycle [81]. The energy recovery rate is speed dependent. Maximal energy recovery is achieved at a speed close to the natural walking speed. Children with cerebral palsy have a 33% smaller energy recovery and 60% greater COM vertical excursion which make them mechanically less efficient in comparisson to able-bodied peers [82]. The vertical displacement of the pelvis during walking is a strong predictor of oxygen consumption in normal subjects walking at variable speeds [83]. Hence, reducing energy expenditure by lowering COM displacement is an important goal in surgical treatment of cerebral palsy children.

Children with cerebral palsy typically expend 2-3 times as much energy as age matched controls [60]. This increased energy expenditure means that these children are less effective, they operate closer to their maximum level of effort and are therefore prone to fatigue [84]. Unnithan and colleagues demonstrated that 43% of additional energy cost in children with cerebral palsy might be explained by co-contraction of the lower extremity muscles while walking [30]. As mentioned earlier in the text, energy expenditure of walking is a good functional tool with known correlation to other gait parameters. It can provide us with a baseline assesment of children's disability or with a critical evaluation of effectivness of a particular treatment. Basically, energy required for walking may be calculated based on mathematical (inverse dynamics) or mechanical (using work-energy theorem [85]) estimation. These two methods are not often used in clinical gait analysis. The prefered method in the most of the laboratories is the metabolic energy expenditure assesment using indirect calorimetry. Nowadays open-circuit systems with breath-by-breath gas analyzers are used to measure oxygen utilization. This is most often expressed as oxygen consumption or oxygen cost. The oxygen consumption (VO_2) is a calculation of the rate of oxygen uptake normalized by body mass (ml/min/kg) and indicates the intensity of physical effort. Oxygen consumption for normal gait is about 14 ml/kg/min and about 3,5 ml/kg/min while standing still [59]. Oxygen cost describes the amount of energy needed to walk a certain distance also normalized to body mass and is a measure of gait efficiency. Energy cost of walking has been proved to be related to the severity of functional involvement in children with cerebral palsy [86].

The less demanding method to asses the energy cost of walking is the Physiological Cost Index [87]. This easy to use method is based on the nearly linear correlation of heart rate on oxygen consumption.

PCI = (Walking heart rate – Resting heart rate)/ Walking velocity

This method is laso reffered as Energy Expenditure Index [60]. The reliability of the Physiological Cost Index as a tool to measure the gait efficiency is often questioned in the literature with inconsistent results [88;89].

Dynamic Electromyography

Dynamic electromyography (EMG) is a substantial component of the instrumental protocol for assessment of motor disturbances in children with neuromuscular pathologies. The dynamic EMG recorded during gait represents the sum of signals from multiple motor unit action potential, thus dynamic EMG record reveals information on the timing and intensity of muscle activity [23] (p.134). EMG signals can be picked up over the skin surface (surface EMG) or by percutaneous fine needle electrodes inserted into the muscle belly [90]. Although activity of muscle groups may be estimated from kinematics and kinetics of gait, activity of a particular muscle might be of high importance when surgical intervention, for example muscle transfer, is planned.

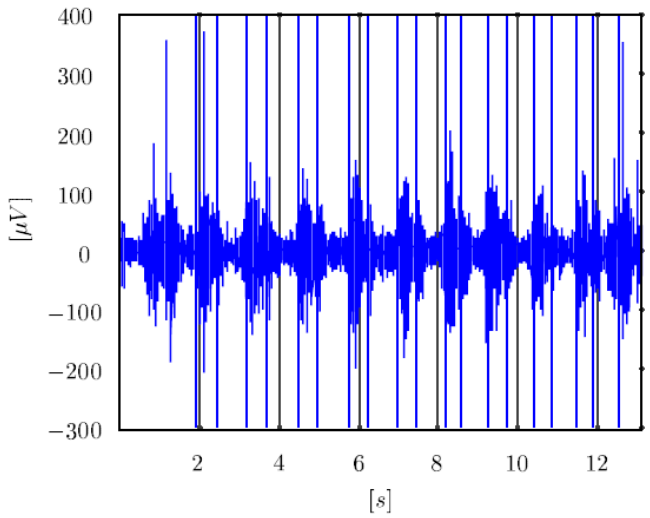

Figure 16. Raw EMG signal of tibialis anterior muscle during walking.

The nature of the EMG signal largely depends on factors associated with signal collection and data processing. The raw EMG signal recorded by surface electrodes is very small, thus it needs to by amplified to display it (Figure 16). The problem with EMG signal amplification is that the backround electrical noise signals would be amplified as well. This problem can be solved by differential preamplifiers which multiplies the difference between its two imput terminals by a large gain, whilst having a very small gain for any voltage that is present on both terminals [72] (p.139). The source of the

electrical noise signals are motion artefacts, positioning of the electrodes, skin impedance, subcutaneous fat, the position of the muscle (superficial muscles are easier to record) and crosstalk. Therefore beside the skin preparation, a proper electrode location is fundamental. A general criterion, proposed quite recently and based on reasonable considerations, is that the electrodes should be located midway between the motor end-point and the muscle-tendon junction [91]. Specific indications and suggestions on this point can be found in the report of the SENIAM European project [92].

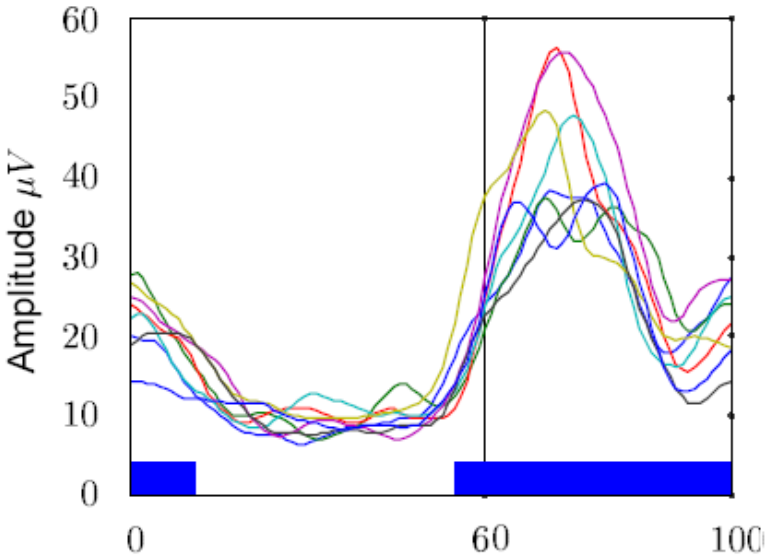

Figure 17. Rectified, enveloped and gait cycle normalized EMG signal of tibialis anterior muscle.

In most kinesiological studies, EMG signal is not presented in its raw form. The EMG data are synchronized with the gait cycle using foot switches or kinematic data measured together with EMG signal. This process is aften called averaging and the aim is to obtain a representative activation pattern in which the random component of variability is removed [90]. The raw EMG signal is biphasic in its nature thus another processing algorhytm called rectification is applied to the raw EMG signal. This is often done tohether with low pass filtering (mostly with cut-off frequency of 3Hz) to obtain smoothed so called „envelope EMG“ [90] (Figure 17). Since there are many factor affecting the amplitude of the EMG signal, some form of normalization procedure is desirable (e.g. root mean square technique which uses maximal voluntary contraction for EMG signal normalization) [93]. However, in practice this is rarely attempted, because it needs patient cooperation and therefore is somewhat subjective. Insted, in most of the gait laboratories EMG tends to be used for on/off information on muscle activity to describe premature, prolonged, out-of-phase, continuous, delayed or absent EMG activity pattern. Gait disturbances are frequently associated with poorly selective activation of functionally antagonist muscles, resulting in increased co-contraction levels. These can be also quantified using dynamic EMG

[90]. Surface EMG recording during walking can be used also for objective detection of the possible interference of a spastic component, i.e. of abnormally increased EMG recruitment during muscle lengthening phases . In fact, spasticity can contribute to hindered locomotor movements, can yield negligible mechanical effects, or can even be profitably included into a motor scheme [94].

Electromyography is an important component of gait analysis. It provides additional information about timing and action of muscles. Understanding the activity of the muscle together with other components of gait analysis is a key knowledge for understanding the root causes of gait abnormality.

Musculoskeletal modeling

With the recent advances in medical imaging technology, numerous software packages have been developed to process, display and manipulate medical images. Biomechanics researchers are interested in muscle forces and how the geometric relationships among the muscles and bones influence these forces. As a result, mathematical models have been developed to study parameters not measurable in vivo, e.g. dynamic muscle lengths changes while walking [95]. The management of the gait abnormalities in children with cerebral palsy is a challenging task. Computer simulations of the musculoskeletal system can be used in combination with gait analysis to enhance our understandig of movement abnormalities and to provide a theoretical basis for planning treatments. Delp et al. introduced the musculoskeletal modeling into the orthopaedic clinical praxis by examining the hamstring and psoas muscle lengths changes in patients with cerebral palsy who developed crouch gait [96]. Until then, hamstrigs were thought to be short in cerebral palsy patients and were routinely surgically lengthened. Delp demonstrated that most (80%) of the subjects with crouch gait had hamstrings of normal length or longer, despite persistent knee flexion during stance. This occurred because the excessive knee flexion was typically accompanied by excessive hip flexion throughout the gait cycle. All of the subjects with crouch gait had a psoas that was shorter than normal by more than 1 SD during walking. These results emphasize the need to consider the geometry and kinematics of multiple joints before performing surgical procedures aimed at correcting gait pathology in children with cerebral palsy.

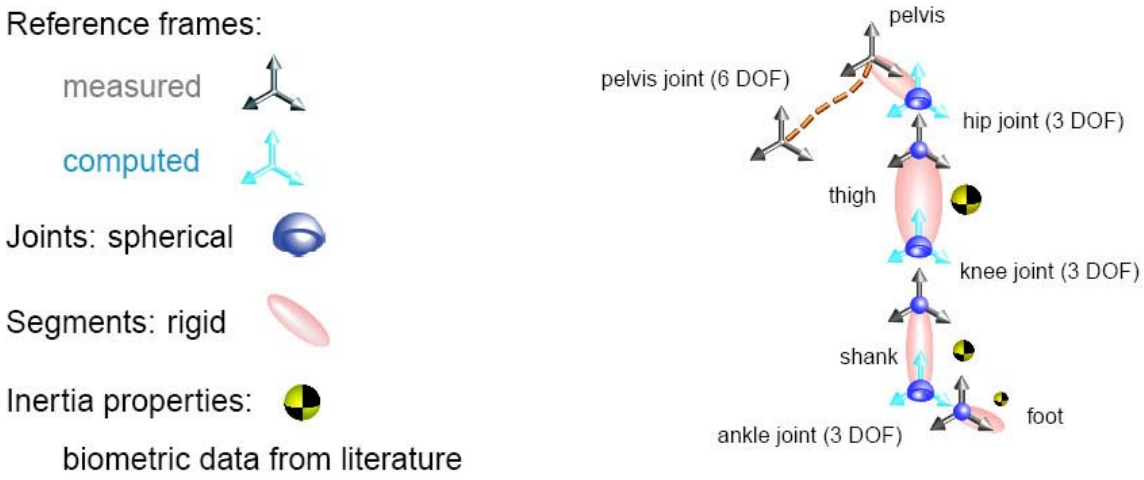

Figure 18. Basic structural elements of the kineostatic transmition chain of our lower extremity model.

The present mathematical models of the lower extremities to study gait consist of the structural and a parameter part. The structural part of the lower extremity model consist of kineostatic transmision chain comprising of a coordinate frames describing the position of the bones in the space, rigid bodies desribing the bones, and ball-and-socket joints describing the anatomic joint properties.(Figure 18) The model parameters are derived from three dimensional gait analysis capturing systems, which has been described earlier in the text. For the dynamic muscle length calculation in our study, only kinematic parameters are needed [97]. The more sophisticated lower extremity models use also kinetic parameters to compute the energy flows through the lower extremities. Nowadays, analyses of gait simulation may provide us with information on contributions of individual muscles to segmental accelerations and powers effecting the ground reaction force [98;99]. However, all these models are based on the previous computerized three-dimensional data from disected species [100]. This models are then scaled to size and mass of the subject studied. The most recent modeling techniques provide us with the possibility of individualized models based on the magnetic resonance imaging [101].

Muscle-tendon lengths are calculated as the distance from origin to insertion along the modelled path of each muscle. Via points are introduced to intersect the muscle-tendon paths taking into account the underlying anatomical constraints and defining the line of action of each muscle. Calculations are based on kinematic parameters obtained from gait analysis. The underlying model was derived from the work

of Yamaguchi [100]. The Figure 19 shows an example of normalized dynamic muscle lenghts over the gait cycle based on the data of healthy children.

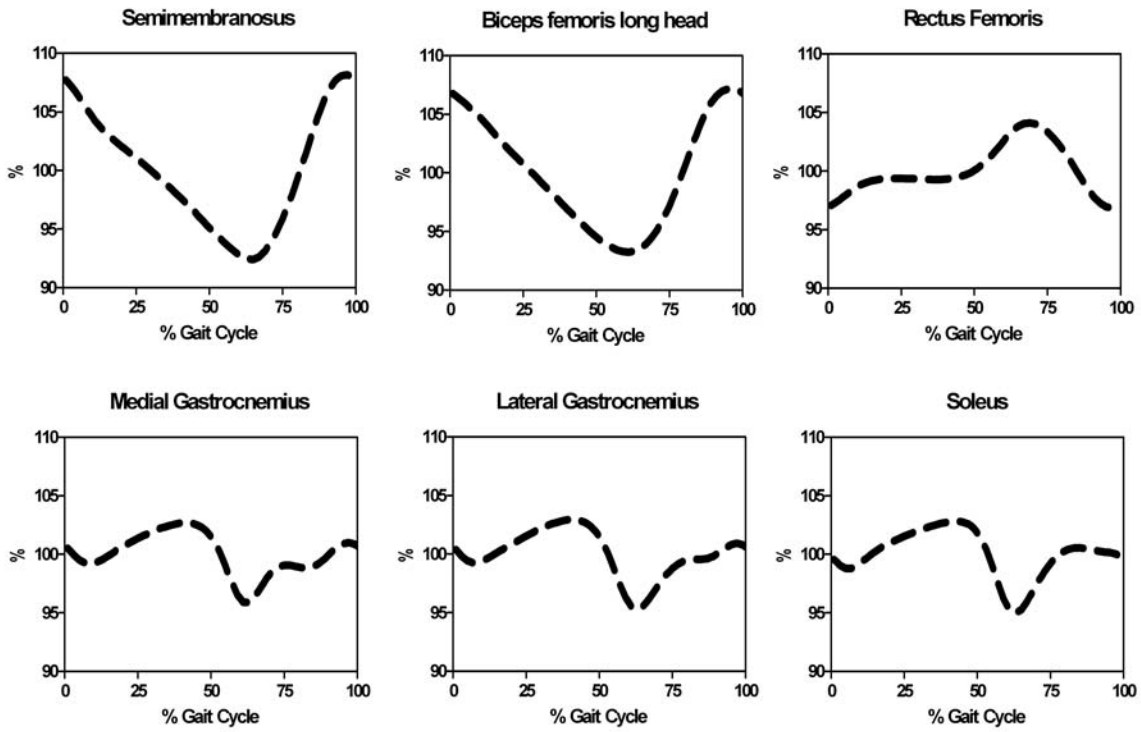

Figure 19. Normalized dynamic muscle lengths over the gait cycle based on the data of healthy children.

PRACTICAL PART

Purpose of the study

Gait and posture disturbances are one of the most common problems in children with cerebral palsy. Its treatment is a challenging task for many healthcare professionals across a wide spectrum of different specialities. The results presented in this thesis broaden the knowledge on pathophysiology of movements abnormalities in children with cerebral palsy and therefore allow us to better understand this disorder with possible implications in clinical practice.

Study 1 – Genu recurvatum in children with cerebral palsy

Hyperextension of the knee in the stance phase of the gait cycle (knee recurvatum) is a common disorder of the knee function present in patients with cerebral palsy. Simon et al. [102] described two distinct groups of knee recurvatum based on the timing of maximum knee hyperextension in stance – early and late recurvatum. The aim of our study was to prove that early knee recurvation is caused by a dynamic equinus deformity and late knee recurvation is linked to the fixed equinus deformity. These two distinct forms of equinus possibly point to a need for different treatment strategies. Therefore, such a differentiation would have direct clinical implications. Second aim was to analyze the role of hamstrings in the pathobiomechanics of these two distinct forms of the knee recurvatum.

Study 2 - Dynamic versus fixed equinus in children with cerebral palsy. How does the triceps sureae work?

Equinus deformity is a very common problem in children with cerebral palsy. Equinus decreases the stability during the stance phase of gait, causes a loss of the smooth translation of the body over the foot and often leads to an inadequate clearance of the foot during the swing phase of gait. The aim of this study was to evaluate the function of the triceps surae complex during walking when either a dynamic triceps surae tightness or a fixed equinus contracture is present in children with cerebral palsy. Findings are compared to a group of normally developing children.

Study 3 - Short-term effects of Botulinum Toxin A and serial casting on triceps surae muscle length and equinus gait in children with cerebral palsy

Equinus gait is one of the most common motor manifestations of muscle spasticity in cerebral palsy. If untreated, protracted spasticity can lead to fixed muscle contractures that may require surgical intervention. To prevent that, dynamic equinus deformity is often treated with botulinum toxin type A (BTX-A). The primary goal for the study was to perform a comprehensive analysis of the short-term

effects of a standardized dose of BTX-A and serial casting on spastic equinus in children with cerebral palsy. A second goal was to evaluate its short-term effect on muscle length of the triceps surae.

Study 4 - Detecting postoperative change in children with cerebral palsy: Net nondimensional versus body mass oxygen normalization

Conservation of energy is a typical aspect of walking. Children with cerebral palsy have been shown to expend greater energy during walking than their typically developing peers. To quantify the energy cost of walking, oxygen consumption is often used. It is important to have an utilization measure independent of children's age, weight and height in order to allow efficiency to be compared between subjects. The nondimensional normalization scheme was found to be superior to body mass normalization. The aim of this study was to investigate if the nondimensional oxygen utilization scheme is able to detect postoperative change in the energy cost of walking in children with cerebral palsy and to compare it to clinically used body mass normalization scheme.

Study 5 - Evolution of walking ability after soft tissue surgery in cerebral palsy patients. What can we expect?

The gait performance in patients with spastic cerebral palsy is impaired compared to healthy children. There is a great deal of evidence showing that the walking ability can improve after the surgery. However, there is not enough evidence to show the evolution of early stages of regaining the ability to walk soon after the soft tissue surgery. The aim of this prospective study was to show the evolution of different gait parameters over a period of nine months following soft tissue surgery.

Research Methods

Subjects

The presented studies were performed on different groups of children but all of them had the spastic cerebral palsy. The subjects of the Study 1-3 were recruited from the patients of Department of Peadiatric Orthopaedics, Department of Paediatric Surgery, Medical University of Graz, Austria during the years 2008-2009. The subjects of the study 4 and 5 were recruited from Department of Children and Adult Orthopaedics and Traumatology, 2nd Faculty of Medicine, Charles University, Prague, Czech Republic. All children were able to walk, even if some of them used walking aids for longer distances. They were evaluated to be level I-III using the Gross Motor Function Classification Scale [103]. The different gait abnormalities, grouping, demographical description and all the inclusion and exclusion criteria can be found in the corresponding papers.

Methods

This section is just a brief survey of methods used in our research. For more detailed information, please refer to corresponding papers or to the text of the Theoretical part of the thesis.

Gait analysis

Computerized gait analysis was performed using video-based motion capturing system and floor-mounted force plates. Reflective markers and clusters were used to define the pelvis and lower extremity segments according to the method of Kadaba [69]. Moment and power parameters were normalized to the weight of the patients. Power generation and absorption patterns in the sagittal plane were calculated and labelled according to the method described by Winter et al. [104]. All patients walked at self-selected speeds. For each patient a minimum of five trials providing a clear foot force plate contact were captured and averaged. Time-distance parameters were normalized to the leg length according to the method of Hof [105]. Vertical Centre of Mass (COM) excursions were measured as a trajectory of sacral marker [106].

Muscle length calculation

Based on kinematic data, Mobile GaitLab software (University Duisburg-Essen, Institute of Mechatronics and System Dynamics, Germany) [107;108] was used to compute individual muscle lengths. The specification of the lower extremity architecture was based on the origin and insertion coordinate data compiled by Yamaguchi [100]. The model defines the musculoskeletal geometry of a normal adult male. Three dimensional muscle-tendon (MT) pathways were modelled as a series of straight-line segments

extending from the origin to insertion. Where appropriate, the MT pathways were sub-sectioned into a number of straight-line segments by introducing via-points. Muscle-tendon length parameters were normalized to allow the comparison among subjects.

Electromyography

Raw surface electromyograms were recorded in a frequency range between 25 and 400 Hz (Noraxon, Myosystem 2000). After an analogue to digital conversion and sampling at 1 kHz, the signals were fully wave rectified and digitally low-pass filtered at 6 Hz. After time normalisation, a minimum of 12 gait cycles was averaged to create an average linear envelope for each muscle. Calculation methods applied have been described in detail by Yang and Winter [109].

Energy expenditure

Energy expenditure was measured while walking on a treadmill (Marquette T2000) and using respiratory gas analysis system (Oxycon beta, Jaeger, Hoechberg, Germany). The monitored parameters were: oxygen consumption (VO_2) and carbon dioxide production (VCO_2). The derived parameters were gross oxygen consumption relative to body mass (mass relative VO_2), nondimensional VO_2 [110] normalized to leg length and respiratory exchange ratio (RER). Selected parameters were recorded at rest and at a given speed of 2 $km.h^{-1}$ and 3 $km.h^{-1}$ at 0% gradient on treadmill. The steady state was defined as a variation of both VO_2 and respiratory exchange ratio lower than 10% [111] and was expressed as the average of the last 30 seconds of the four minutes stage.

Statistical Methods

Means and standard deviations (SD) were calculated for each subject group. To reveal the differences between groups, Student's t-test, Analysis of Variance with Fisher's post-hoc test or nonparametric Mann-Whitney U-test were used where appropriate. Level of significance in all tests was set to $p<0,05$. Sperman rank correlation coefficient was calculated to find out significant relationships between selected parameters. All the statistical tests were performed using STATISTICA 6 software (StatSoft, Tulsa, USA).

Results

Only the most important results are presented in this section. For more detailed information on precise numerical and statistical significances, please refer to the corresponding papers in the Appendix of the thesis.

Study 1

Genu recurvatum in children with cerebral palsy

Study 1 – Part I

Children with cerebral palsy and recurvatum walked slower, had a shorter stride length and longer stance time in comparison to normally developing children (Study 1-Part I, Tab. 1). However, there was no difference between the Early Recurvatum and Late Recurvatum group. Although, both groups rapidly dorsi-flexed the ankle up to a mild dorsi-flexion during the loading response, dorsi-flexion stopped at 12% of the gait cycle in the Early Recurvatum group and then the foot moved back to plantar-flexion (Study 1-Part I, Fig. 2). The Late Recurvatum group showed an arrest in tibia advancement at about 17% of the gait cycle and remained in a mild dorsi-flexion over the whole stance phase (Study 1-Part I, Fig. 3). None in either group were able to achieve normal dorsi-flexion during stance (Figure 20). Electromyographic evaluation of muscle activation patterns revealed no differences between the REC groups in gastrocnemius activity. Both groups had an early high activity in the first double support followed by abnormally high activity during the single stance phase. A similar phasic activity was observed for the soleus muscle in both groups but only the Early Recurvatum group demonstrated an abnormally high activity during the single stance phase ($p=0.042$).

Muscle-tendon length parameters revealed substantial differences among the early and late recurvatum groups and a group of normally developing children. The medial gastrocnemius in the Early Recurvatum group elongated rapidly to its maximum during the first double-support phase (approximately the first 10% of the gait cycle). It subsequently shortened gradually during the rest of the stance phase. In contrast, the medial gastrocnemius of Late Recurvatum group gradually elongated to its maximum length that was reached at 46% of the gait cycle (Figure 20). The soleus muscle of the Early Recurvatum group elongated quickly from initial contact to its peak at 11% of the gait cycle. At the start of single limb support, the muscle shortened and remained at a constant length during the rest of the single stance phase. The soleus in the Late Recurvatum group elongated slowly to its maximal length and did not change over the single stance phase (Figure 20).

The differences in the patterns of muscle length changes during the single support were described by the slope quotient of muscle lengths. While positive values were obtained for the medial gastrocnemius and soleus muscles in the Norm group, the Late Recurvatum group showed slope quotient values close to zero and for the Early Recurvatum group negative values were calculated. Negative values indicated muscle shortening during the single support phase (Study 1-Part I, Table 2).

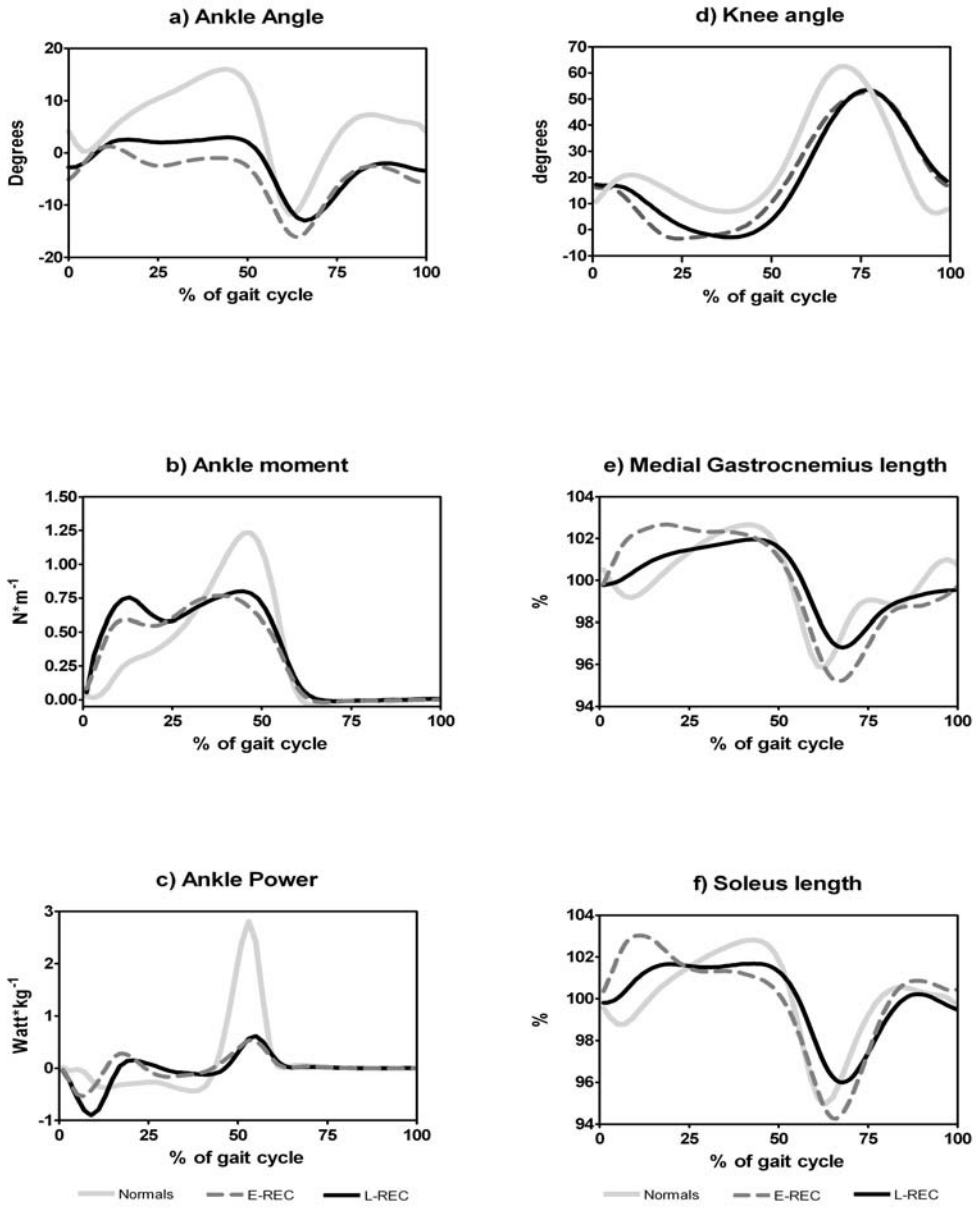

Figure 20. Time-distance parameters, kinematics, kinetics and normalized muscle-tendon lengths of the Early (E-REC) and Late (L-REC) Recurvatum groups compared to a group of normally developing children (Norm). The vertical lines define the single stance phase of the gait cycle for each recurvatum group.

Study 1 – Part II

Both recurvatum groups demonstrated increased external extensor moments across the knee in the stance phase of the gait cycle. The increase was greater in the Early Recurvatum group. All extremities with the knee recurvatum showed a forefoot landing. Electromyographic evaluation of muscle activation patterns revealed no differences between the Early Recurvatum and the Late Recurvatum. In contrast to a normal muscle activation pattern, 87% of the spastic limbs demonstrated prolonged activity of the medial hamstrings during stance and their early activation during swing phase.

The normalized muscle-tendon lengths of both medial and lateral hamstrings for both recurvatum groups were abnormally long at initial contact (Figure 21) when compared to the control group (Study 1-Part II, Tab. 2). During the single limb support phase, hamstrings of both recurvatum groups were contracting faster and shortened approximately 16% of the mean muscle-tendon length compared to a shortening of 10% for the control group respectively (Study 1-Part II, Tab. 2). In single limb support the mean hamstring muscle-tendon length was shorter in the Early Recurvatum group than in the Late Recurvatum.

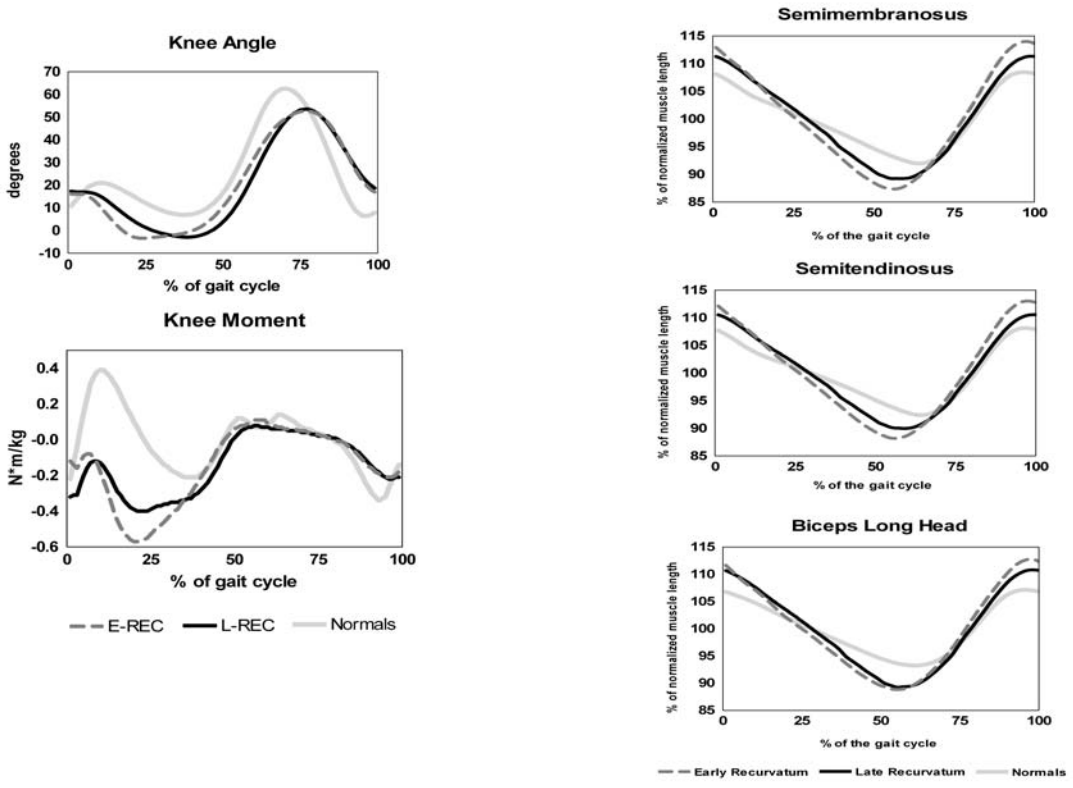

Figure 21. The knee kinematics, kinetics and dynamic muscle-tendon lengths over the gait cycle. Graphical comparison of means over the gait cycle among all the three groups. (E-REC = Early recurvatum group, L-REC = Late recurvatum group, Norm = Control group of normally developing children).

Study 2

Dynamic versus fixed equinus in cerebral palsy in children with cerebral palsy. How does the triceps sureae work?

Analysis of ankle joint motion revealed that none of the equinus feet reached a neutral position at any time during the gait cycle (Study 2, Fig 1). The maximal dorsiflexion in stance was not different between the Dynamic Equinus and Fixed Equinus groups. The plantarflexion at toe-off and the ankle range of motion were different for the three groups with the Dynamic Equinus group showing more plantarflexion than the Fixed Equinus group, which exceeded the values of the Control group (Study 2, Table 1). The ankle slope parameter was different among the three study groups (Study 2, Table 1). Negative values for both equinus groups indicated a pathological plantarflexion motion during single support, which was more pronounced for the Dynamic Equinus group (Study 2, Fig 1). About 50-70% of the propulsive power for walking is generated by plantarflexor muscles at the end of single support stance. Although the Dynamic Equinus group generated more power during plantar flexion than the Fixed Equinus group (Study 2, Fig 1) the maximum ankle power generation during the stance phase of gait was reduced for both equinus groups when compared to the Control group. There was an early timing of maximum ankle power generation in Dynamic Equinus group (29 percent of the gait cycle). Kinematics of the knee revealed a decreased range of knee motion for both equinus groups. Also, the maximal knee flexion occurs later during the swing phase in both equinus groups (Study 2, Table 1).

Muscle-tendon length parameters of the soleus, medial and lateral gastrocnemius muscles were abnormal for both equinus groups when compared to the Control group (Study 2, Table 2). While in healthy children the triceps surae muscles elongated during the single stance phase, the muscle complex appeared to plateau in its length for the Fixed Equinus group and even showed shortening for the Dynamic Equinus group (Figure 22). During the stance phase the timing and the amount of the maximal muscle-tendon lengths of the soleus and the medial gastrocnemius were different among the equinus groups (Study 2, Table 2). The muscle-tendon lengths at toe-off were abnormally short in Fixed Equinus group compared to Dynamic Equinus and healthy children. Equinus limbs demonstrated slower contraction/elongation velocity than limbs of the Control group. Moreover, the muscle-tendon velocity was slower in the Fixed Equinus group when compared to the Dynamic Equinus group (Study 2, Table 2). The graphical summary of important results distinguishing dynamic and fixed equinus using gait analysis and musculoskeletal modeling is given in Table 4.

	DEQ	FEQ	NORM (mean ± SD)
Ankle ROM	↓	↓↓	31.09 ± 5.62 ˚
Timing of maximal plantarflexor power	↓↓	↓	53.07 ± 1.25 % of GC
Max plantarflexion power	↓	↓↓	3.27 ± 0.51 Watt*kg-1
Ankle angle at toe-off	↓↓	↓	-11.35 ± 4.47 ˚
Ankle slope parameter	↓↓	↓	9.86±5.11 ˚
Maximal medial gastrocnemius muscle-tendon length	Norm.	↓↓	102.78 ± 0.56 %
Maximal medial gastrocnemius muscle-tendon velocity	↓	↓↓	0.19 ± 0.03 m*s-1

Table 4. The major differences between dynamic (DEQ) and fixed (FEQ) equinus groups in comparison to normally developing children (NORM). (↓=decreased in comparison to normals, ↓↓= very decreased in comparison to normals, Norm.=within the normal range)

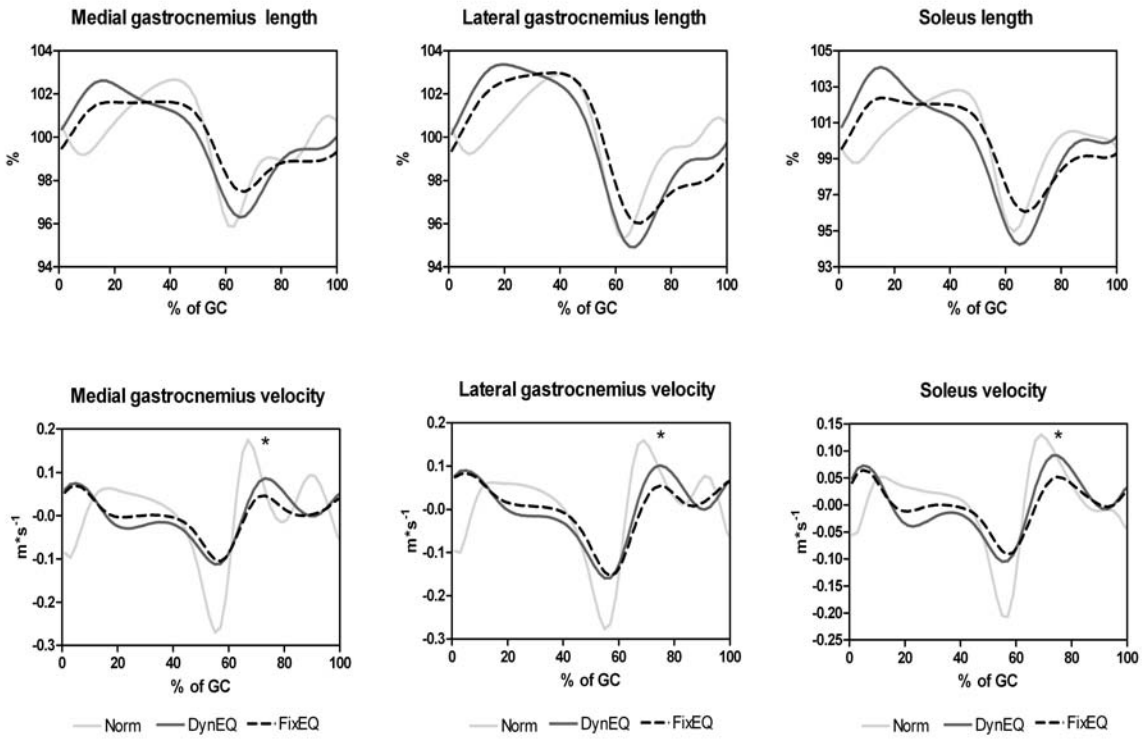

Figure 22. Muscle length and velocities of triceps surae in children with dynamic and fixed equinus deformity in comparison to normally developing children. The * (star) shows the statistically significant differences in the peak lengthening velocity of each head of triceps surae muscle.

Study 3

Short-term effects of Botulinum Toxin A and serial casting on triceps surae muscle length and equinus gait in children with cerebral palsy

The walking velocity and the stride length did not change after the BTX-A application. Ankle kinematics and kinetics changed towards normal 10 weeks after the treatment (Figure 23). The maximal ankle angle at the end of single support increased (p=0,045) and also the timing of the maximal angle was shifted towards normal values (Study 3, Tab. 2). The ankle angle slope quotient changed to normal positive values (p=0,001). We did not observe any changes in the knee kinematics. While the maximum ankle moment values remained unchanged, the abnormal ankle moment peak in the first part of the single support phase decreased and the timing of maximum ankle moment occurred later in the stance phase (p=0,024). An abnormal power generation in the first part of the single support stance phase decreased markedly (p=0,012). Musculo-skeletal modeling revealed no lengthening of the triceps surae muscles compared to the baseline assessment (Study 3, Tab. 2). We did not observe any adverse effects in our study group.

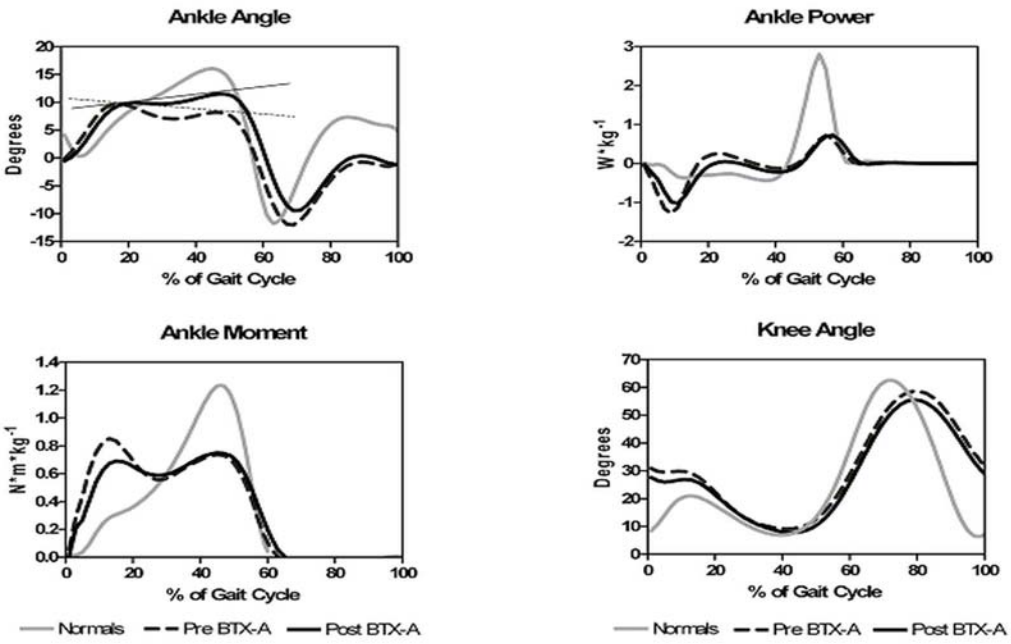

Figure 23. The sagittal plane ankle and knee kinematics and kinetics plots over the gait cycle. The straight lines in the Ankle Angle graph show the slope quotient pre- and post application of Botulinum toxin A. A rising "slope quotient" indicates improvement of ankle motion. There is an obvious improvement to normal positive values of slope quotient. (Normals = normally developing children, Pre BTX-A = patients with cerebral palsy before the Botulinum toxin A application, Post BTX-A = patients with cerebral palsy after the Botulinum toxin A application)

Study 4

Detecting postoperative change in children with cerebral palsy: Net nondimensional versus body mass oxygen normalization

The average comfortable walking speed (2.025 $km.h^{-1}$) did not change after the surgery. Gait analysis demonstrated normalization of the ankle motion. The ankle position at the initial contact changed from 9.6˚ of plantarflexion to 1.7˚ of dorsiflexion (p=0.001) and the maximal dorsiflexion during the stance phase of the gait cycle increased from 1.7˚ to 12.2˚ (p=0.022). Knee flexion angle at initial contact decreased from 33.2° to 21.1° (p<0.0001). During single limb support, the knee flexion angle also decreased by 14.5° (p<0.0001).

There was no difference between pre- and postoperative resting VO_2 and respiratory exchange ratio. Oxygen utilization data are shown in Figure 24. There were significant decreases in both mass relative and nondimensional VO_2 parameters at 2 $km.h^{-1}$ as well as at 3 $km.h^{-1}$. Preoperative and postoperative coefficients of variation of the net nondimensional VO_2 were notably lower at both speeds when compared to mass relative VO_2 (Study 4, Table 1). The Spearman rank correlation revealed a significant association between mass relative and net nondimensional VO_2 at both speeds preoperatively, as well as postoperatively.

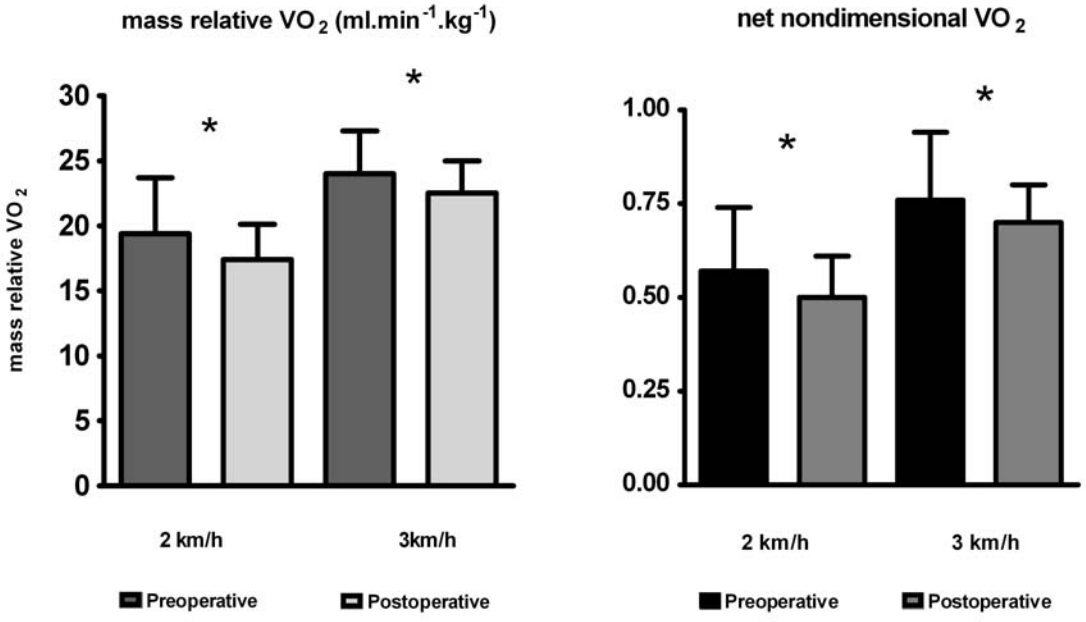

Figure 24. Comparison of pre- and postoperative oxygen consumption results using two different normalization schemes (* indicates the statistical significance at the *P*<0.05).

Study 5

Evolution of walking ability after soft tissue surgery in cerebral palsy patients. What can we expect?

We found a decrease in time-distance parameters after three month, but this was followed by progress in all parameters at nine months postoperative gait analysis. Step length and velocity decreased at first follow-up and by the time of the second follow-up increased to higher value than preoperatively. At initial contact the position of the foot changed from an average 8,28° plantarflexion to neutral position at the first follow-up and was maintained until the last gait analysis. Mean ankle angle in single support normalized nine months after surgery. Push-off range of ankle motion significantly decreased after surgery, but was restored to the same level as preoperatively after nine months period. (Figure 25; Study 5, Tab. 2). We observed an improvement of knee joint movement towards extension at stance phase with maintainence of knee flexion in swing, resulting in increased knee range of motion. Overall hip joint motion had a tendency to more extension pattern. Maximal hip extension over the gait cycle improved. Maximal hip flexion during a swing was not changed over the time. The centre of mass vertical excursion decreased markedly during three postoperative months, but did not reach the statistical significance. (Study 5, Fig.4) Physical examination confirmed the results of gait analysis and revealed increase in hip flexion and extension during the passive range of motion. There were no changes in hip rotations. Passive knee flexion increased. The most dramatic changes were seen at the ankle: passive dorsiflexion increased from 7.44° of plantarflexion to 8.18° of dorsiflexion after three months and this remained throughout the whole follow-up period.

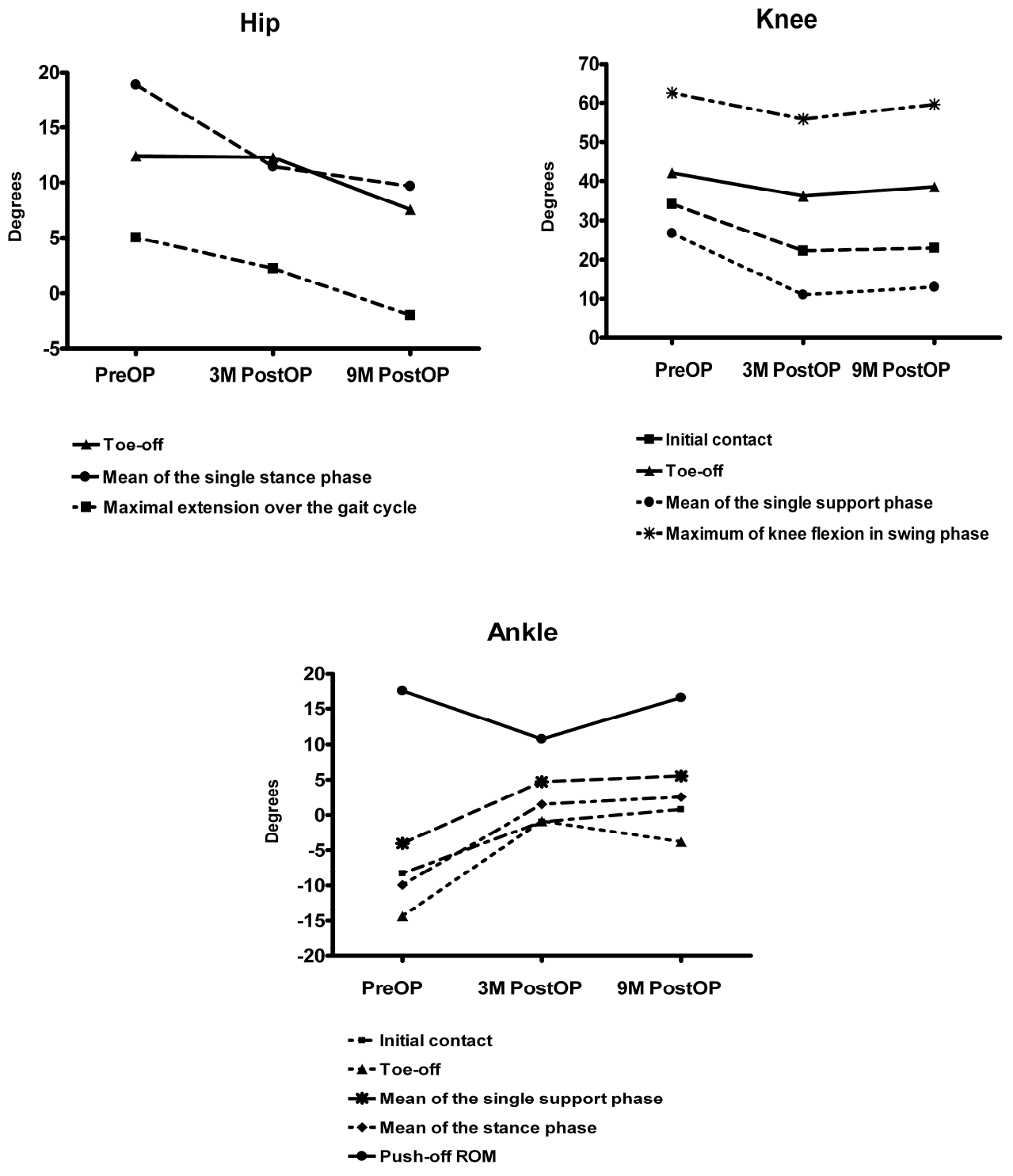

Figure 25. The evolution of postoperative gait parameters of ankle, knee and hip joint. (PreOP = Preoperatively, 3M PostOP= 3 months postoperatively, 9M PostOP = 9 months postoperatively)

Discussion

The Musculoskeletal Modeling

The first three studies presented use musculoskeletal modeling techniques to evaluate the the pathobiomechanics of the spastic muscle. As there is no direct way to measure muscle length in humans, muscle length calculations are based on mathematical models of the lower extremities and on measurements of ankle, knee, hip and pelvis kinematics. It is obvious that such approaches to mathematical modeling have limitations, but the method is well accepted in the literature [112;113] because it provides additional information which can help us to better understand the biomechanics of a spastic muscle. Even if the studies cover a wide variety of modeled muscles, there are some limitations to be considered. The Mobile GaitLab utilizes musculoskeletal data of a normal adult male [100;114]. This aproach is widely accepted and there are no children models available. Moreover, similar models have also been used as an outcome measure in studies on children with cerebral palsy [96;115]. To get rid of the of the dependance of the real adult meaures, the dynamic muscle length are normalized and presented as dimensionless number. The use of an adult model should not affect normalized muscle lengths [116]. A more elaborate way to study muscle-tendon lengths would be to use an individualized MRI-based model. However, at the present time, building an individualized MRI-based model would be costly and labour intensive and is not feasible for retrospective evaluation. Therefore these models have been only used for a very limited number of patients. Another consideration should be that children with cerebral palsy cannot be assumed to have normal bone architecture, osseous deformities could alter muscle attachment points and muscle paths. Schutte showed the effect of femoral anteversion on psoas muscle length [117]. To our knowledge, no study has yet determined the influence of bone deformities (tibial torsion, ankle varus/valgus) on the calculation of triceps surae muscle length. The method of Eames [118] has frequently been applied to calculate gastrocnemius muscle-tendon length. Eames used a simple 2-dimensional model based on the measurements of the knee X-rays and MRI. This model does not account for the two heads of the gastrocnemius muscle and therefore it might be too simplistic. There is an evidence that the medial and lateral heads of the gastrocnemius muscle differ considerably in their architecture [119], their length and pattern of activation [120]. The Mobile GaitLab muscle model uses separate attachement points for the two gastrocnemius heads according to Yamaguchi‘s data [100]. Therefore the results in our study might be more realistic because they account also for the rotational movements of the lower extremity.

There is another important limitation of the mechanical models of lower extremities, such as the Mobile GaitLab. These models do not have antropomorphic muscles and therefore they are unable to understand the muscle coordination [121]. In the very recent studies, new type of models occured. Simulation based analyses of the muscle actions, in combination with measured gait kinematics and electromyographic (EMG) recordings, have tremendous potential to advance our understanding of normal and pathological movements. In addition to the model used in our studies, muscles are not simulated as a straight lines spanning between two points, but have their own properties and are driven by force plates measurements and EMG signals. Simulation derived from dynamic models with physiologic-like muscles have a very high potential of understanding of coordination of gait. These models may identify the instantaneous contribution of individual muscles to acceleration and energetics of the individual body segment. Analysis of these muscle-induced segmental accelerations and powers have shown how muscles work together in synergy, or independently to coordinate movement of the body [98]. For example, Hicks at al. [122] showed that the capacities of almost all the major hip and knee extensors were markedly reduced in a crouched gait posture, with the exception of the hamstrings muscle group, whose extension capacity was maintained in a crouched posture. Crouch gait also increased the flexion accelerations induced by gravity at the hip and knee throughout single limb stance. These findings help explain the increased energy requirements and progressive nature of crouch gait in patients with cerebral palsy. Another study [99] quantified the muscle contributions to vertical support and forward progression over a range of walking speeds. They found that during very slow and slow walking speeds, vertical support in early stance is primarily provided by a straighter limb, such that skeletal alignment, rather than muscles, provides resistance to gravity. When walking speed increased from slow to free, contributions to support from vasti and soleus increased dramatically. Greater stance-phase knee flexion during free and fast walking speeds caused increased vasti force, which provided support but also slowed progression, while contralateral soleus simultaneously provided increased propulsion. These types of modeling studies demand a deep mathematical and biomechanical backround, knowledge of forward dynamics and do not allow to process greater number of subjects. They are therefore not feasible for clinical research.

Study 1

Genu recurvatum in children with cerebral palsy

Knee hyperextension during the stance phase of the gait cycle is commonly observed in children with cerebral palsy. Children with cerebral palsy often have poor motor control at the ankle. In all CP children included in this study, knee recurvatum occurred secondary to abnormal movement of the tibia during the stance phase. Our results based on the musculoskeletal modeling, show two distinct patterns of the triceps muscle behaviour during the stance phase of the gait cycle.

In the Early Recurvatum group, tibia advancement over the planted foot is suddenly stopped by an exaggerated stretch reflex [123] of the soleus muscle, and the foot is brought back to equinus position. Such a spastic “catch-up” is clear evidence of muscle spasticity and the dynamic nature of the equinus deformity in the Early recurvatum group. This behaviour of the soleus is further confirmed by the electromyographic hyperactivity, present throughout the single stance phase, and was the only statistically significant difference in EMG activity between the REC groups. More evidence of the dynamic nature of equinus in Early recurvatum group is given by the clinical examination, which agrees with our hypothesis.

The gastrocnemius of Early recurvatum elongates, similarly to the soleus, to its maximal length during the loading response. Because the gastrocnemius is one of the knee stabilizers, a rapid elongation might add to an existing instability to the knee joint, and the gastrocnemius could then not act as a restraint [124]. The rapid elongation of the gastrocnemius medialis might also support the soleus muscle in its pathological function of pulling the tibia backwards. The gastrocnemius muscle’s rapid elongation to its maximum length could also be indication of weakness which has been reported as one of the causes of knee recurvatum [102;125;126]. In our study this is only an indirect sign of weakness obtained from gait analysis and we do not have clinical data to back up this finding.

The triceps surae muscle of the Late Recurvatum (Late recurvatum) group demonstrated an entirely different pattern of action. The soleus muscle slowly elongates to its maximal length and then stops the tibia during forward progression. It then remains at a constant length during the whole single stance phase. The soleus and the medial gastrocnemius have a smaller range of dynamic muscle lengths. This could be interpreted as a sign of increased muscle stiffness. Increased muscle stiffness would be expected in the presence of a fixed equinus deformity. The dynamic behaviour of the gastrocnemius muscle in the Late recurvatum group showed a pattern closer to normal. Perhaps a good parameter to

describe the dynamic action of the soleus and the gastrocnemius muscles in the single stance phase is the slope of muscle length. The slope quotient demonstrates that while triceps surae muscles in the Early recurvatum group contract and shorten, they remain at a constant length, or show a slight elongation in the Late recurvatum group. As walking velocity was comparable for both recurvatum groups, the group differences observed in musculoskeletal modeling should not be biased by this factor.

In a study of children with hemiplegic cerebral palsy, Hullin et al. proposed that the difference between the two groups of recurvatum patients is the degree of soleus tightness – early hyperextenders have a relatively tighter soleus than late hyperextenders [124]. In contrast to his observation, our results clearly indicate that early knee recurvatum is more likely associated with dynamic equinus deformity. A smaller range of dynamic muscle lengths of the medial gastrocnemius and soleus muscles in the Late recurvatum is to be considered as a sign of the fixed contracture. Moreover, there is a 85,8% agreement between the fixed equinus deformity assessed by clinical examination and the Late recurvatum group.

In the second part of the study, kinematic and kinetic analyses together with musculoskeletal modeling and surface electromyography were used to investigate the role of the hamstrings in children with cerebral palsy who developed knee recurvatum. Hamstrings are biarticular muscles providing hip extension and knee flexion. The length of these biarticular muscles does not intuitively reflect angular changes of a single joint, but is dependent on angular changes of multiple joints as well as on the moment arms *about* these joints. Delp introduced musculoskeletal modeling into the clinical routine, reporting that 80% of patients with cerebral palsy who developed crouch gait had hamstrings of normal length or longer, despite persistent knee flexion during the stance phase of gait [96]. This was an unexpected finding as until then hamstrings were considered to be too short and were routinely addressed by surgery in children with crouch gait.

According to electromyography studies of normal gait, hamstrings are active in the terminal swing and just after the initial contact during the loading response. Hamstrings, in the terminal swing, produce the hip extensor moment that promotes knee extension and the knee flexor moment that controls and slows that motion [23]. Because the lever arm of the hamstrings at the knee is shorter, compared to its lever arm at the hip, hamstrings are more effective as hip extensors [127]. They play an important role during initial contact when hamstrings inhibit the knee extension and assist in generating extension power at the hip [23]. This study demonstrated both medial and lateral hamstrings to be abnormally long at initial contact in knee recurvatum limbs. For both recurvatum groups these muscles act over a wider range of functional muscle lengths over the gait cycle. As hamstrings inhibit the knee extension at initial contact,

abnormally long hamstrings could result in a knee instability, which could lead to the knee hyperextension. This finding goes hand in hand with the well documented fact that surgical hamstring over-lengthening can cause knee hyperextension in the stance phase of gait [128].

In contrast to Lin's findings [129] and in accordance with Simon's report [102] the outcome of this study showed prolonged activity of the hamstrings during the stance phase of gait. Hamstring muscles of both recurvatum groups shortened approximately 16% during the single support stance phase compared to only 10% shortening for the control group. Moreover, the Early recurvatum group had a shorter mean muscle-tendon length of the hamstrings in the single support stance phase, which points to a more dynamic muscle tightness in Early recurvatum group. The increased hamstring shortening during the stance phase of gait in the recurvatum limbs could represent a compensation for functionally long hamstrings at initial contact. This interpretation is supported by the finding of a prolonged electromyographic activity of the hamstrings for both recurvatum groups.

Study 2

Dynamic versus fixed equinus in cerebral palsy in children with cerebral palsy. How does the triceps sureae work?

The distinction between dynamic calf muscle tightness and a fixed calf muscle contracture is critical in clinical decision making [130;131]. While dynamic tightness might be addressed by conservative treatments [132], fixed muscle contracture represents an indication for surgery [131]. In cerebral palsy increased muscle tone and spasticity make it difficult to asses the muscle-tendon length of a single muscle group in the awake state. While the clinical range of motion testing to detect fixed contractures might have considerable sensitivity, the specificity of the tests is documented to be poor [133]. Therefore, if surgical decision-making had to rely on clinical examination alone, several dynamically tight calf muscles would be evaluated as requiring surgical lengthening procedures.

Ankle kinematics differed between the Dynamic Equinus and the Fixed Equinus group (Study 2, Fig. 1). During the stance phase of gait, normal ankle motion shows a gradual dorsiflexion. Here, ankle motion in the Fixed Equinus group demonstrated persistant equinus and in the Dynamic Equinus group the ankle is even plantarflexing. This abnormal ankle motion in the Dynamic Equinus group relates to early maximal power generation during single stance. In contrast, normal children show ankle power absorption during single limb support. The nearly opposing patterns of ankle kinematics and ankle powers in children with a Dynamic equinus can be interpreted as the effect of an out of phase muscle contraction due to a muscle stretch following a forefoot landing. The Dynamic nature of this contraction is documented by the high muscle-tendon velocity for the triceps surae in the Dynamic Equinus group. For the Fixed Equinus group the kinematic, kinetic and muscle-tendon length parameters illustrate the fixed nature of the equinus. Here, the ankle shows only minor motion and the muscle-tendon lengths remain almost unchanged during single limb support. The muscle-tendon velocity was close to zero. The reduction in ankle motion contributes to a reduction in ankle power generation values that were the lowest of all the three study groups.

Besides the reduction of muscle-tendon lengths in children with cerebral palsy, we feel that the shortening and lengthening muscle-tendon velocity of the triceps surae during gait represents a valuable parameter to evaluate spastic equinus. Our results indicate that all three triceps surae muscles achieve significantly lower peak lengthening velocities in the Fixed Equinus group when compared to the Dynamic Equinus group. Decreased peak lengthening velocity might be an effect of increased muscle stiffness due to an increase in passive tension of the triceps surae muscle fibres. Fridén and Leiber

proved that the mechanical properties of isolated muscle fibers are different in patients with cerebral palsy who developed contractures [134]. Cells of spastic muscles have shorter resting sarcomere lengths and develop passive tension on significantly shorter sarcomere lengths compared to healthy individuals. Another explanation for the decreased peak muscle-tendon lengthening velocity could be a change in the architecture of spastic muscles. Rose et al. demonstrated that spastic muscles in children with cerebral palsy show a predominance of slow-twitch type I muscle fibers and that fiber sizes were different when compared to healthy individuals [135]. Weakness of the dorsiflexor muscles, which is common among children with cerebral palsy who walk with equinus pattern, could represent another factor for decreased peak lengthening velocity [39]. This would, however, not explain the difference in peak lengthening velocity between fixed contracture and Dynamic tightness.

Study 3

Short-term effects of Botulinum Toxin A and serial casting on triceps surae muscle length and equinus gait in children with cerebral palsy

This study utilised three dimensional gait analyses to evaluate effects of BTX-A and serial casting on kinematic and kinetic gait parameters in children with cerebral palsy who demonstrate equinus gait. Short-term effects were analysed ten weeks after BTX-A injections. To evaluate changes in dynamic muscle length, musclulo-skeletal modeling techniques were applied. The results of this study confirm previously reported beneficial short-term outcome when equinus gait is treated with BTX-A and serial casting in children with cerebral palsy. In accordance with other authors [38;42] we observed significant improvements of ankle kinematics and kinetics. These improvements include a reduction in equinus and a change of timing and modulation of ankle motion patterns towards normal. During the “second rocker” of ankle motion we documented improved dorsal-flexion at the end of the stance phase of the gait cycle [130]. Abnormal power absorption was observed whenever a forefoot landing occurred and the ankle was loaded through a forefoot lever. This power absorption can be interpreted as a calf muscle stretch, which is followed by a burst of power generation in the first half of single support. This burst of power generation points towards a spasticity driven stretch reflex after loading the ankle via a forefoot lever arm. When treated with BTX-A and serial casting the reflex induced power generation was markedly reduced. In this study musculo-skeletal modeling revealed no changes in muscle length that could be attributed to the interventions of BTX-A and serial casting. As the changes in kinematics and kinetics cannot be attributed to changes in muscle length, possibly BTX-A affects intrafusal muscle spindles, reducing the effects of the gamma-loop stretch reflex. In a recent study of long-term effects of BTX-A treatment on muscle tone and range of motion in a group of children with cerebral palsy Tedroff et al. [136] proved that BTX-A was efficient in reducing the muscle tone but the improvement of range of motion lasted only for a short period of time in the gastrocnemius muscle. This evidence would support our conclusion that during the first weeks after the application of BTX-A, the therapy might have a more significant influence on muscle spasticity than on muscle growth.

The dose of BTX-A used in this trial (7 IU/kg of BTX-A for the triceps surae complex) is rather high when compared to the literature [137;138]. However, a randomized double blinded trial demonstrated that high-dose BTX-A treatment has a better effect on gait and spasticity improvement than low-dose BTX-A treatment [139]. Similar dosages of BTX-A have been evaluated by other authors [42;113] who did not observe any increase in adverse effects. There is no general agreement on the use of serial casting

together with BTX-A treatment in children with cerebral palsy [140]. It has been shown that serial casting enhanced and prolonged the effect of BTX-A treatment [38;42]. In a more recent study, Newman et al. [141] showed additional benefits in the recurrence of spasticity when casting is delayed after BTX-A injections. Based on these supportive data and our personal experience we adopted this procedure for routine use. The cast is not applied immediately after BTX-A administration but on the same day (approximately four to five hours after injecting BTX-A). This time is used to move and stretch the triceps surae muscle. It has been proved in an animal model that even 10 passive stretch/relax cycles can increase the efficacy of BTX-A [142]. However, to our knowledge no published data could confirm this finding in a group of children with cerebral palsy. On the other hand, electrical stimulation was proved to prolong the gait improvement in children with spastic diplegic cerebral palsy [143].

Management of cerebral palsy is primarily directed towards maintaining or regaining muscle length. Use of BTX-A here is based on the concept that muscle fibres in children with spastic cerebral palsy are short, and weakening the muscles with BTX-A injections might allow them to be stretched and thus grow in length. Therefore the progression from dynamic to a fixed deformity may be delayed [144] and any orthopaedic surgical procedures could be postponed or reduced in frequency [45]. Although muscle growth following the BTX-A injection has also been found in animal models [24;144], the findings of our study did not support this in children with cerebral palsy in a short-term follow up. We speculate that a period of ten weeks is too short to expect any measurable gain in muscle length by muscle growth. However, we observed significant improvements in ankle kinematics and kinetics. We interpret the beneficial changes in kinematics and kinetics observed in this study as an effect of BTX-A on intrafusal muscle fibres [145]. Therefore, the application of BTX-A might have a more significant influence on muscle spasticity than on muscle growth. The BTX-A is effective to reduce the over-activity of the spastic muscle and improve its function [146]. Moreover, even if the main effect of BTX-A might be to weaken the muscle, we did not observe any decrease in power generation in terminal stance, which is the most important factor for forward propulsion in walking. Again, this could be explained by different influences of BTX-A on intrafusal and extrafusal muscle fibres.

Study 4

Detecting postoperative change in children with cerebral palsy: Net nondimensional versus body mass oxygen normalization

Energy expenditure as measured by oxygen consumption can be considered as an objective tool for assessment of functional ability. Fixed selected speeds of 2 $km.h^{-1}$ and 3 $km.h^{-1}$ should be comparable to speeds perceived in the patient's everyday life [30]. While resting oxygen consumption remained unchanged, gross mass relative oxygen consumption decreased and thus efficiency of walking increased. Lower oxygen consumption point out the lower energy demand and thus longer time till fatigue occurs. In contrast to healthy persons, the major reason for the increased energy cost of locomotion in children with cerebral palsy is ineffective gait, due to simultaneous activation of antagonist muscles [30;31]. The preoperative values of VO_2 in our group of children with cerebral palsy are considerably higher in comparison to values of the population of healthy children but similar to previously published data on children with cerebral palsy [61]. Moreover, the comfortable walking speed is more than double in healthy subjects [59].

Because of the strong dependence of gross oxygen consumption on anthropometrical data there is a need for normalization. Gross oxygen consumption relative to body mass ($ml.kg^{-1}$) is the most widely used parameter representing the intensity of physical effort during exercise. The mass relative normalization scheme provides widely accepted but not entirely effective normalization and its performance decreases with increasing height, mass and age [110]. The net nondimensional normalization scheme, using the nondimensional variables described by Hof [105], is essentially independent of all three above mentioned factors. This scheme was shown to perform better than standard mass relative normalization in able-bodied children [110] and it was also used to evaluate gait efficiency in children with cerebral palsy [147]. To our knowledge, it has not been used to detect the improvement of gait efficiency after the soft-tissue surgery in children with cerebral palsy.

Our results demonstrate that the net nondimensional normalization enables a better pre- and postoperative comparison of energy consumption in children with cerebral palsy of different ages and anthropometrical parameters. This was reflected by the lower variability of the net nondimensional VO_2 at both speeds when compared to mass relative VO_2. Moreover, the Spearman rank correlation pre- and postoperatively indicates a close association between the two normalization methods.

Study 5

Evolution of walking ability after soft tissue surgery in cerebral palsy patients. What can we expect?

Evaluating our surgical results in cerebral palsy population we have to be cautious about underestimating the immediate effect of surgical intervention which results in stabilization of a child's gait pattern [148]. The aim of our study was to prove following clinical observation: although we can surgically help our patients to stabilize their walking level or even to improve their gait in long-term run [149-151], there is a period of time up to 6 months just after the surgery [152], when the walking ability deteriorates.

The indications for selection of surgical procedures are often discussed in the literature. It is well documented, that adductor muscles surgical lengthening may be inappropriate in an attempt to reduce excesive internal rotation of the hip [153], but contracture of adductors may be associated with hip lateralisation or even subluxation or luxation of femoral head in spastic cerebral palsy [25]. Specifically the adductor longus, have been noted to have the greatest effect in causing hip dislocation [154]. This paltological sequence may be prevented by surgical release of adductor muscles [155]. In agreement with other authors [150;156] we consider the restriction of abduction more than 30º as a contracture of adductors and we provide adductor muscle tenotomy as a preventive hip surgery, because adductor tenotomy reduces trend towards lateral displacement of the hip joint [157]. Hip flexor contracture is belived to result in gait deviations including restricted stride length, excessive anterior pelvic tilt, increased lumbar lordosis and limited hip extension in stance [158]. Proximal rectus femoris release was initially proposed to reduce hip flexion contracture and lumbar lordosis while improving knee flexion in swing [159]. Inappropriate activity of rectus femoris during swing is belived to contribute to stiff-knee gait in cerebral palsy patients [160]. Distal rectus femoris transfer posterior to the knee was proposed by Perry [161] to treat this gait deviation. This procedure was thought to convert the rectus femoris from knee extensor to knee flexor, but it shows that beneficial effects of rectus femoris tendon transfer are derived from reducing the effects of the rectus femoris muscle as a knee extensor rather than from converting the muscle to knee flexor [101]. Our aim was to address anterior pelvic tilt, hyperlordosis and reduced hip extension by proximal rectus femoris release. Contrary to results of some authors [158] we have a good clinical experience with this procedure which we have already published [162].

At the first follow-up we found a trend to more extension pattern at the hip and the knee which is pointed out as an important mechanism in cerebral palsy patients [148]. These changes allow normal weight acceptance through the ankle and knee. At the knee there was a dramatic improvement towards extension at initial contact and during single support stance phase, but maximal knee flexion in swing decreased. This might be due to decreased walking velocity and power generation during push-off [163]. The push-off range of motion decreased which goes along with slower velocity and step length and implicates a reduction in plantar flexor power. The normalization of foot position in swing, as it was observed in our group of patients is in agreement with Reimers [164], who states that antagonist function improves when spasticity of the agonist is reduced by tendon lengthening.

At the second follow-up (nine month postoperatively) an overall improvement was present. Velocity, step length and cadence increased. Even though parents of our patients reported stability improvement, we did not find any significant change in double support time or step width. This discrepancy might be explained by small sample size in our study. At the hip the increased extension pattern during stance phase was still present and the change in maximum hip extension reached statistical significance. On the contrary to findings of Abel [149], the maximum knee flexion in swing increased back to preoperative level, although the improved knee extension in initial contact and stance remained. Significant improvement was seen in push-off range of movement compared to first follow-up. This parameter reached back the preoperative level and indicates better propulsion. This finding is consistent with findings by Steinwender et al. [131] who did not find any changes in push-off range of motion after surgery and the energy generated at push-off was significantly increased.

Analysis of Center of Mass vertical movement seems to be a good biomechanical factor in monitoring of walking efficacy [82]. We observed a decrease of Center of Mass vertical displacement at first follow-up, but it did not reach statistical significance. However, there was a significant improvement of COM vertical excursion at second follow-up ($p=0.0298$) which indicates better walking efficacy of our patients.

Functional Mobility Scale (FMS) proved an excellent ability to demonstrate a difference between pre- and postoperative state and to detect improvement and deterioration in walking ability of cerebral palsy children [21]. There was no improvement seen after three month period in FMS, but there was found a highly significant increase in parent's rating of children walking ability after nine months.Subjective parent's perception of children's gait improvement goes together with other kinematic data.

Gait analysis in Cerebral Palsy – Pros & Cons

Cerebral palsy is the most common cause of chronic childhood disability. Because of negative effects of upper motoneuron syndrome on developing muscles, they fail to keep pace with growing bones and the occurrence of muscle contractures is the result [24]. These are initially dynamic, but often become fixed over the time. Abnormal muscles produce abnormal forces on the growing skeleton and lead to secondary bony deformities and joint instability [23] (p.185). Further more, the response to these primary and secondary abnormalities may differ among the children. To differentiate between the primary and secondary abnormalities and coping responses is the key for understanding the gait pathology in children with cerebral palsy.

The three-dimensional kinematic and kinetic gait analysis is a widely used tool for research of gait abnormalities as well as for clinical assessment and decision making in children with cerebral palsy. Gait analysis provides an objective record of gait [165]. Clinical gait analysis typically seeks to discriminate between normal and abnormal gait and to assess change in walking over time. Repeated gait measurements can be used to evaluate the response to therapeutic interventions such as surgery, physiotherapy, medications and orthotics. Variability between 'before' and 'after' measurements may be due to treatment effects or measurement variation, or a combination of both. Knowledge of the error magnitude can enable clinical teams to minimise the risk of over-interpreting small differences as meaningful [166]. Therefore the variability represents an absolute prerequisite of validity for any measurement. The sources of variability in the gait analysis are the patients themselves, the motion laboratory equipment, data aquisition and interpretation. Patients with cerebral palsy have been found to have worse intra-subject reliability than healthy children [167]. This facts make the comparison of pre- and post-treatment measurements a bit cumbersome and the interpreter must pay attention to the variation of the parameter measured. The measurement error rising from the technical equipment of the gait laboratory is dependent on the resolution of cameras, their positioning and the the calibrated volume as have been discussed in the theoretical part of the thesis. There is still a need for modifying measurement techniques to reduce levels of error. Many current techniques rely heavily on the skill of assessors in accurately placing markers and inaccurate marker placement is almost certainly the principal source of error. New techniques are now emerging based on functional calibration techniques which are in principle less dependent on the accuracy of marker placement [168]. A recent review of McGinley et al. on reliability of three-dimensional kinematic gait measurement [169] concluded that although most errors in gait analysis are acceptable, they are generally not small enough to be ignored during clinical data interpretation. The highest reliability indices, according this review, occurred in the hip and knee in

the sagittal plane, with lowest errors in pelvic rotation and obliquity and hip abduction. Lowest reliability and highest error frequently occurred in the hip and knee transverse plane. Most studies providing estimates of data error reported values (S.D. or S.E.) of less than 5˚, with the exception of hip and knee rotation. Therefore a goal of any clinical measurement technology must be to provide measurements that are free from any measurement error that might affect interpretation. To ensure this, a quality control and new dynamic joint centering techniques could be the right step forward in the accuracy of measurement. Those, who have already implemented these techniques, report the intertherapist, intersession and intertrial errors of less than 4˚ [166].

Despite of all these limitations and data variability, three-dimensional kinematic and kinetic gait analysis has been proved to be a reliable research and clinical tool which is widely accepted by orthopaedic, biomechanic, neurologic and physiotherapeutic communities. Thanks to the gait analysis we can measure and objectively assess parameters, which can not be visually aspected (joint moments, powers, dynamic muscle length etc.). Exactly these parameters, which distinguish computerized gait from observational analysis, led to a change of treatment philosophy of gait problems in children with cerebral palsy. It was a shift from morphologic way of thinking to a more functional approach to management of cerebral palsy. The example of such a step towards functionality may be the evolution of ankle orthosis. Initially, only rigid ankle-foot orthosis were manufactured to treat the crouch gait. They block and hold the movement in the neutral position. However, such an orthosis not only completely blocks acceleration forces of third rocker and thus any power generated by the calf muscles in the terminal stance is lost, but also interferes with second rocker and plantarflexion/knee-extension couple. Better understanding of ankle function during gait and ground reaction forces applied to the foot led to the development of hinged floor-reaction ankle-foot orthosis which blocks dorsiflexion in single stance but still allowing plantarflexion for propulsion in terminal stance [23] (p.281). Another example of functional approach to cerebral palsy concerning the paediatric orthopaedics, would be the concept of lever-arm dysfunction and its treatment popularized by professor Gage [170].

A common topic of the studies presented in this thesis is the gait analysis in children with cerebral palsy. Although they span from studying the pathobiomechanics of movement (Study 1 and 2) over a application of new normalization methods (Study 4) to clinically orientated question-driven research (Study 3 and 5), they all might have a direct impact on management of children with cerebral palsy. As the gait analysis itself has been developed as a reasearch and clinical tool to allow a better understanding of gait problems, it is important to answer the question: „Does gait analysis alter

treatment decision making?“. The first one, who tried to answer this question in his study was DeLuca et al. [171]. He compared the surgical recommendations for 91 children with cerebral palsy made by clinicians experienced in gait analysis when using information provided from the clinical examination and videotape, with recommendations made after the addition of kinematic, kinetic and electromyographic data. Addition of gait analysis data resulted in changes in surgical recommendations in 52% of the patients with an associated reduction in costs of surgery, not to mention the human impact of an inappropriate surgical decision, which is more likely without gait analysis. Chang et al. in his study addresses the clinical value of the gait analysis when used as a diagnostic tool [172]. He found that patients who complied with gait analysis surgical recommendations were 3.68 times more likely to experience a positive outcome than matched patients who chose not to follow gait analysis recommendations. An important finding was made by Desloovere et al. in her study of the correlation between gait analysis data and clinical measurements [66]. She evaluated the combined predictive value of static and dynamic clinical measurements on gait data of 200 children with cerebral palsy. Only fair to moderate correlations were found between clinical measurements and gait data, the overall highest correlation being 0.60. Multiple regression analysis revealed that adding dynamic clinical measurements (spasticity, strength and selectivity) to a static model enhanced the link between clinical measurements and gait data. The interpretation of this study stresses the fact that gait analysis data cannot be sufficiently predicted by a combination of clinical measurements. The independence of the measurements supports the notion that both, clinical examination and gait analysis data provide important information for delineating the problems of children with cerebral palsy. An interesting finding that documents the benefit of the gait analysis is that of Molenaers et al. who retrospectively reviewed a sample of 424 children with cerebral palsy and found that the introduction of gait analysis increased the age of the first orthopaedic surgical procedure [45].

Obviously, there is not only positive acceptance of the gait analysis. Narayanan [173] in his 2007 review evocates that although gait analysis has been shown to alter decision making, there is a little evidence that the decision based on gait analysis lead to a better outcome and consequently concluded that gait analysis remains controversial. Another criticism of gait analysis in the literature is the paper of Noonan and collegues [174]. To determine the efficacy of gait analysis in the treatment of ambulation problems in cerebral palsy, the authors set out to assess the consistency of both the quantitative data and recommendations for treatment from 4 different motion analysis laboratories. To accomplish this, they sent 11 children to four separate gait analysis centers and then compared the clinical data and treatment recommendations from these centers. They found that gross inaccuracies existed in the clinical data,

with the sagittal plane data being the most reproducible and the transverse plane the least. Treatment recommendations were similar in only 2 of the 11 patients and were widely disparate in the other 9. From this they concluded that traditional methods of physical examination and observational gait analysis were more reliable and that methods other than gait analysis were needed to determine the outcomes of treatment of gait problems in cerebral palsy. The authors speculated about but did not attempt to determine the reasons for the disparate data. The poor agreement on treatment recommendations is an important finding, but it relates more to the opinions on treatment strategy of particular surgeons than to the gait analysis data itself. The interpretation of the gait analysis data is subject of individual variability similar to the interpretation of electrocardiogram. In both papers the authors underline the variability of gait analysis data. This topic has been discussed earlier in the text and I would therefore only note that the reliability of gait analysis data has been found to be acceptable [169] keeping in mind its technical limitations. Futhermore, since the Narayanan's 2007 review new data supporting the value of gait analysis have been published. Filho et al. showed in his study [175] that the patients whose treatment matched the recommendations from three-dimensional gait analysis showed a more significant improvement in walking. Moreover, gait analysis prooved to be helpful in distinguishing children with bilateral spastic cerebral palsy who would benefit from surgery from those in whom nonoperative management is appropriate [176]. Therefore its routine clinical use is encouraged in the management of children with cerebral palsy.

Studies presented in this thesis enhance the up-to-date knowledge in the field of pathophysiology of movement disorders in children with cerebral palsy. Based on the literature review, our team was the first one to introduce the three-dimensional gait analysis in children with cerebral palsy in the Czech Republic. In cooperatin with the Medical University of Graz and University Duisburg-Essen, Institute of Mechatronics and System Dynamics new technique of the dynamic muscle length modeling was introduced. Outcomes of our clinical studies have a direct implication in the treatment strategy of children with cerebral palsy. As cerebral palsy is the most common cause of chronic childhood disability and motor disorders are the leading sign of this syndrome, the work presented here might be of interest for a wide spectrum of audience including paediatric orthopaedic surgeons, neurologists, physiotherapists, rehabilitation doctors and biomechanical engineers.

Summary of main outcomes:

Study 1 – Genu recurvatum in children with cerebral palsy

This study used gait analysis and musculoskeletal modeling to provide the link between the type of equinus deformity and the timing of knee hyperextension in children with cerebral palsy. Early knee recurvation is caused by a dynamic equinus deformity and the late knee recurvation is linked to the fixed equinus deformity. Such a finding might simplify the decision as to which treatment to select for equinus deformity, present in patients with genu recurvatum.

Semimembranosus, semitendinosus and the long head of biceps femoris are abnormally long at initial contact and operate over a wider range of muscle-tendon lengths in patients with cerebral palsy who developed knee recurvatum. Functionally long hamstrings might result in some degree of knee joint instability and could cause its hyperextension. This interpretation is supported by the clinical observation that surgical over-lengthening of hamstrings leads to knee hyperextension in children with cerebral palsy. In our opinion, abnormally long hamstrings together with equinus position of the foot at initial contact are the main causes of genu recurvatum in children with spastic cerebral palsy.

Study 2 - Dynamic versus fixed equinus in children with cerebral palsy. How does the triceps sureae work?

The distinction between a dynamic and a fixed equinus was based on an examination under general anaesthesia. This allowed the evaluation of kinematics, kinetics and calf muscle-tendon length parameters in well defined groups. Characteristic changes in ankle joint kinematics and abnormal kinetic parameters were found for both types of equinus. The rate of change of muscle-tendon length might be a promising parameter to distinguish between fixed and dynamic equinus gait in children with cerebral palsy.

Study 3 - Short-term effects of Botulinum Toxin A and serial casting on triceps surae muscle length and equinus gait in children with cerebral palsy

The BTX-A is effective in reducing the over-activity of the spastic muscle and improving its function. We did not observe any change in the peak muscle-tendon length of the gastro-soleus muscle complex. The application of BTX-A might have a more significant influence on muscle spasticity than on muscle growth. As the changes in kinematics and kinetics cannot be attributed to changes in muscle length, possibly BTX-A affects intrafusal muscle spindles, reducing the effects of the gamma-loop stretch reflex. The study

demonstrated beneficial functional changes in ankle kinematics and kinetics after the application of BTX-A when combined with serial casting.

Study 4 - Detecting postoperative change in children with cerebral palsy: Net nondimensional versus body mass oxygen normalization

The net nondimensional VO2 was able to detect the postoperative improvement of gait efficiency with smaller variability compared to standard mass relative normalization. Because of its independance on age, weight and height it might be recommended in studies concerning children with cerebral palsy.

Study 5 - Evolution of walking ability after soft tissue surgery in cerebral palsy patients. What can we expect?

We found the soft-tissue surgery to be an effective method for correction of contracted muscle deformities. Although six months period is reported to be a sufficient time for recovery after soft tissue surgery, some of the important gait parameters (push-off range of motion at ankle joint) did not recover to preoperative level until nine months postoperatively. Despite the normalization of range of motion immediately after operation, there is an obvious period of deterioration after a corrective surgery in cerebral palsy patients and we have to inform our patients about this fact in advance. Our results also highlight the importance of postoperative physiotherapy and the necessity of muscle and gait training programs after soft-tissue gait improvement surgery.

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Appendix

STUDY 1

- Švehlík Martin, Ernst B. Zwick, Gerhardt Steinwender, Vinay Saraph, Wolfgang E. Linhart. Genu Recurvatum in Cerebral Palsy – part A: Influence of dynamic and fixed equinus deformity on timing of knee recurvatum in children with cerebral palsy. *Journal of Pediatric Orthopaedics, Part B*, **IF 0,734 –** accepted for publication
- Švehlík Martin, Ernst B. Zwick, Gerhardt Steinwender, Vinay Saraph, Wolfgang E. Linhart. Genu Recurvatum in Cerebral Palsy – part B: Hamstrings are abnormally long in children with cerebral palsy showing knee recurvatum. *Journal of Pediatric Orthopaedics, Part B*, **IF 0,734 –** accepted for publication

STUDY 2

- Švehlík Martin, Ernst B. Zwick, Gerhardt Steinwender, Tanja Kraus, Wolfgang E. Linhart. Dynamic versus Fixed Equinus Deformity in Children with Cerebral Palsy: How does the triceps surae muscle work?. Archives of Physical Medicine and Rehabilitaion, 2010 – **IF 2,159 –** submitted

STUDY 3

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STUDY 1 – Part I

Genu Recurvatum in Cerebral Palsy

Influence of dynamic and fixed equinus deformity on timing of knee recurvatum in children with cerebral palsy

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Abstract

The aim of the study was to confirm the hypothesis of the influence of the dynamic and fixed equinus deformity on timing of knee recurvation (hyperextension). According to our hypothesis, dynamic equinus is linked to early and fixed equinus to late knee hyperextension. A group 35 children with CP (47 lower limbs) was divided into two subgroups according to the timing of maximum knee hyperextension. Clinical examination confirmed our hypothesis. The gait analysis and musculoskeletal modelling results were compared to 12 normally developing children. Both recurvatum groups had forefoot landing and neither achived normal ankle dorsiflexion. EMG examination revealed a abnormally high soleus activity in single stance. Muscle length changes of medial gastrocnemius and soleus were in agreement with our hypothesis. Such a finding might simplify the decision as to which treatment to select for equinus deformity, present in patients with genu recurvatum.

Introduction

Hyperextension of the knee in the stance phase of the gait cycle (knee recurvatum) is a common disorder of the knee function present in patients with stroke or traumatic brain injury [1], poliomyelitis [2] and patients with cerebral palsy [3]. Knee recurvatum is not only a pathological kinematic pattern but produces significantly increased peak extensor torque across the knee [2], placing the capsular and ligamentous structures of the posterior aspect of the knee at risk of injury, possibly causing pain, ligamentous laxity [4], or bony deformity [5]. Recurvatum has also been shown to increase energy consumption because of the interruption of the smooth transfer of potential energy to kinetic energy [6,7]. Simon, based on the results of kinematic analysis of children with cerebral palsy, postulated two different groups of the knee recurvatum [3]. In the first group (Early recurvatum) recurvation occurred within the first half of the stance phase and in the second group during the second half (Late recurvatum). The timing of peak recurvation has been shown to be an important predictor of the knee extensor torque. Late stance knee recurvatum patients have significantly lower extensor torque compared to the early recurvation group [2].

There are several contributing factors to knee recurvatum: triceps surae spasticity or weakness, quadriceps spasticity, anterior trunk lean [3], weakening of hamstrings [8], or bony deformities of the proximal tibia [5]. The primary cause mentioned in every study is the equinus position of the foot. The role of the triceps surae and the importance of the musculus soleus in the timing of recurvation has already been mentioned in the literature [9]. Hullin et al. postulated that early hyperextenders have a relatively tighter soleus than late hyperextenders. We agree that it is the soleus muscle which plays an important role in the timing of the peak hyperextension. However based on our clinical knowledge, it is early knee recurvation which is caused by a dynamic equinus deformity, and late knee recurvation is linked to the fixed equinus deformity. These two distinct forms of equinus possibly point to a need for different treatment strategies, and therefore such a differentiation would have direct clinical implications. The aim of our study was to evaluate our clinical hypothesis. Kinematic and kinetic gait analysis together with electromyography and musculoskeletal modelling techniques were used to describe and distinguish between the different behaviour of gastrocnemius and soleus muscles with regard to timing of the peak hyperextension in children with cerebral palsy.

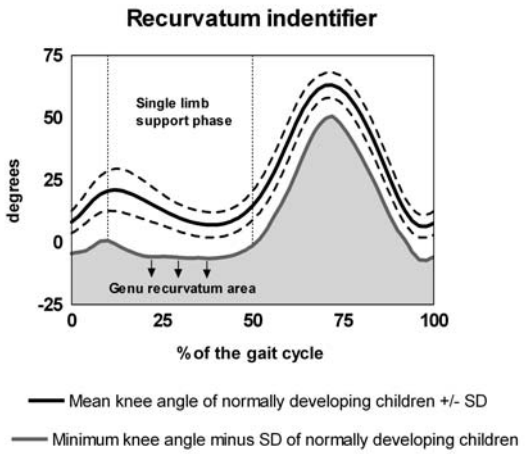

Figure 1. The "Recurvatum identifier" represents the graphical definition of knee recurvatum in the present study.The black and dashed lines represent the normal values ± one standard deviation of the knee angle over the gait cycle in a group of normally developing children. The vertical dotted lines define the single support stance phase of the gait cycle during which the knee recurvation occurred. The grey area represents the minimal knee angle minus one stanard deviation in normally developing children. Whenever knee angle values of a cerebral palsy child fell below this reference during any time of the single support stance phase, the limb was considered to have the knee recurvatum.

Methods

Subjects

Knee recurvatum was defined as a knee angle showing more extension than a reference knee angle during the stance phase of the gait cycle. In our study The reference knee angle was obtained from a group of normally developing children. Within this group the most pronounced knee extension minus one standard deviation was calculated for every percentage point of the gait cycle forming the reference knee angle (Fig. 1). This reference knee angle ("recurvatum indentifier") was used as a cut-off to select the patients with knee recurvatum.

The inclusion criteria were: spastic diplegic or hemiplegic form of cerebral palsy, the knee recurvatum as defined above, the GMFCS level of I-II, no prior triceps surae surgery, no application of Botullinum toxin A within the last 6 months, the full set of kinematic and kinetic data available. Thirty-five children (21 boys and 14 girls) were included in this retrospective study. Twelve were diplegic and twenty-three hemiplegic. The recurvatum group of 47 extremities was divided into two subgroups according to a timing of the peak hyperextension [3]. The group demonstrating early recurvatum (group "E-REC") (14 extremities) had an average age of 8.01±2.7 years, and was defined to have a peak hyperextension during the

first half of the stance phase. The group showing late recurvatum (group "L-REC") consisted of 33 extremities with an average age of 7.62±2.36 years, and had the peak hyperextension in the second half of the stance phase. A group of 12 normally developing (group "Norm") children (24 extremities) without any known neurologic or orthopaedic condition influencing the gait served as a reference collective. The average age of the Norm group was 10.32±2.96. The type of equinus deformity was examined clinically. If the foot could not be dorsiflexed beyond the neutral position with the knee fully extended, the equinus was considered to be fixed. If the child had an equinus gait and the foot could be dorsiflexed beyond the neutral position with the knee fully extended, the equinus was defined to be dynamic. The Ethics Committee of the local medical University approved the study.

Gait analysis

Computerized gait analysis was performed using a six-camera, video-based motion capturing system (Vicon 370, Oxford Metrics, Oxford, U.K.) and two floor-mounted force plates (Kistler Instruments Limited, Winterthur, Switzerland). Thirteen reflective markers were used to define the pelvis and lower extremity segments in accordance with the Vicon Clinical Manager model (Oxford Metrics, Oxford, U.K.). Moment and power parameters were normalized to the weight of the patients. Power generation and absorption patterns in the sagittal plane were calculated and labelled according to the method described by Winter et al. [10]. All patients walked at self-selected speeds along a 12-meter walkway. For each patient a minimum of five trials providing a clear foot force plate contact were captured and averaged.

Muscle length calculation

Based on kinematic data, Mobile GaitLab software (University Duisburg-Essen, Institute of Mechatronics and System Dynamics, Germany) [11,12] was used to compute individual muscle lengths. The specification of the lower extremity architecture was based on the origin and insertion coordinate data compiled by Yamaguchi [13]. The model defines the musculoskeletal geometry of a normal adult male. Three dimensional musculo-tendon (MT) pathways were modelled as a series of straight-line segments extending from the origin to insertion. Where appropriate, the MT pathways were sub-sectioned into a number of straight-line segments by introducing via-points. Musculo-tendon length parameters were normalized to allow the comparison among subjects. To describe the different patterns of muscle length changes during the stance phase, a so-called "slope quotient" was introduced. The slope quotient was computed as a subtraction of the muscle length at the beginning of single support from the muscle length at the end of the single support. Positive values of the slope quotient would therefore indicate muscle elongation, negative values muscle shortening and values near to zero would indicate that the muscle stays of the same length during the whole single support phase of the gait cycle.

Electromyography

Surface electromyograms were obtained from the medial gastrocnemius and soleus of 5 children of E-REC group (3 diplegic and 2 hemiplegic) and 10 children of L-REC group (3 diplegic and 7 hemiplegic) included in this study. Raw electromyograms were recorded in a frequency range between 25 and 400 Hz (Noraxon, Myosystem 2000). After AD-conversion and sampling at 1 kHz, the signals were fully wave rectified and digitally low-pass filtered at 6 Hz. After time normalisation, a minimum of 12 gait cycles were averaged to create an average linear envelope for each muscle. Calculation methods applied have been described in detail by Yang and Winter [14].

Statistics

One-way ANOVA with Fischer post-hoc test (Statistica 6.0, StatSoft, Tulsa, OK, USA) was used for comparisons between the controls, and the early and late recurvatum groups. The significance level was set to $p<0.05$. Electromyographic data were evaluated and described by means of descriptive statistics. Kinematics and kinetics in the sagittal plane, as well as the lengths of the medial gastrocnemius and soleus muscles over the gait cycle were considered as main outcome measures for this study.

Results

The recurvatum groups were age-matched but not sex-matched and walking velocities were not statistically different between the groups. Children with CP and recurvatum walked slower, had a shorter sride length and longer stance time in comparison to normally developing children (Tab. 1). However, there was no difference between the E-REC and L-REC group. The maximal knee recurvation of the E-REC group on average occurred at 25% of the gait cycle compared to 39% of L-REC group (Fig. 4). Based on the clinical evaluation, all 14 extremities from the E-REC group were assessed as dynamic equinus deformity and

26 of 33 extremities from the L-REC group were assessed to be fixed.

Kinematics, kinetics and EMG

There was no difference in ankle angle at initial contact and both groups had a forefoot landing (Tab. 1). Although, both groups rapidly dorsi-flexed the ankle up to a mild dorsi-flexion during the loading response, dorsi-flexion stopped at 12% of the gait cycle in the E-REC group and then the foot moved back to plantar-flexion (Fig. 2). The L-REC group showed an arrest in tibia advancement at about 17% of the gait cycle and remained in a mild dorsi-flexion over the whole stance phase (Fig. 3). None in either group were able to achieve normal dorsi-flexion during stance (Fig. 4). There was no significant difference in the ankle moments when comparing E-REC and L-REC. However, compared to group Norm both the REC groups had lower and earlier maximum of the ankle moment. The L-REC had higher power absorption during the first double-support phase of the gait cycle. The maximum power generation was not different between the REC groups but power production at the end of the stance did not reach the level of the Norm group. Electromyographic evaluation of muscle activation patterns revealed no differences between the REC groups in gastrocnemius activity. Both groups had an early high activity in the first double support followed by abnormally high activity during the single stance phase. Also pathological swing phase activity of the gastrocnemius occurred in both groups. A similar phasic activity was observed for the soleus muscle in both groups but only the E-REC group demonstrated an abnormally high activity during the single stance phase ($p=0.042$).

Muscle-tendon length simulation

Muscle-tendon length parameters were different among the three groups. The medial gastrocnemius in the E-REC group elongated rapidly to its maximum during the first double-support phase (aproximatelly the first 10% of the gait cycle). It subsequently shortened gradually during the rest of the stance phase. In contrast, the medial gastrocnemius of L-REC group gradually elongated to its maximum length that was reached at 46% of the gait cycle (Fig. 4). The maximal lengths of the medial gastrocnemius during the stance phase of gait were similar between the groups. At toe-off, the medial gastrocnemius length for L-REC group was markedly longer when compared to the E-REC and the Norm groups. Medial gastrocnemius and soleus operated over a smaller range of muscle lengths in both REC groups (Table 2).

The soleus muscle of the E-REC group elongated quickly from initial contact to its peak at 11% of the gait cycle. At the start of single limb support, the muscle shortened and remained at a constant length during the rest of the single stance phase. The soleus in the L-REC group elongated slowly to its maximal length and did not change over the single stance phase (Fig. 4). The maximal lengths of the soleus were not different between the REC groups. The range of soleus lengths was smaller in the L-REC group (Table 2).

The differences in the patterns of muscle length changes during the single support were described by the slope quotient of muscle lengths. While positive values were obtained for the medial gastrocnemius and soleus muscles in the Norm group, the L-REC group showed slope quotient values close to zero and for the E-REC group negative values were calculated. Negative values indicated muscle shortening during the single support phase (Table 2).

Discussion

Knee hyperextension during the stance phase of the gait cycle is commonly observed in children with cerebral palsy. Children with cerebral palsy often have poor motor control at the ankle. In all CP children included in this study, knee recurvatum occurred secondary to abnormal movement of the tibia during the stance phase. Our results, based on the musculoskeletal modelling, show two distinct patterns of the triceps muscle behaviour during the stance phase of the gait cycle.

In the Early Recurvatum group (E-REC), tibia advancement over the planted foot is suddenly stopped by an exaggerated stretch reflex [15] of the soleus muscle, and the foot is brought back to equinus position. Such a spastic “catch-up” is clear evidence of muscle spasticity and the dynamic nature of the equinus deformity in the E-REC group. This behaviour of the soleus is further confirmed by the electromyographic hyperactivity, present throughout the single stance phase, and was the only statistically significant difference in EMG activity between the REC groups. More evidence of the dynamic nature of equinus in E-REC group is given by the clinical examination, which agrees with our hypothesis.

The gastrocnemius of E-REC elongates, similarly to the soleus, to its maximal length during the loading response. Because the gastrocnemius is one of the knee stabilizers, a rapid elongation might add to an existing instability to the knee joint, and the gastrocnemius could then not act as a restraint [9].

The rapid elongation of the gastrocnemius medialis might also support the soleus muscle in its pathological function of pulling the tibia backwards. The gastrocnemius muscle's rapid elongation to its maximum length could also be indication of weakness which has been reported as one of the causes of knee recurvatum [3,4,16]. In our study this is only an indirect sign of weakness obtained from gait analysis and we do not have clinical data to back up this finding.

The triceps surae muscle of the Late Recurvatum (L-REC) group demonstrated an entirely different pattern of action. The soleus muscle slowly elongates to its maximal length and then stops the tibia during forward progression. It then remains at a constant length during the whole single stance phase. The soleus and the medial gastrocnemius have a smaller range of dynamic muscle lengths. This could be interpreted as a sign of increased muscle stiffness. Increased muscle stiffness would be expected in the presence of a fixed equinus deformity. The dynamic behaviour of the gastrocnemius muscle in the L-REC group showed a pattern closer to normal. Perhaps a good parameter to describe the dynamic action of the soleus and the gastrocnemius muscles in the single stance phase is the slope of muscle length. The slope quotient demonstrates that while triceps surae muscles in the E-REC group contract and shorten, they remain at a constant length, or show a slight elongation in the L-REC group. As walking velocity was comparable for both recurvatum groups, the group differences observed in musculoskeletal modelling should not be biased by this factor.

In a study of children with hemiplegic cerebral palsy, Hullin et al. proposed that the difference between the two groups of recurvatum patients is the degree of soleus tightness – early hyperextenders have a relatively tighter soleus than late hyperextenders [9]. In contrast to his observation, our results clearly indicate that early knee recurvatum is more likely associated with dynamic equinus deformity. A smaller range of dynamic muscle lengths of the medial gastrocnemius and soleus muscles in the L-REC is to be considered as a sign of the fixed contracture. Moreover, there is a 85,8% agreement between the fixed equinus deformity assessed by clinical examination and the L-REC group.

There are some limitations of our study to be considered. First, the musculoskeletal model used in this study has several limitations. The Mobile GaitLab utilizes musculoskeletal data of a normal adult male [13,17]. However, similar models have also been used as an outcome measure in studies on children with cerebral palsy [18,19]. The use of an adult model should not affect normalized muscle lengths [20]. The second limitation concerns the retrospective nature of our study. Unfortunately, electromyographic data were not available for all patients. Also some additional clinical data, e.g. triceps surae testing under general anaesthesia to distinguish between the fixed and dynamic equinus deformities, would enhance the power of our study. Another consideration should be that children with cerebral palsy cannot be assumed to have normal bone architecture, osseous deformities could alter muscle attachment points and muscle paths. Schutte showed the effect of femoral anteversion on psoas muscle length [21]. To our knowledge, no study has yet determined the influence of bone deformities (tibial torsion, ankle varus/valgus) on the calculation of triceps surae muscle length.

Treatment options for knee recurvatum range from physiotherapy, ankle-foot orthoses [22,23], and electrogoniometric feedback [4], to the application of Botulinum toxin A [24] and surgery [15]. Our results might have a direct clinical implication for the treatment of knee recurvatum. While dynamic tightness of the triceps surae muscle might be addressed by conservative treatments [25], fixed muscle contracture represents an indication for surgery [26]. We believe that surgical correction of equinus deformity in the presence of knee hyperextension should focus on the soleus muscle, which seems to play an important role in the inducing of the stance knee extension [27]. The Baumann [28] procedure, which permits selective lengthening of the gastrocnemius and soleus, could be a feasible solution.

The timing of the peak hyperextension is easily distinguishable by observational gait analysis. As gait analysis and musculoskeletal modelling techniques are not always available in the clinical setting, an association between the timing of the peak knee hyperextension and the type of the equinus deformity could help to determine appropriate treatment in children with cerebral palsy who have developed knee recurvatum.

In conclusion, this study used gait analysis and musculoskeletal modelling to provide the link between the type of equinus deformity and the timing of knee hyperextension in children with cerebral palsy. Such a finding might simplify the decision as to which treatment to select for equinus deformity, present in patients with genu recurvatum.

	E v L REC	E v Norm	L v Norm	E-REC		L-REC		NORM	
	p-level	p-level	p-level	Mean	SD	Mean	SD	Mean	SD
Cadence (s^{-1})	0.106	0.429	0.343	126.265	37.940	143.128	38.395	131.022	14.711
Velocity (m/s)	0.459	**0.002**	**0.002**	1.001	0.373	1.057	0.212	1.288	0.151
Stride length (m)	0.863	**0.000**	**0.000**	0.929	0.126	0.937	0.141	1.189	0.164
Stance Time (% of GC)	0.602	**0.002**	**0.000**	61.555	2.480	61.953	2.710	59.074	1.803
Ankle angle at IC	0.548	**0.000**	**0.000**	-7.424	8.051	-6.003	9.110	3.173	2.991
Maximum dorsiflexion of the ankle	0.237	**0.000**	**0.000**	0.676	7.656	3.422	8.957	14.802	3.028
Maximum plantarflexion of the ankle	0.463	0.190	0.437	-20.383	10.654	-18.214	10.547	-16.284	5.646
Maximum ankle moment	0.190	**0.000**	**0.001**	0.919	0.249	1.026	0.293	1.252	0.186
Ankle power generation at the beginning of single suport	**0.005**	0.942	**0.001**	-0.164	0.477	-0.787	0.935	-0.147	0.112
Ankle maximum power generation	0.174	**0.000**	**0.000**	0.754	0.296	0.990	0.629	3.275	0.509
Timing of ankle maximum power generation	0.839	0.077	**0.049**	45.214	15.177	46.061	16.319	53.083	1.248
Knee angle at toe-off	**0.001**	**0.001**	**0.000**	29.852	5.933	23.849	5.921	36.371	5.088
Maximum knee flexion angle	0.708	**0.002**	**0.001**	54.234	9.124	55.183	9.369	62.741	4.022
Timing of maximum knee flexion angle	0.323	**0.000**	**0.000**	78.214	4.458	79.333	4.044	71.417	1.666
Minimum knee angle at stance	0.992	**0.000**	**0.000**	-3.795	3.098	-3.807	3.753	5.013	4.536
Maximum hip flexion ange	0.637	**0.029**	**0.028**	40.823	8.820	39.647	8.997	34.963	4.794
Minimum hip angle	0.082	0.662	0.127	-8.002	7.630	-12.313	7.820	-9.135	7.473
Mean pelvic tilt over the GC	0.754	**0.023**	**0.014**	14.842	6.848	14.215	6.831	9.970	4.872

Table 1
Summary of ANOVA statistic results with Fisher post-hoc tests on time-distance, kinematics and kinetics parameters of the Early (E-REC) and Late (L-REC) Recurvatum groups compared to a group of normally developing children (Norm). Statistically signifacant results are in bold. (IC=Initial Contact. GC=Gait Cycle)

	E v L REC	E v Norm	L v Norm	E-REC		L-REC		NORM	
	p-level	p-level	p-level	Mean	SD	Mean	SD	Mean	SD
MG at toe-off	**0.000**	0.286	**0.000**	**95.571**	2.050	**98.076**	1.407	**96.179**	0.673
MG maximum length	0.173	0.427	0.363	**103.077**	1.026	**102.572**	0.943	**102.782**	0.555
MG range of muscle lengths	**0.014**	0.439	**0.007**	**8.206**	2.424	**6.131**	1.968	**7.567**	1.395
MG slope quotient	**0.013**	**0.000**	**0.043**	**-1.530**	3.292	**0.906**	2.444	**2.128**	1.085
SOL at toe-off	**0.001**	0.112	**0.003**	**94.420**	3.080	**97.364**	2.125	**95.757**	0.776
SOL maximum length	0.256	0.308	0.858	**103.586**	1.934	**103.031**	1.092	**103.085**	0.713
SOL range of muscle lengths	0.060	0.499	0.054	**9.753**	4.636	**7.488**	2.506	**8.944**	2.034
SOL slope quotient	**0.022**	**0.000**	**0.004**	**-2.943**	5.051	**0.171**	3.321	**2.616**	1.392

Table 2. Normalized muscle-tendon length parameters of medial gastrocnemius and soleus. Summary of ANOVA statistic results with Fisher post-hoc tests of the Early (E-REC) and Late (L-REC) Recurvatum groups compared to a group of normally developing children (Norm). Statistically signifacant results are in bold. (MG = medial gastrocnemius. SOL = soleus)

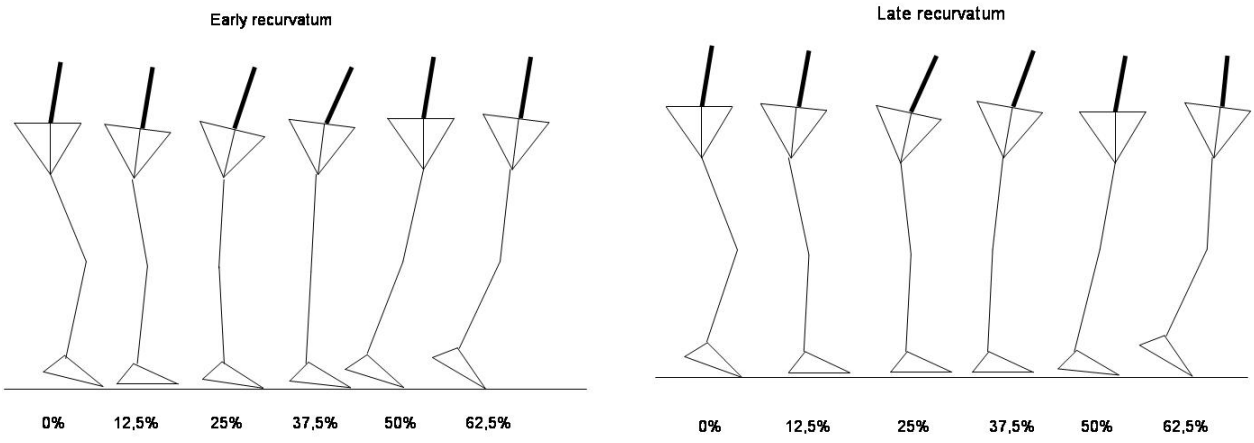

Figure 2. Ilustration of the stance phase of the gait cycle in Early Recurvatum group. The difference compared to the Late Recurvatum group is not only to be seen in the timing of the knee hyperextension but also the equinus position of the foot in the stance phase of the gait cycle.

Figure 3. Ilustration of the stance pahse of the gait cycle in Late Recurvatum group. There is not only late timing of the maximum knee hyperextension but the foot is in the neutral position during the whole single limb support.

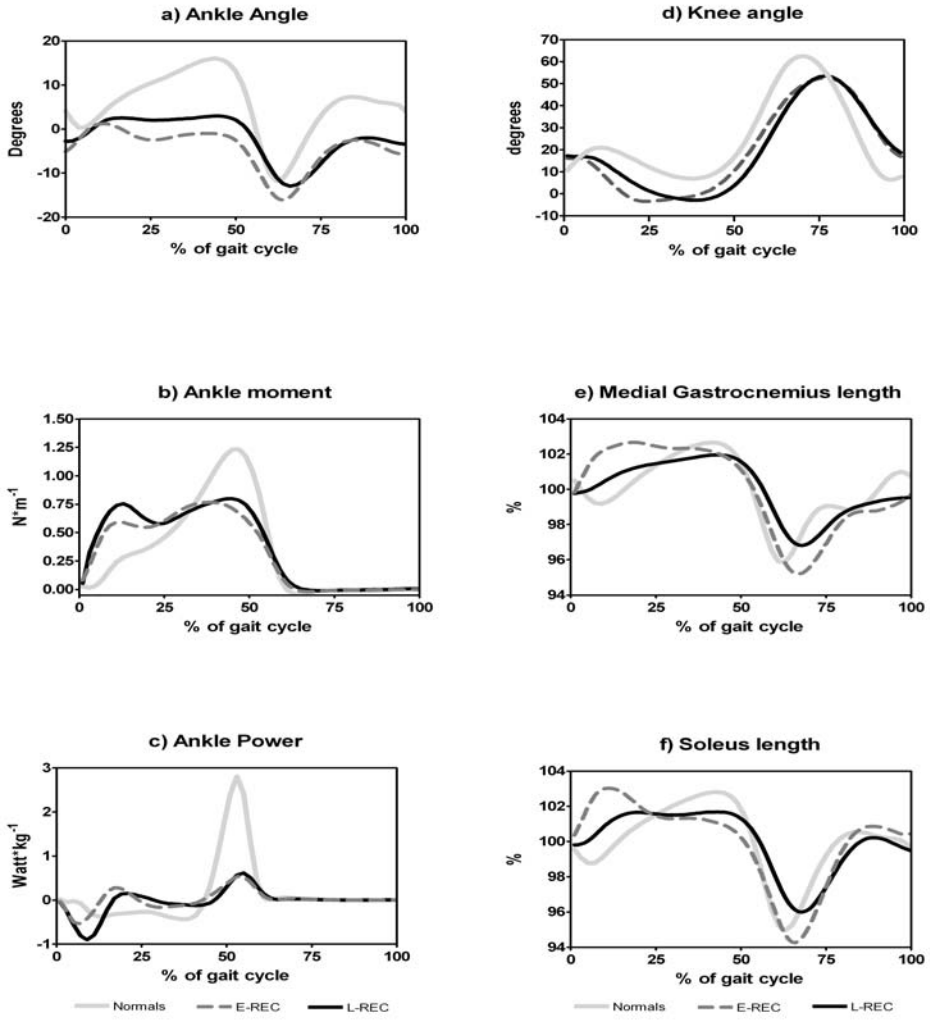

Figure 4. Time-distance parameters, kinematics, kinetics and normalized muscle-tendon lengths of the Early (E-REC) and Late (L-REC) Recurvatum groups compared to a group of normally developing children (Norm). The vertical lines define the single stance phase of the gait cycle for each recurvatum group.

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STUDY 1 – Part II

Genu Recurvatum in Cerebral Palsy

Hamstrings are abnormally long in children with cerebral palsy showing knee recurvatum

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Abstract

Hyperextension of the knee in stance (knee recurvatum) is a common disorder in patients with spastic cerebral palsy (CP). A group 35 children with CP (47 lower limbs) was divided into two subgroups according to the timing of maximum knee extension during the stance phase of gait. Gait analysis and musculoskeletal modelling data were compared to a control group of twelve normally developing children. We observed no difference in kinematics between the CP groups who demonstrated an equinus position of the foot at initial contact. Both groups demonstrated increased external extensor moments across the knee. The muscle-tendon lengths of the hamstrings were abnormally long at initial contact, and in both recurvatum groups, contracted faster compared to the control group. Surface electromyography revealed prolonged activity of the hamstrings in stance and early activation in swing. Abnormally long hamstrings at initial contact together with equinus position of the foot are the main causes of genu recurvatum in children with CP.

Introduction

Gait is a complex pattern of movement and gait pathology is common in children with neurological impairment. In the stance phase of the gait cycle the knee serves three principal functions: transverse rotation, shock absorption, and energy conservation [1]. Hyperextension of the knee in the stance phase of the gait cycle (knee recurvatum) is a common disorder of the knee function in patients after stroke or traumatic brain injury [2], poliomyelitis [3], and also in children with cerebral palsy [4]. There are several causes of knee recurvatum described in the literature: triceps surae spasticity or weakness, contracture of tendo Achilles, quadriceps spasticity, anterior lean of the trunk [4], bony deformities of the proximal tibia [5], ligament or capsular laxity at the knee [6], weakness of hamstrings [7], or it may develop after the distal lengthening of the hamstrings [8]. Knee recurvatum leads to an increased external extensor moment across the knee [4], placing the capsular and ligamentous structures of the knee at risk of injury [3]. When a significant hyperextension of the knee is ignored during growth, a growth disturbance of the proximal tibia may lead to severe instability of the knee [9]. Also, due to an increased vertical excursion of the center of mass, knee recurvatum increases the energy consumption during walking [7]. The treatment options for knee recurvatum range from physiotherapy, electro-goniometric bio-feedback [10], ankle foot orthoses [6], application of Botulinum-toxin A [11] to orthopaedic surgery [7].

Hamstrings are important stabilizers of the knee. They play a crucial role during initial contact by limiting knee extension. Their role in crouch gait has been extensively studied and Delp et al. have shown that 80% of cerebral palsy subjects with crouch gait have hamstrings of normal length or longer [12]. This was an interesting finding as surgical lengthening of the hamstrings is a common treatment of crouch gait. It is well documented in the literature that surgical over-lengthening of hamstrings can lead to the development of genu recurvatum [8]. Surprisingly, the role of hamstrings in children with cerebral palsy showing knee recurvatum has not been evaluated in detail.

Simon et al. [4] described the two distinct groups of knee recurvatum based on the timing of maximum knee hyperextension in stance. Since that time, there is a lack of literature concerning with the pathophysiology of the knee recurvatum. The aim of this study was to analyze the role of hamstrings in the pathobiomechanics of two distinct groups - Early and Late Recurvatum. Kinematic and kinetic parameters, surface electromyography, and musculoskeletal modelling techniques were applied to evaluate pathobiomechanics of the knee recurvatum and the role of hamstrings.

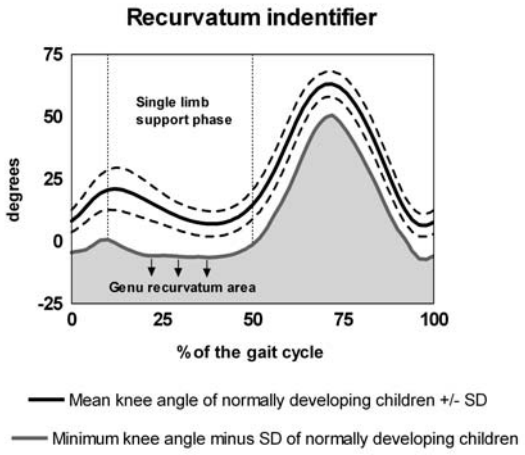

Figure 1. The grey area represents the graphical definition of the knee recurvatum in the present study.

Methods

Subjects

Knee recurvatum was defined as a knee angle showing more extension than a reference knee angle during the stance phase of the gait cycle. The reference knee angle in our study was obtained from a group of normally developing children. Within this group the most pronounced knee extension minus one standard deviation was calculated for every percentage point of the gait cycle to determine the reference knee angle (Fig. 1). This reference knee angle ("recurvatum identifier") was used as a cut-off to select the patients with knee recurvatum.

The inclusion criteria were: spastic diplegic or hemiplegic form of cerebral palsy, knee recurvatum as defined above, GMFCS level of I-II, no prior triceps surae or hamstrings surgery, the full set of kinematic and kinetic data available. Following the above-mentioned criteria thirty-five children (21 boys and 14 girls) were included in this study. Twelve children had diplegic cerebral palsy, twenty-three the hemiplegic type. This collective of 47 extremities was divided into two subgroups according to the description of Simon on the timing of peak recurvation [4]. The Early Recur**vatum** group (group "E-REC") (14 extremities) had an average age of 8.01±2.7 years, and was defined to have a peak recurvation during the first half of the stance phase. The Late Recurvatum group (group "L-REC") consisted of 33 extremities with an average age of 7.62±2.36 years, and had the peak recurvation in the second half of the stance phase. A group of 12 normally

developing children (24 extremities) without any known neurologic or orthopaedic condition influencing their gait was used as a reference collective (group "Norm"). The average age of the "Norm" group was 10.32±2.96 years. The Ethics Committee of the local University approved the retrospective study.

Gait analysis

Computerized gait analysis was performed using a six-camera, video-based motion capturing system (Vicon 370, Oxford Metrics, Oxford, U.K.) and two floor-mounted force plates (Kistler Instruments Limited, Winterthur, Switzerland). Thirteen reflective markers were used to define the pelvis and lower extremity segments in accordance with the Vicon Clinical Manager model (Oxford Metrics, Oxford, U.K.). Kinetic parameters were normalized to the weight of the patients. All patients walked at self-selected speeds along a 12-meter walkway. For each patient a minimum of five trials providing a clear foot force plate contact were captured and averaged.

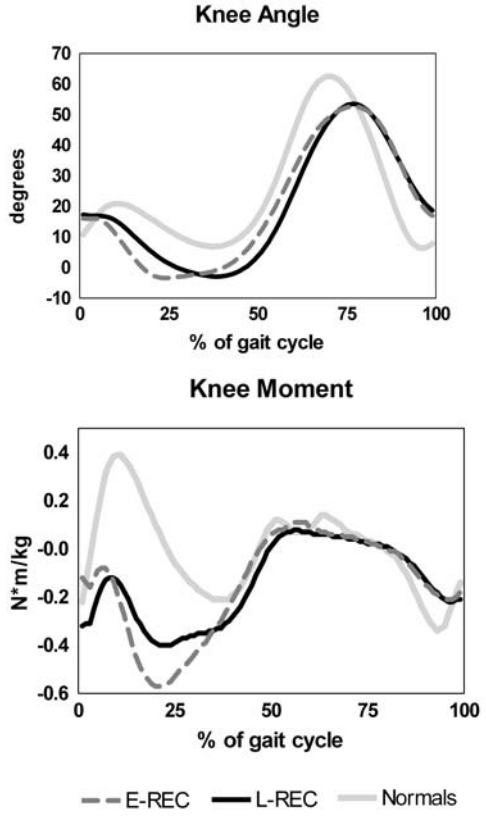

Figure 2. The knee kinematics and kinetics. Graphical comparison of all the three groups. (E-REC = Early recurvatum group, L-REC = Late recurvatum group, Norm = Control group of normally developing children)

Muscle length calculation

Using kinematic data, the Mobile GaitLab software (University Duisburg-Essen, Institute of Mechatronics and System Dynamics, Germany) [13,14] was utilised to compute individual muscle lengths of semimembranosus (ST), semitendinosus (SM) and the long head of biceps femoris (BCL). The specification of the lower extremity architecture was based on the origin and insertion coordinate data compiled by Yamaguchi [15]. The model defines the musculoskeletal geometry of a normal adult male. Three dimensional muscle-tendon pathways were modelled as a series of straight-line segments extending from the origin to insertion. Where appropriate, the muscle-tendon pathways were sub-sectioned into a number of straight-line segments by introducing via-points. Muscle-tendon length parameters were normalized to allow the comparison among subjects.

Electromyography

Surface electromyograms of the medial and lateral hamstrings were available for 15 CP children included in this study (5 children from E-REC, 10 children from L-REC). Raw electromyograms were recorded in a frequency range between 25 and 400Hz (Noraxon, Myosystem 2000). After AD-conversion at a sampling rate of 1kHz, the signals were full wave rectified and digitally low pass filtered at 6Hz. After time normalisation a minimum of 12 gait cycles were averaged to create an average linear envelope for each muscle. Calculation methods applied have been described in detail by Yang and Winter [16].

Statistics

One-way ANOVA with Fischer post-hoc test (Statistica 6.0, StatSoft, Tulsa, OK, USA) was used for comparison between the healthy children and the two recurvatum sub-groups. The significance level was set to $p<0.05$.

Kinematics and kinetics in the sagittal plane, the normalized lengths of the semimembranosus, semitendinosus and the long head of the biceps muscles and electromyographic data were considered as outcome measures for this study.

Results

There was no difference in the minimum knee angle during the stance phase of the gait cycle between the E-REC and L-REC groups, but both were abnormal when compared to the control group (Tab. 1). The maximal knee flexion angles in both recurvatum groups were lower and delayed in timing compared to the control group. Both recurvatum groups demonstrated increased external extensor moments across the knee in the stance phase of the gait cycle. The increase was greater in the E-REC group. All extremities with the

knee recurvatum showed a forefoot landing. Figure 2 represents the knee kinematics and kinetics for the study groups. Electromyographic evaluation of muscle activation patterns revealed no differences between the E-REC and the L-REC. In contrast to a normal muscle activation pattern, 87% of the spastic limbs demonstrated prolonged activity of the medial hamstrings during stance. Also, more than two thirds of the spastic limbs showed an early activation of the medial hamstrings during swing phase.

The normalized muscle-tendon lengths of both medial and lateral hamstrings for both recurvatum groups were abnormally long at initial contact (Fig. 3) when compared to the control group (Tab. 2). The semimembranosus, semitendinosus and the long head of biceps femoris of both the recurvatum groups were contracting and elongating over a wider range of muscle-tendon lengths when compared to the control group. There was no difference in the range of hamstring muscle-tendon lengths between recurvatum groups. During the single limb support phase, hamstrings of both recurvatum groups were contracting faster and shortened approximately 16% of the mean muscle-tendon length compared to a shortening of 10% for the control group respectively (Tab. 2). In single limb support the mean hamstring muscle-tendon length was shorter in the E-REC group than in the L-REC.

Discussion

Hyperextension of the knee during the stance phase of the gait cycle is frequently observed and commonly treated in children with cerebral palsy. Such a pathological gait pattern puts the knee under an abnormal and increased external extensor moment which may cause joint laxity, knee pain [10] and it has also been reported to induce growth disturbance of the proximal tibia [9].

In this study, kinematic and kinetic analyses together with musculoskeletal modelling and surface electromyography were used to investigate the role of the hamstrings in children with cerebral palsy who developed knee recurvatum. Hamstrings are biarticular muscles providing hip extension and knee flexion. The length of these biarticular muscles does not intuitively reflect angular changes of a single joint, but is dependent on angular changes of multiple joints as well as on the moment arms *about* these joints. Delp introduced musculoskeletal modelling into the clinical routine, reporting that 80% of patients with cerebral palsy who developed crouch gait had hamstrings of normal length or longer, despite persistent knee flexion during the stance phase of gait [12]. This was an unexpected finding as until then hamstrings were considered to be too short and were routinely addressed by surgery in children with crouch gait.

According to electromyography studies of normal gait, hamstrings are active in the terminal swing and just after the initial contact during the loading response. Hamstrings, in the terminal swing, produce the hip extensor moment that promotes knee extension and the knee flexor moment that controls and slows that motion [17]. Because the lever arm of the hamstrings at the knee is shorter, compared to its lever arm at the hip, hamstrings are more effective as hip extensors [18]. They play an important role during initial contact when hamstrings inhibit the knee extension and assist in generating extension power at the hip [17]. This study demonstrated both medial and lateral hamstrings to be abnormally long at initial contact in knee recurvatum limbs. For both recurvatum groups these muscles act over a wider range of functional muscle lengths over the gait cycle. As hamstrings inhibit the knee extension at initial contact, abnormally long hamstrings could result in a knee instability, which could lead to the knee hyperextension. This finding goes hand in hand with the well documented fact that surgical hamstring over-lengthening can cause knee hyperextension in the stance phase of gait [8].

The musculoskeletal model used in this study has several limitations. The Mobile GaitLab software utilizes musculoskeletal data of a normal adult male [15]. As the data are scaled to the model, relative differences in muscle lengths should not be affected even if the an adult model is used [19]. Similar models have been used as an outcome measure in studies of children with cerebral palsy [12,20]. Children with cerebral palsy cannot be assumed to have normal bony architecture. Osseous deformities might alter muscle attachment points and muscle paths. Schutte et al. showed the effect of femoral anteversion on psoas muscle length, but the lengths of hamstring muscles were unaffected [21]. A more elaborate way to study muscle-tendon lengths would be to use an individualized MRI-based model. However, at the present time, building an individualized MRI-based model would be costly and labour intensive and is not feasible for retrospective evaluation.

In contrast to Lin's findings [22] and in accordance with Simon's report [4] the outcome of this study showed prolonged activity of the hamstrings during the stance phase of gait. Hamstring muscles of both recurvatum groups shortened approximately 16% during the single support stance phase compared to only 10% shortening for

the control group. Moreover, the E-REC group had a shorter mean muscle-tendon length of the hamstrings in the single support stance phase, which points to a more dynamic muscle tightness in E-REC group. The increased hamstring shortening during the stance phase of gait in the recurvatum limbs could represent a compensation for functionally long hamstrings at initial contact. This interpretation is supported by the finding of a prolonged electromyographic activity of the hamstrings for both recurvatum groups.

In summary, this study shows that semimembranosus, semitendinosus and the long head of biceps femoris are abnormally long at initial contact and operate over a wider range of muscle-tendon lengths in patients with cerebral palsy showing knee recurvatum. The mean hamstring muscle-tendon length was shorter in the E-REC group compared to L-REC in the single support stance phase. Functionally long hamstrings might result in some degree of knee joint instability and could cause its hyperextension. Our interpretation is supported by the clinical observation that surgical over-lengthening of hamstrings leads to knee hyperextension in children with cerebral palsy. In our opinion, abnormally long hamstrings together with equinus position of the foot at initial contact are the main causes of genu recurvatum in children with spastic cerebral palsy.

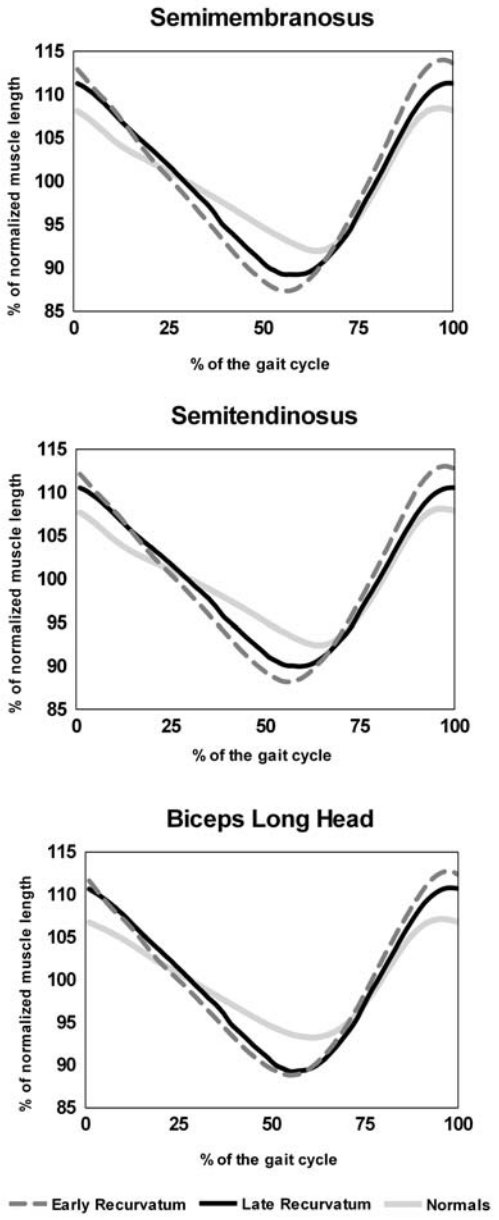

Figure 3. Normalized muscle-tendon lengths of hamstrings. Comparison of Early and Late recurvatum group means with a control group.

	E-REC v L-REC	E-REC v Norm	L-REC v Norm	E-REC		L-REC		NORM	
	P-level	P-level	P-level	Mean	SD	Mean	SD	Mean	SD
Ankle angle at initial contact	0.5482	**0.0001**	**0.0000**	-7.42	8.05	-6.00	9.11	3.17	2.99
Maximal knee extension in stance	0.9922	**0.0000**	**0.0000**	-3.79	3.10	-3.81	3.75	5.01	4.54
Maximal knee flexion in GC	0.7084	**0.0021**	**0.0007**	54.23	9.12	55.18	9.37	62.74	4.02
Timing of maximal knee flexion in GC	0.3233	**0.0000**	**0.0000**	78.21	4.46	79.33	4.04	71.42	1.67
External extensor moment in stance	0.2798	**0.0000**	**0.0000**	-0.64	0.25	-0.58	0.21	-0.25	0.08
Maximal hip flexion in GC	0.6375	**0.0286**	**0.0283**	40.82	8.82	39.65	9.00	34.96	4.79
Maximal hip extension in GC	0.0824	0.6618	0.1270	-8.00	7.63	-12.31	7.82	-9.13	7.47
Mean pelvis tilt in GC	0.7535	**0.0233**	**0.0136**	14.84	6.85	14.21	6.83	9.97	4.87

Table 1. Summary of the kinematics and kinetics results. (E-REC = Early recurvatum group, L-REC = Late recurvatum group, Norm = Control group of normally developing children)

		E-REC v L-REC	E-REC v Norm	L-REC v Norm	E-REC		L-REC		Norm	
		P-value	P-value	P-value	Mean	SD	Mean	SD	Mean	SD
SM	Initial contact	0.5872	**0.0082**	**0.0008**	112.21	1.33	111.40	0.64	108.12	0.67
	Maximum in SS	0.3499	0.6500	**0.0290**	105.48	1.08	106.61	0.52	104.93	0.54
	Minimum in SS	0.3789	**0.0006**	**0.0000**	88.94	1.37	90.29	0.66	94.53	0.68
	Mean in SS	**0.0026**	**0.0000**	**0.0060**	96.85	2.11	98.70	1.43	99.74	0.79
	ROM in SS	0.9316	**0.0205**	**0.0005**	16.55	2.30	16.33	1.10	10.41	1.15
	ROM	0.3913	**0.0045**	**0.0013**	23.94	2.46	21.47	1.18	16.03	1.23
ST	Initial contact	0.5685	**0.0106**	**0.0015**	111.45	1.24	110.66	0.60	107.77	0.62
	Maximum in SS	0.3619	0.6168	**0.0265**	105.13	0.98	106.13	0.47	104.58	0.49
	Minimum in SS	0.3372	**0.0006**	**0.0001**	89.78	1.27	91.15	0.61	94.97	0.64
	Mean in SS	**0.0033**	**0.0000**	**0.0088**	97.18	2.01	98.88	1.35	99.82	0.75
	ROM in SS	0.8758	**0.0180**	**0.0005**	15.35	5.39	14.99	6.46	9.61	3.09
	ROM	0.3716	**0.0059**	**0.0024**	23.94	2.46	21.47	1.18	16.03	1.23
BCL	Initial contact	0.3619	0.6168	**0.0265**	111.63	4.52	110.65	3.41	106.89	1.77
	Maximum in SS	0.2516	0.9452	0.0871	104.77	2.36	106.17	3.50	104.85	1.32
	Minimum in SS	0.6053	**0.0010**	**0.0000**	89.47	4.14	90.20	3.65	94.43	2.03
	Mean in SS	**0.0036**	**0.0000**	**0.0011**	96.67	1.91	98.31	1.30	99.48	0.80
	ROM in SS	0.7892	0.0547	**0.0007**	15.30	5.84	15.96	6.78	10.42	3.29
	ROM	0.4344	**0.0009**	**0.0000**	24.27	9.50	22.07	7.29	14.34	3.22

Table 2. Summary of the normalized muscle-tendon lengths results. (E-REC = Early recurvatum group, L-REC = Late recurvatum group, Norm = Control group of normally developing children, SM = semimembranosus, ST = semitendinosus, BCL = biceps femoris long head, SS = single support phase of the stance, ROM = range of muscles lengths)

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STUDY 2

Dynamic versus Fixed Equinus Deformity in Children with Cerebral Palsy

How does the triceps surae muscle works?

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Abstract

Equinus deformity is a common problem in children with cerebral palsy. Distinguishing between the dynamic tightness and fixed triceps surae contracture is a key-point in clinical decision making. We compared 12 dynamic (DEQ) and 19 fixed equinus (FEQ) deformities based on kinematic and kinetic gait analysis and muscluloskeletal modeling. Ankle range of motion, maximal plantarflexor power and its timing were different between the groups. Maximal muscle lengths of medial gastrocnemius and soleus were longer in DEQ compared to FEQ group. Peak lengthening velocity of triceps surae muscle was significantly slower for all triceps surae muscles in FEQ group and occured in the early swing phase. The presented results indicate that peak lengthening velocity of triceps surae muscle might be one of the discriminating factors between fixed and dynamic equinus deformity in children with cerebral palsy, which could help the clinical decision-making on treatment of equinus gait pattern.

Introduction

In children with cerebral palsy, motor impairment is due to a number of deficits, including poor muscle control, weakness, impaired balance, hypertonicity and spasticity. As a consequence, muscle tendon units frequently become contracted over time. Equinus deformity is a very common problem in children with cerebral palsy. Equinus decreases the stability during the stance phase of gait, causes a loss of the smooth translation of the body over the foot and often leads to an inadequate clearance of the foot during the swing phase of gait [1]. A shortened gastrocnemius-soleus complex results in a coupled motion between ankle plantarflexion and knee extension [2]. Surgical techniques to lengthen the triceps sureae complex (Baker, Strayer, Baumann) [3-5] or the Achilles tendon [6] represent appropriate treatments for fixed equinus contractures. In contrast, dynamic triceps surae tightness can be addressed by ankle-foot orthoses, the application of Botulinum Toxin A and serial casting [7]. Differntiating a fixed contracture from a dynamic muscle tightness can pose a challenge in clinical decission making. Here, the clinical examination is limited by its static nature and there is only fair to moderate correlation to muscle function during walking [8]. Moreover, dynamic equinus deformities cannot be accurately assessed clinically through traditional muscle tests such as the Silverskiôld test because the findings might be invalid if the tests are performed in the awake state [1]. To date, the examination of ankle motion under general anesthesia represents the gold standard for distinguishing between dynamic triceps muscle tightness and fixed equinus contracture and allows an assessment of mechanical muscle properties for each of the two equinus deformities possible. This gold standard has, however, rarely been utilised in projects investigating spastic equinus deformities which could have biased the interpretation of previous research on dynamic and fixed equinus deformities in cerebral palsy.

This report presents results of a study investigating the function of the triceps surae complex during walking when either a dynamic triceps surae tightness or a fixed equinus contracture is present in children with cerebral palsy. To describe differences between dynamic and fixed equinus deformities we applied 3D gait analysis and analysed kinematic and kinetic parameters. To provide further insight into the different types of equinus we utilized musculoskeletal modelling to study length changes of muscle-tendon units during gait. Findings are compared to a group of normally developing children.

Methods

This prospective evaluation utilized data from gait analysis, musculo-skeletal modelling and hospital medical records. Inclusion criteria were: 1) spastic diplegic or hemiplegic type of cerebral palsy; 2) equinus gait; 3) examination of the triceps surae under general anesthesia; 4) complete gait analysis data to perform musculoskeletal modeling. Exclusion criteria were: 1) previous surgery on the triceps surae; 2) Botulinum toxin A treatment in the previous 6 months preceding gait analysis; 3) other than spastic form of cerebral palsy.

Equinus gait was defined to be present when peak ankle dorsiflexion during the stance phase of gait measured more than one standard deviation below the normal average [9]. Within eight weeks after gait analysis patients were examined under general anesthesia to discriminate fixed contracture from dynamic tightness. The indication for general anesthesia was either the application of Botulinum Toxin A or surgery. Under general anaesthesia, with the knee fully extended a maximum dorsiflexion at the ankle of less than 5 degrees was defined to represent a fixed contracture.

Based on these inclusion and exclusion criteria, twenty-three children (31 limbs) with spastic cerebral palsy and equinus gait were included in this study. Eight children were diplegic and fifteen hemiplegic. According to the definition of the type of equinus 12 limbs showed a fixed contracture (Fixed Equinus Group, FEQ) and 19 limbs demonstrated dynamic tightness of the triceps muscle (Dynamic Equinus Group, DEQ). The average age in the FEQ group was 11 years and 6 months and 9 years and 4 months in the DEQ group. The level of Gross Motor Classification System (GMFCS) was I or II in all children with cerebral palsy [10]. A group of 12 healthy children without any neurological or orthopaedic disorder served as the control group. The study was approved by the the local.ethics committee.

Gait analysis

Computerized gait analysis was performed using a ten-camera video based motion-capturing system (Vicon MX, Oxford Metrics, Oxford, U.K.) and four floor-mounted force plates (AMTI). Marker arrangement, calculation methods and model assumptions that were applied have been described in detail by Kadaba et al. [11]. Calculations of kinematic parameters for the pelvis, and the hip, knee and ankle joints as well as kinetic parameters for the hip, knee and ankle joint were performed using the Vicon Clinical Manager software (VICON, Oxford Metrics, Oxford, UK). Moment and power parameters were normalized

to the weight of the patients. Power generation and absorption patterns in the sagittal plane were calculated and labeled according to the method described by Winter et al [12]. Patients walked barefoot along a 10-m long walkway at self-selected speed. For each limb a minimum of five valid trials with clear foot contacts on a force plate were recorded.

Musculoskeletal modelling

The "Mobile GaitLab" software (University Duisburg-Essen, Institute of Mechatronics and System Dynamics, Germany) [13-15] was utilized to estimate muscle–tendon lengths and velocities of muscle–tendon length changes. Calculations are based on kinematic parameters obtained from gait analysis. The underlying model is based on the work of Yamaguchi [16]. Muscle–tendon lengths were calculated as the distance from origin to insertion along the modelled path of each muscle. Via points were introduced to intersect the muscle-tendon paths taking into account the underlying anatomical constraints and defining the line of action of each muscle. Muscle–tendon velocity was estimated by computing the first derivative of the muscle–tendon length data with respect to time. The muscle–tendon lengths were normalized to allow comparison between groups. Similar to a study by Arnold et al. no attempt was made to scale the model prior to normalization [17]. For this study muscle-tendon length parameters were evaluated for the soleus and the medial and lateral head of the gastrocnemius muscle.

Statistics

Sagital plane kinematic and kinetic parameters of the ankle and knee joint as well as muscle-tendon length and velocity parameters of the medial gastrocnemius (MGAC), the lateral gastrocnemius (LGAC) and the soleus (SOL) muscles were considered as outcome measures. One way ANOVA with the Fisher post-hoc test (Statistica 6.0, StatSoft, Tulsa, OK) were used to describe the differences between dynamic and fixed equinus groups and both groups were compared to the control group of healthy children.

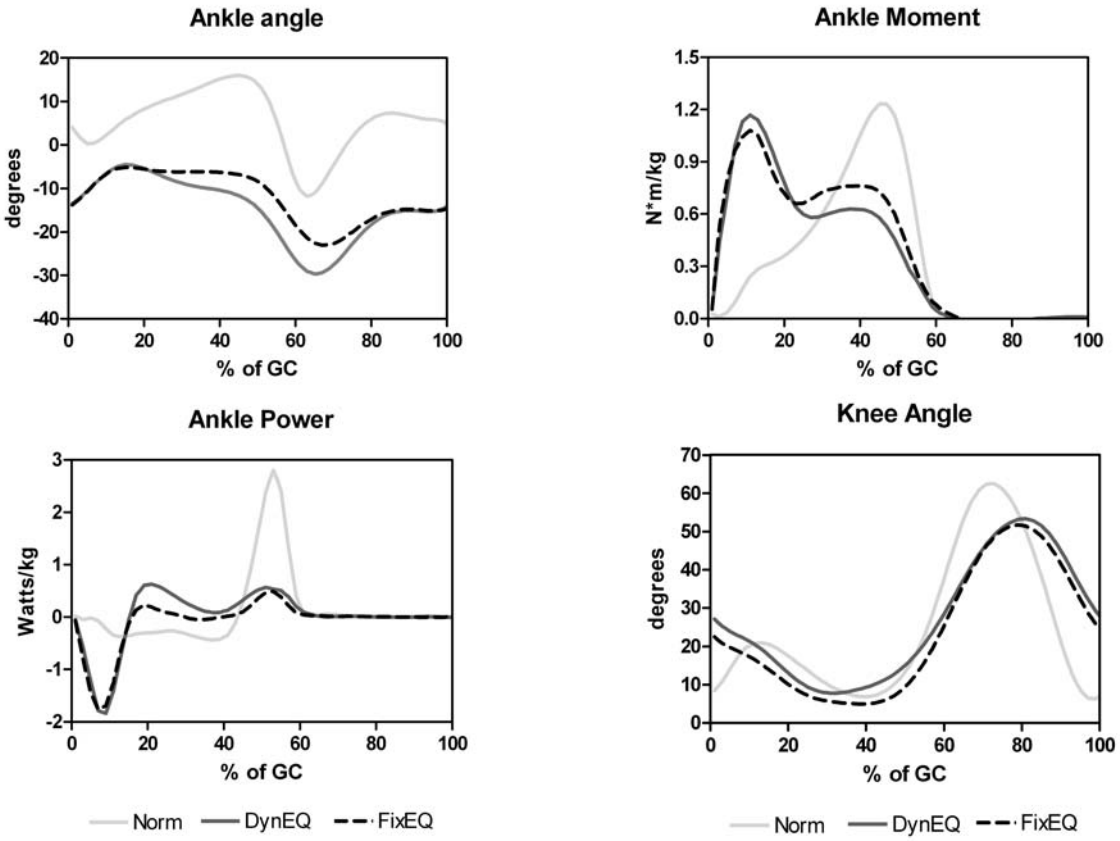

Figure 1. Kinematic and kinetic parameters of children with dynamic and fixed equinus deformity in comparison to normally developing children.

Results

The study groups were comparable in age and there was no statistical difference in the self-selected walking speed between the DEQ and FEQ groups. Analysis of ankle joint motion revealed that none of the equinus feet reached a neutral position at any time during the gait cycle (Fig 1). The maximal dorsiflexion in stance was not different between the DEQ and FEQ groups. The plantarflexion at toe-off and the ankle range of motion were different for the three groups with the DEQ group showing more plantarflexion than the FEQ group, which exceeded the values of the NORM group (Table 1). Subtracting ankle angles at the start of single support from the ankle angle values at the end of single support provided information about the direction of motion at the ankle joint during single limb support. This parameter was called “ankle slope parameter”. The ankle slope parameter was different among the three study groups (Table 1). Negative values for both equinus groups indicated a pathological plantarflexion motion during single support, which was more pronounced for the DEQ group (Fig 1). Ankle joint kinetics was abnormal in both equinus groups. While the maximal ankle moment values during stance did not differ between the two groups, the maximal ankle moments of DEQ and FEQ occurred early at the beginning of the single stance phase (DEQ at 12 and FEQ at 16 percent of the gait cycle). In contrast, in the NORM group maximal ankle moment values were observed at the end of single support at about 45 percent of the gait cycle. About 50-70% of the propulsive power for walking is generated by plantarflexor muscles at the end of single support stance. Although the DEQ group generated more power during plantar flexion than the FEQ group (Fig 1) the maximum ankle power generation during the stance phase of gait was reduced for both equinus groups when compared to the NORM group. There was an early timing of maximum ankle power generation in DEQ group (29 percent of the gait cycle). On the other hand, the timing of maximum ankle power generation for the FEQ occured at the end of the stance phase and was similar to the NORM group. The total power generated during the second double support phase was reduced for both equinus groups. Kinematics of the knee revealed a decreased range of knee motion for both equinus groups. Also, the maximal knee flexion occurs later during the swing phase in both equinus groups (Table 1).

Muscle-tendon length parameters of the soleus, medial and lateral gastrocnemius muscles were abnormal for both equinus groups when compared to the NORM group (Table 2). During the first double support stance phase both equinus groups demonstrated fast initial elongation. While in healthy children the triceps surae muscles elongated during the single stance phase, the muscle complex appeared to plateau in its length for the FEQ group and even showed shortening for the DEQ group (Fig. 2). During the stance phase the timing and the amount of the maximal muscle-tendon lengths of the soleus and the medial gastrocnemius were different among the equinus groups. Muscle-tendon lengths of the lateral gastrocnemius were similar for both the DEQ and FEQ groups (Table 2). The muscle-tendon lengths at toe-off were abnormally short in FEQ group compared to DEQ and healthy children. Analyzing the muscle-tendon velocity revealed group differences. Here equinus limbs demonstrated slower contraction/elongation than limbs of the NORM group. Moreover, the muscle-tendon velocity was slower in the FEQ group when compared to the DEQ group (Table 2). The graphical summary of important results distinguishing dynamic and fixed equinus using gait analysis and musculoskeletal modeling is given in Table 3.

Discussion

The distinction between dynamic calf muscle tightness and a fixed calf muscle contracture is critical in clinical decision making [1;18]. While dynamic tightness might be addressed by conservative treatments [19], fixed muscle contracture represents an indication for surgery [18]. In cerebral palsy increased muscle tone and spasticity make it difficult to asses the muscle-tendon length of a single muscle group in the awake state. While the clinical range of motion testing to detect fixed contractures might have considerable sensitivity, the specificity of the tests is documented to be poor [20]. Therefore, if surgical decision-making had to rely on clinical examination alone, several dynamically tight calf muscles would be evaluated as requiring surgical lengthening procedures.

The musculoskeletal model used in this study has several limitations. The Mobile GaitLab utilizes musculoskeletal data of an adult male [16]. However, similar models were used to calculate outcome measures in studies evaluating children with cerebral palsy [21;22], and an adult model should not affect normalized muscle lengths [9]. Children with cerebral palsy cannot be assumed to have normal bone architecture. Rather their muscle attachments and muscle-tendon paths

might be altered by bony deformities. For the psoas muscle Schutte showed the effect of femoral anteversion on muscle-tendon length [23]. So far no study has documented the influence of bony deformities (tibial torsion, ankle varus/valgus) on the calculation of triceps surae muscle-tendon length parameters.

The method of Eames [24] has frequently been applied to calculate gastrocnemius muscle-tendon length. As this model does not account for the two heads of the gastrocnemius muscle and might therefore be too simplistic. There is evidence that the medial and lateral heads of the gastrocnemius muscle differ considerably in their architecture [25], their length and pattern of activation [26]. The Mobile GaitLab muscle model uses separate attachement points for the two gastrocnemius heads according to Yamaguchi's data [16]. This might explain the differences in muscle-tendon length parameters of the medial and the lateral head of gastrocnemius in this study.

Ankle kinematics differed between the DEQ and the FEQ group (Fig. 1). During the stance phase of gait, normal ankle motion shows a gradual dorsiflexion. Here, ankle motion in the FEQ group demonstrated persistant equinus and in the DEQ group the ankle is even plantarflexing. This abnormal ankle motion in the DEQ group relates to early maximal power generation during single stance. In contrast, normal children show ankle power absorption during single limb support. The nearly opposing patterns of ankle kinematics and ankle powers in children with a dynamic equinus can be interpreted as the effect of an out of phase muscle contraction due to a muscle stretch following a forefoot landing. The dynamic nature of this contraction is documented by the high muscle-tendon velocity for the triceps surae in the DEQ group. For the FEQ group the kinematic, kinetic and muscle-tendon length parameters illustrate the fixed nature of the equinus. Here, the ankle shows only minor motion and the muscle-tendon lengths remain almost unchanged during single limb support. The muscle-tendon velocity was close to zero. The reduction in ankle motion contributes to a reduction in ankle power generation values that were the lowest of all the three study groups.

Besides the reduction of muscle-tendon lengths in children with cerebral palsy, we feel that the shortening and lengthening muscle-tendon velocity of the triceps surae during gait represents a valuable parameter to evaluate spastic equinus. Our results indicate that all three triceps surae muscles achieve significantly lower peak lengthening velocities in the FEQ group when compared to the DEQ group. Decreased peak lengthening velocity might be an effect of increased muscle stiffness due to an increase in passive tension of the triceps surae muscle fibres. Fridén and Leiber proved that the mechanical properties of isolated muscle fibers are different in patients with cerebral palsy who developed contractures [27]. Cells of spastic muscles have shorter resting sarcomere lengths and develop passive tension on significantly shorter sarcomere lengths compared to healthy individuals. Another explanation for the decreased peak muscle-tendon lengthening velocity could be a change in the architecture of spastic muscles. Rose et al. demonstrated that spastic muscles in children with cerebral palsy show a predominance of slow-twitch type I muscle fibers and that fiber sizes were different when compared to healthy individuals [28]. Weakness of the dorsiflexor muscles, which is common among children with cerebral palsy who walk with equinus pattern, could represent another factor for decreased peak lengthening velocity [29]. This would, however, not explain the difference in peak lengthening velocity between fixed contracture and dynamic tightness.

This study compared dynamic and fixed equinus gait in children with cerebral palsy. The distinction between a dynamic and a fixed equinus was based on an examination under general anaesthesia. This allowed the evaluation of kinematics, kinetics and calf muscle-tendon length parameters in well defined groups. Characteristic changes in ankle joint kinematics and abnormal kinetic parameters were found for both types of equinus. The rate of change of muscle-tendon length might be an interesting parameter to distinguish between fixed and dynamic equinus gait in children with cerebral palsy.

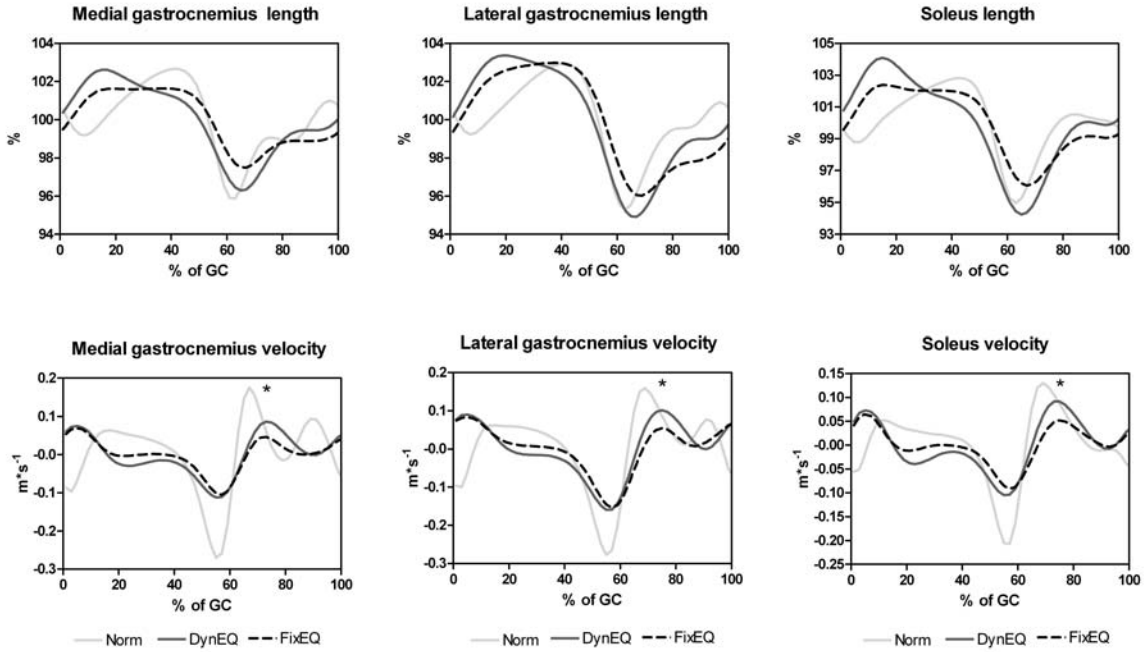

Figure 2. Muscle length and velocities of triceps surae in children with dynamic and fixed equinus deformity in comparison to normally developing children. The * (star) shows the statistically significant differences in the peak lengthening velocity of each head of triceps surae muscle.

	p-level	p-level	p-level	DEQ				FEQ				NORM			
	DEQ v FEQ	DEQ v Norm	FEQ v Norm	Mean	CI (5%)	CI (95%)	SD	Mean	CI (5%)	CI (95%)	SD	Mean	CI (5%)	CI (95%)	SD
Maximal ankle dorsiflexion	0.6096	**<0.0001**	**<0.0001**	-4.85	-8.88	-0.83	8.35	-3.64	-8.13	0.84	6.27	14.80	13.52	16.08	3.03
Ankle plantarflexion at Toe-Off	**0.0076**	**<0.0001**	**0.0111**	-29.59	-35.65	-23.53	12.58	-20.05	-25.58	-14.53	7.72	-11.35	-13.23	-9.46	4.47
Maximal ankle dorsiflexion during swing	0.8778	**<0.0001**	**<0.0001**	-13.53	-17.78	-9.29	8.81	-13.15	-18.45	-7.86	7.40	6.56	5.72	7.40	1.99
Ankle range of motion (ROM)	**0.0065**	0.0577	**<0.0001**	27.23	23.32	31.15	8.12	20.05	16.92	23.19	4.39	31.09	28.71	33.46	5.62
Ankle slope	**0.0048**	**<0.0001**	**<0.0001**	-10.16	-14.61	-5.72	9.23	-2.23	-5.93	1.48	5.18	9.86	7.70	12.01	5.11
Maximal anke moment	0.8127	0.5818	0.8384	1.20	0.99	1.41	0.44	1.23	0.97	1.48	0.35	1.25	1.17	1.33	0.19
Timing of max ankle moment (% of GC)	0.1834	**<0.0001**	**<0.0001**	12.32	9.10	15.53	6.67	15.80	7.18	24.42	12.04	45.17	44.38	45.95	1.86
Absorption power in single stance	**0.0255**	**<0.0001**	**<0.0001**	1.07	0.66	1.48	0.85	0.49	0.33	0.65	0.22	2.37	2.13	2.60	0.56
Maximal power generation	**0.0181**	**<0.0001**	**<0.0001**	1.23	0.78	1.69	0.94	0.59	0.41	0.77	0.26	3.27	3.06	3.49	0.51
Timing of max ankle power generation (% of GC)	**0.0007**	**<0.0001**	0.1353	29.26	21.20	37.33	16.74	46.20	35.07	57.33	15.55	53.08	52.56	53.61	1.25
Sum of Push-off power	0.6672	**<0.0001**	**<0.0001**	4.34	1.58	7.10	5.73	3.61	1.95	5.26	2.32	18.20	16.65	19.75	3.67
Maximal knee flexion	0.2497	**<0.0001**	**<0.0001**	55.78	52.18	59.37	7.47	52.79	46.17	59.42	9.26	62.74	61.04	64.44	4.02
Knee range of motion (ROM)	0.9275	**0.0002**	**0.0015**	48.61	44.34	52.89	8.87	48.31	38.36	58.25	13.90	59.17	57.15	61.19	4.78
Timing of max knee flexion (% of GC)	0.6616	**<0.0001**	**<0.0001**	81.00	78.75	83.25	4.67	80.40	77.46	83.34	4.12	71.42	70.71	72.12	1.67

Table 1. Results of ANOVA comarison of kinematic and kinetic parameters among dynamic (DEQ) , fixed (FEQ) equinus and normally developing children (NORM). Statistically significant results (P<0,05) are in **bold.**
(GC = gait cycle, CI = confidence interval, SD = standard deviation)

	p-level	p-level	p-level	DEQ				FEQ				NORM			
	DEQ v FEQ	DEQ v Norm	FEQ v Norm	Mean	CI (5%)	CI (95%)	SD	Mean	CI (5%)	CI (95%)	SD	Mean	CI (5%)	CI (95%)	SD
Muscle length of GAM at the end of DS1	**0.0057**	**<0.0001**	**<0.0001**	102.32	101.76	102.89	1.17	101.41	100.90	101.92	0.71	99.37	99.20	99.54	0.41
Muscle length of GAL at the end of DS1	**0.0419**	**<0.0001**	**<0.0001**	102.72	102.12	103.31	1.23	102.00	101.37	102.63	0.89	99.44	99.26	99.62	0.42
Muscle length of SOL at the end of DS1	**0.0028**	**<0.0001**	**<0.0001**	103.61	102.80	104.43	1.69	102.17	101.33	103.01	1.17	99.27	99.07	99.48	0.49
Maximum muscle length of GAM	**0.0039**	0.4292	**0.0160**	102.97	102.45	103.49	1.08	102.06	101.78	102.34	0.39	102.78	102.55	103.02	0.56
Maximum muscle length of GAL	0.1702	0.0586	0.8933	103.74	103.23	104.24	1.05	103.23	102.45	104.01	1.09	103.18	102.86	103.50	0.77
Maximum muscle length of SOL	**0.0015**	**0.0009**	0.5365	104.25	103.53	104.96	1.49	102.83	102.24	103.43	0.83	103.09	102.78	103.39	0.71
Length of GAM at toe-off	**0.0111**	0.2138	**0.0004**	96.65	95.79	97.51	1.78	97.90	97.30	98.51	0.85	96.18	95.90	96.46	0.67
Length of GAL at toe-off	**0.0007**	0.1923	**0.0101**	95.47	94.72	96.22	1.56	97.16	96.32	98.00	1.18	95.96	95.61	96.31	0.82
Length of SOL at toe-off	**0.0070**	**0.0532**	0.1980	94.73	93.55	95.91	2.44	96.59	95.48	97.70	1.55	95.76	95.43	96.09	0.78
Maximum lenghtening velocity of GAM	**0.0040**	**<0.0001**	**<0.0001**	0.12	0.10	0.13	0.03	0.08	0.07	0.10	0.02	0.19	0.18	0.20	0.03
Maximum lenghtening velocity of GAL	**0.0042**	**<0.0001**	**<0.0001**	0.13	0.12	0.14	0.02	0.09	0.07	0.11	0.03	0.17	0.16	0.19	0.04
Maximum lenghtening velocity of SOL	**0.0001**	**0.0227**	**<0.0001**	0.12	0.11	0.13	0.02	0.08	0.06	0.09	0.02	0.14	0.13	0.15	0.03

Table 2. Results of ANOVA comarison of muscles length and velocity parameters of triceps surae among dynamic (DEQ) , fixed (FEQ) equinus and normally developing children (NORM). Statistically significant results (P<0,05) are in **bold.**
(GC = gait cycle, CI = confidence interval, SD = standard deviation, GAM = medial gastrocnemius, GAL = lateral gastrocnemius, SOL = soleus)

	DEQ	FEQ	NORM (mean ± SD)
Ankle ROM	↓	↓↓	31.09 ± 5.62 degrees
Timing of maximal plantarflexor power	↓↓	↓	53.07 ± 1.25 % of GC
Max plantarflexion power	↓	↓↓	3.27 ± 0.51 Watt*kg^{-1}
Ankle angle at toe-off	↓↓	↓	-11.35 ± 4.47 degrees
Ankle slope parameter	↓↓	↓	9.86±5.11 degrees
Maximal medial gastrocnemius muscle-tendon length	Norm.	↓↓	102.78 ± 0.56 %
Maximal medial gastrocnemius muscle-tendon velocity	↓	↓↓	0.19 ± 0.03 m*s^{-1}

Table 3. The major differences between dynamic (DEQ) and fixed (FEQ) equinus groups in comparison to normally developing children (NORM). (↓=decreased in comparison to normals, ↓↓= very decreased in comparison to normals, Norm.=within the normal range)

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STUDY 3

Short-term effects of Botulinum Toxin A and serial casting on triceps surae muscle length and equinus gait in children with cerebral palsy

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Abstract:

Aims: The aim of the study was to perform a comprehensive analysis of the short-term effects of a standardized high-dose of BTX-A and serial casting on spastic equinus in children with cerebral palsy. A second goal was to evaluate this short-term effect on muscle lengths of the triceps surae.

Materials and Methodology: Prospective study on 10 children (17 limbs) with spastic cerebral palsy and equinus gait pattern. Three-dimensional kinematic and kinetic gait analysis with musclulo-skeletal modelling was used to evaluate pre- and 10 weeks post-treatment results. Paired student's t-test were used for data analysis.

Results: The walking velocity and the stride length did not change after the BTX-A application. The maximal ankle angle at the end of single support increased (p=0,045) which led to a change of the ankle angle slope quotient towards normal positive values (p=0,001). Abnormal ankle power generation in the first part of single stance changed towards normal (p=0,012). Notably, the ankle plantar-flexor power was not reduced 10 weeks after BTX-A injection (p=0,486). No lengthening of the triceps surae muscles compared to the baseline assessment was found.

Conclusions: Beneficial functional changes in ankle kinematics and kinetics after the application of BTX-A when combined with serial casting were demonstrated. The results of our study underline the possible differences in effect of BTX-A on intrafusal and extrafusal muscle fibres.

Short-term Effects of Botulinum Toxin A and Serial Casting on Triceps Surae Muscle Length and Equinus Gait in Children with Cerebral Palsy

Vliv léčby botulinum toxinem-A a redresního sádrování na délku musculus triceps surae a ekvinózní postavení nohy během chůze u pacientů s dětskou mozkovou obrnou

Abstract

Aims: The aim of the study was to perform a comprehensive analysis of the short-term effects of a standardized high dose of BTX-A and serial casting on spastic equinus in children with cerebral palsy. A second goal was to evaluate this short-term effect on muscle lengths of the triceps surae. *Materials and methodology:* Prospective study on 10 children (17 limbs) with spastic cerebral palsy and equinus gait pattern. Three-dimensional kinematic and kinetic gait analysis with musclulo-skeletal modelling was used to evaluate pre- and 10 weeks post-treatment results. The paired Student's T-test was used for data analysis. *Results:* Walking velocity and stride length did not change after BTX-A application. The maximal ankle angle at the end of single support increased ($p = 0.045$) which led to a change of the ankle angle slope quotient towards normal positive values ($p = 0.001$). Abnormal ankle power generation in the first part of single stance changed towards normal ($p = 0.012$). Notably, the ankle plantar-flexor power was not reduced 10 weeks after BTX-A injection ($p = 0.486$). No lengthening of the triceps surae muscles compared to the baseline assessment was found. *Conclusions:* Beneficial functional changes in ankle kinematics and kinetics after the application of BTX-A when combined with serial casting were demonstrated. The results of our study underline the possible differences in effect of BTX-A on intrafusal and extrafusal muscle fibres.

Souhrn

Cíle: Cílem této studie bylo provést komplexní analýzu krátkodobého účinku vysokodávkovaného botulinum toxinu-A (BTX-A) aplikovaného společně s redresním sádrováním z důvodu ekvinózního postavení nohy během chůze u pacientů s dětskou mozkovou obrnou (DMO). Dalším cílem bylo zhodnotit vliv této léčby na délku musculus triceps surae. *Materiál a metodika:* Jedná se o prospektivní studii, do které bylo zařazeno 10 pacientů (17 končetin) se spastickou formou DMO a ekvinózním postavením nohy. K hodnocení byla použita metoda trojdimenzionální kinematické a kinetické analýzy chůze společně s počítačovou modelací délky musculus triceps surae. Pacienti byli vyšetřeni před a 10 týdnů po léčbě. Ke statistické analýze byl použit párový t-test. *Výsledky:* Rychlost chůze a délka kroku se po aplikaci BTX-A nezměnila. Maximální dorziflexe na konci fáze jedné opory se zvýšila ($p = 0,045$), což vedlo k normalizaci „slope quotient" ($p = 0,001$). Došlo také k odstranění patologické generace síly během první poloviny fáze jedné opory ($p = 0,012$). Oproti očekávání, síla plantiflexorů nebyla aplikací BTX-A ovlivněna ($p = 0,486$). Neprokázali jsme prodloužení musculus triceps surae. *Závěr:* Aplikace BTX-A společně s redresním sádrováním vede ke zlepšení kinematiky a kinetiky chůze pacientů s ekvinózní deformitou a DMO. Výsledky naší studie upozorňují na možnost rozdílného působení BTX-A na intrafuzální a extrafuzální svalová vlákna.

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Introduction

Cerebral palsy refers to a group of disorders in the development of posture and motor control, occurring as the result of a non-progressive lesion of the developing central nervous system [1]. Children with cerebral palsy may display a range of movement disorders, alone or in combination, including dystonia, athetosis, ataxia and spasticity [2]. Equinus gait is one of the most common motor manifestations of muscle spasticity in cerebral palsy [3]. Equinus decreases stability during the stance phase of gait, interferes with the smooth translation of the body over the foot and leads to inadequate clearance of the foot in the swing phase. [4]. If untreated, protracted spasticity can lead to fixed muscle contractures that may require surgical intervention.

Previous reports have found that the presence of spasticity increases muscle stiffness and thereby inhibits muscle growth [4,5]. Besides conservative therapies such as physiotherapy, serial casting, orthotics and oral medication, a variety of invasive treatment methods are now commonly available to manage spasticity in cerebral palsy. These include selective dorsal rhizotomy (SDR), intrathecal administration of baclofen (ITB) or botulinum toxin type A (BTX-A) injections, which has been used clinically for more than 20 years. BTX-A induces muscle weakness by preventing the release of acetylcholine from the presynaptic axon at the motor endplate [5]. The degree of weakening depends on the dose of BTX-A and on the number of synapses affected [6]. Spasticity reduction due to the effects of BTX-A injections typically lasts from 12 to 16 weeks. Re-innervation takes place by sprouting of new nerve terminals, a process that peaks at 60 days in humans [7]. Functional benefits may last for up to six months or even longer [8]. In addition to the effect of muscle weakening, BTX-A has been shown to influence the longitudinal growth of an injected muscle in an animal model [9]. Based on computer simulation and modelling, the same effect was confirmed in humans four weeks after BTX-A application [10].

The motivation for the present study was to perform a comprehensive analysis of the short-term effects of a standardized dose of BTX-A and serial casting on spastic equinus in children with cerebral palsy. A second goal was to evaluate this short-term treatment on muscle length of the triceps surae. To evaluate functional outcome and changes in muscle length, we applied three-dimensional gait analysis and calculated individual dynamic peak muscle lengths by using musculo-skeletal modelling techniques.

Methods

Design

The study is a prospective analysis of the short-term effects of a standardized high dose of BTX-A (7 IU/kg) applied to the gastro-soleus complex and serial casting. The patients were examined before and 10.1 weeks (SD 1.3) after a BTX-A application. The pre-treatment gait analysis was used as a baseline for validation of treatment effect.

Subjects

For this investigation, equinus gait was defined as a peak ankle dorsal-flexion of more than one standard deviation below the normal average during the stance phase of gait [11]. Inclusion and exclusion criteria are listed in Table 1. Based on these criteria, 10 children with spastic cerebral palsy were included in the trial (4 males, 6 females). The average age of our study group was 7 years and one month (SD 2 years and 3 months). Seven children were diplegic and three hemiplegic. A total of 17 lower extremities were analysed and used for statistical analysis. The ethics committee of the local medical university approved the study.

Intervention

BTX-A injections were performed under general anaesthesia. A BTX-A dose of 7 IU/kg (Botox©, Allergan Inc, Irvine, CA, USA) was applied to multiple sites over the muscle belly of the gastrocnemius and soleus. A multiple injection site technique was applied in which every site was injected with 10 IU BTX-A. The positioning of the needle in the muscle was as described by Baker et al [10]. A few hours after the BTX-A injections a below-knee walking cast was applied in maximal ankle dorsal-flexion for a duration of three weeks. Casts were changed every week. Special physiotherapy training was performed after the removal of the third cast in all cases, on an out-patient basis two times a week for the whole period of the study. The physiotherapy included passive muscle stretching and gait improvement training; either the Vojta concept or the Bobath concept was utilized.

Gait analysis

Computerized gait analysis was performed using a six-camera, video-based motion-capturing system (Vicon MX, Oxford Metrics, Oxford, U.K.) and four floor-mounted force plates (Kistler Instruments Limited, Winterthur, Switzerland). Thirteen reflective markers were used to define the pelvis and lower extremity segments in accordance with the Vicon Clinical Manager model (Oxford Metrics, Oxford, U.K.). Moment and power parameters were normalized to the weight of the patients. Power generation and absorption patterns in the sagittal plane were calculated and labelled according to the method described by Winter [12]. All pa-

Table 1. Inclusion and exclusion criteria.

Inclusion criteria	Exclusion criteria
1. Spastic diplegic or hemiplegic cerebral palsy	1. Previous surgery of the triceps surae
2. Equinus gait	2. Botulinum toxin A treatment within six months before entering the trial
3. Ability to walk at least 12 m without walking aids	3. Dystonic, athetotic or ataxic form of cerebral palsy
4. Gross Motor Function Classification System Level I–II	
5. Good vision and willingness to participate	
6. Complete gait analysis data sets to perform musculo-skeletal modelling	

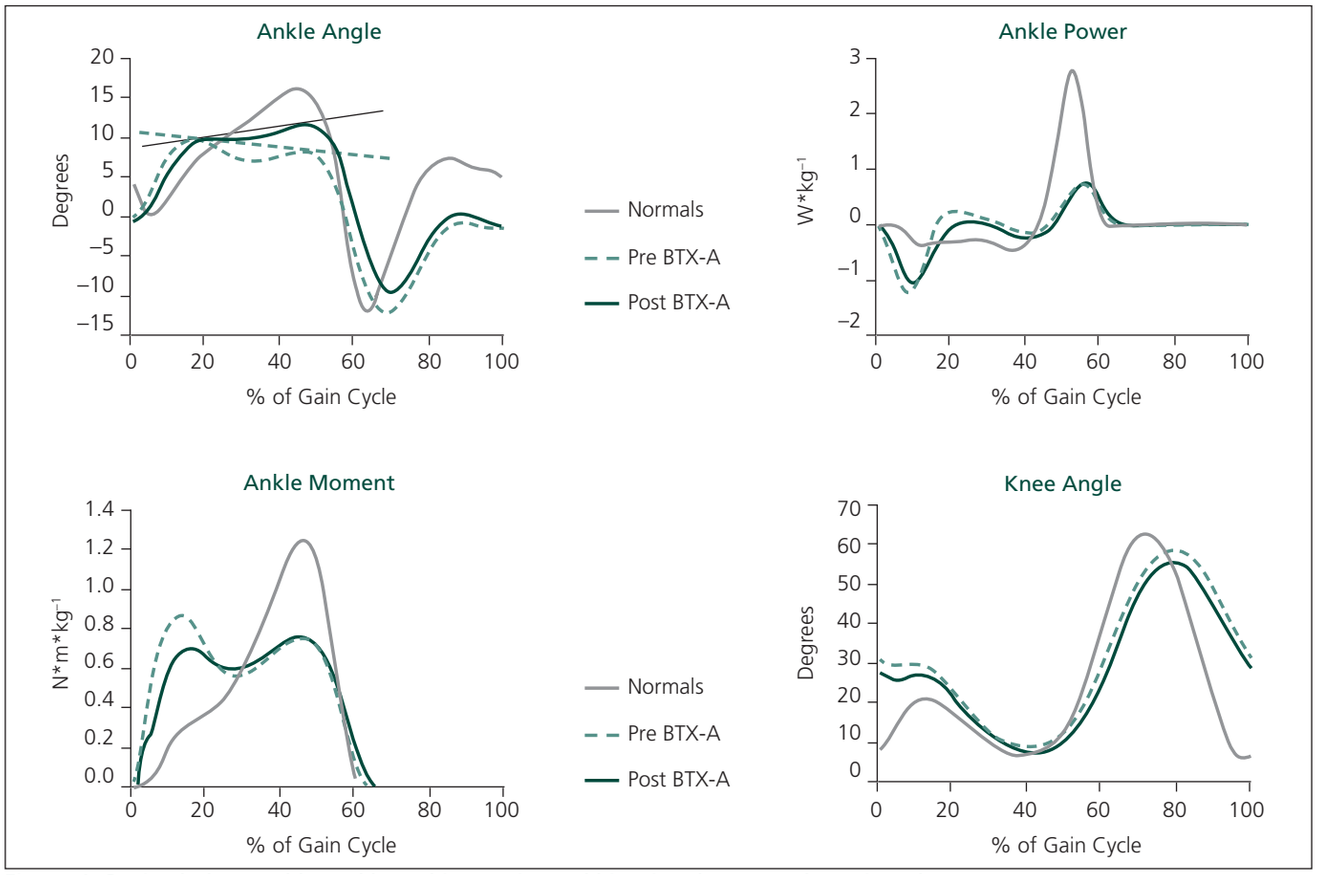

Figure 1. Sagittal plane ankle and knee kinematics and kinetics plots over the gait cycle.

Ankle Angle: the straight lines in the Ankle Angle graph show the slope quotient pre- and post botulinum toxin A application. A rising "slope quotient" indicates improvement of ankle motion. There is an obvious improvement to normal positive values of slope quotient.
Ankle Moment: there is a decrease in abnormal ankle moment during the first part of the single stance phase.
Ankle Power: decrease of abnormal power generation in the first part of the single stance phase.
Knee Angle: there is no significant difference.
(Normals: normally developing children; Pre-BTX-A: patients with cerebral palsy before the botulinum toxin A application; Post BTX-A: patients with cerebral palsy after botulinum toxin A application)

tients walked at self-selected speeds along a 12-metre walkway. For each patient a minimum of five trials, providing clear foot-force plate contact, were captured and averaged. To better describe the different patterns of ankle kinematics during the single stance phase, a "slope quotient" was employed [13]. The slope quotient of the sagittal plane ankle angle during walking was computed as a subtraction of the ankle angle at the beginning of single support from the ankle angle at the end of the single support. Positive values of the slope quotient indicate muscle elongation, negative values indicate muscle shortening and values close to zero show that a muscle has stayed at the same length during the single support phase of the gait cycle.

Muscle length calculation

Based on kinematic data, Mobile GaitLab software (Duisburg-Essen University, Institute of Mechatronics and System Dynamics, Germany) [14] was used to compute individual muscle lengths of the gastrocnemius and soleus. The specification of the lower extremity architecture was based on the origin and insertion coordinate data compiled by Yamaguchi [15]. The model defines the musculoskeletal geometry of a normal adult male. Three-dimensional musculo-tendon (MT) pathways were modelled as a series of straight-line segments extending from origin to insertion. Where appropriate, the MT pathways were sub-sectioned into a number of straight-line segments by introducing via-points. Musculo-tendon length parameters were normalized to allow comparison between subjects.

Statistics

Ankle and knee kinematic and kinetics in the sagittal plane as well as maximal length of the medial gastrocnemius (MGAC), lateral gastrocnemius (LGAC) and the soleus (SOL) muscles over the gait cycle were considered as main outcome measures for this study. Student's T-test (Statistica 6.0, StatSoft, Tulsa, OK) was used to describe the pre- and post-treatment differences.

Results

The walking velocity and the stride length did not change after the BTX-A application. Ankle kinematics and kinetics changed towards normal 10 weeks after the treatment

Table 2. Summary of results

		Pre-BTX				Post-BTX			
	p	Mean	CI 5%	CI 95%	SD	Mean	CI 5%	CI 95%	SD
Slope quotient	**0.001**	–0.489	-2.234	1.256	3.394	4.226	1.997	6.454	4.335
Maximal ankle angle at the end of single support (°)	**0.045**	6.739	3.836	9.642	5.646	11.016	7.781	14.250	6.291
Timing of maximal ankle angle (% of gait cycle)	**0.003**	28.412	21.134	35.690	14.156	42.765	36.469	49.061	12.245
Maximal ankle moment (N × m × kg^{-1})	0.362	0.921	0.807	1.035	0.221	0.854	0.749	0.958	0.203
Timing of maximal ankle moment (% of gait cycle)	**0.024**	23.000	15.085	30.915	15.395	35.588	27.608	43.568	15.520
Maximal power generation in the first part of single support (Watt × kg-1)	**0.012**	0.300	0.181	0.419	0.232	0.094	-0.017	0.206	0.216
Maximal power generation over the gait cycle (Watt × kg-1)	0.486	0.998	0.829	1.166	0.328	0.914	0.728	1.100	0.362
Maximal knee flexion (°)	0.162	58.733	55.566	61.900	6.159	55.169	50.944	59.393	8.216
Maximal knee extension (°)	0.747	7.614	4.251	10.977	6.541	6.897	3.672	10.123	6.273
Maximal length of medial gastrocnemius (% of normalized muscle length)	0.128	101.746	101.572	101.920	0.314	102.101	101.823	102.379	0.502
Maximal length of lateral gastrocnemius (% of normalized muscle length)	0.930	102.494	102.036	102.952	0.827	102.518	102.177	102.859	0.616
Maximal length of soleus (% of normalized muscle length)	0.385	101.863	101.585	102.142	0.503	101.998	101.825	102.171	0.312

The table summarizes the results of pre- and post- botulinum toxin A application analysis of ankle and knee kinematics, kinetics and muscle-tendon lengths of triceps surae. Significant changes in the ankle kinematics document an improvement after BTX-A application. Not only the abnormal power generation in the first part of single support phase of stance normalized, but there was no decrease in maximal power generation, which is the most important factor for forward propulsion in walking. No change in muscle lengths was observed after BTX-A application. (p: level of statistical significance, CI: confidence interval, SD: standard deviation)

(Fig. 1). The maximal ankle angle at the end of single support increased (p = 0.045) and also the timing of the maximal angle shifted towards normal values (Table 2). The ankle angle slope quotient changed to normal positive values (p = 0.001). We did not observe any changes in knee kinematics. While the maximum ankle moment values remained unchanged, the abnormal ankle moment peak in the first part of the single support phase decreased and the timing of maximum ankle moment occurred later in the stance phase (p = 0.024). An abnormal power generation in the first part of the single support stance phase decreased markedly (p = 0.012). Musculo-skeletal modelling revealed no lengthening of the triceps surae muscles compared to the baseline assessment (Table 2). We did not observe any adverse effects in our study group.

Discussion

This study utilised three-dimensional gait analyses to evaluate the effects of BTX-A and serial casting on kinematic and kinetic gait parameters in children with cerebral palsy who exhibit equinus gait. Short-term effects were analysed ten weeks after BTX-A injections. To evaluate changes in dynamic muscle length, musclulo-skeletal modelling techniques were applied. The results of this study confirm previously reported beneficial short-term outcome when equinus gait is treated with BTX-A and serial casting in children with cerebral palsy. Like other authors [7,16] we observed significant improvements of ankle kinematics and kinetics. These improvements include a reduction in equinus and a change of timing and modulation of ankle motion patterns towards normal. During the "second rocker" of ankle motion we documented improved dorsal flexion at the end of the stance phase of the gait cycle [4]. As it has been demonstrated that equinus gait increases the energy expenditure during walking in children with cerebral palsy [17], the improvement of the ankle kinematics might allow for longer walking distances, which could improve the quality of life. The changes in ankle moments and powers observed in this study provide insight into the underlying patho-physiology of the gastro-soleus muscle in spastic equinus gait. Abnormal power absorption was observed whenever a forefoot landing occurred and the ankle was loaded through a forefoot lever. This power absorption can be interpreted as a calf muscle stretch, which is followed by a burst of power generation in the first half of single support. This burst of power generation points towards a spasticity-driven stretch reflex after loading the ankle via a forefoot lever arm. When treated with BTX-A and serial casting the reflex-induced power generation was markedly reduced. In this study, musculo-skeletal modelling revealed no changes in muscle length that could be attributed to the interventions of BTX-A and serial casting. This finding contrasts with previous reports [10,18,19]

that documented changes in calf muscle length as a beneficial short-term outcome. As the changes in kinematics and kinetics cannot be attributed to changes in muscle length, possibly BTX-A affects intrafusal muscle spindles, reducing the effects of the gamma-loop stretch reflex. In a recent study of the long-term effects of BTX-A treatment on muscle tone and range of motion in a group of children with cerebral palsy, Tedroff et al [20] demonstrated that BTX-A was efficient in reducing the muscle tone but the improvement of range of motion lasted for only a short period of time in the gastrocnemius muscle. This evidence would support our conclusion that during the first weeks after the application of BTX-A, the therapy might have a more significant influence on muscle spasticity than on muscle growth. On the other hand, Kanovsky et al showed a lack of deterioration in maximum passive ankle dorsiflexion for both groups in a paper concerning the long-term efficacy and tolerability of 4-monthly versus yearly BTX-A treatment for lower limb spasticity in children with cerebral palsy [21].

The dose of BTX-A used in this trial (7 IU/kg of BTX-A for the triceps surae complex) is rather high when compared in the literature [22,23]. However, a randomized, double-blinded trial demonstrated that high-dose BTX-A treatment has a better effect on gait and spasticity improvement than low-dose BTX-A treatment [24]. Similar dosages of BTX-A have been evaluated by other authors [7,25] who did not observe any increase in adverse effects. There is no general agreement on the use of serial casting together with BTX-A treatment in children with cerebral palsy [26]. It has been shown that serial casting enhanced and prolonged the effect of BTX-A treatment [7,16]. In a more recent study, Newman et al [27] showed additional benefits in the recurrence of spasticity when casting is delayed after BTX-A injections. Based on these supportive data and our personal experience, we adopted this procedure for routine use. The cast is not applied immediately after BTX-A administration but on the same day (approximately four to five hours after injecting BTX-A). This time is used to move and stretch the triceps surae muscle. It has been proved in an animal model that as few as 10 passive stretch/relax cycles can increase the efficacy of BTX-A [28]. However, to the best of our knowledge, no published data confirms this finding in a group of children with cerebral palsy. On the other hand, electrical stimulation has been proved to prolong gait improvement in children with spastic diplegic cerebral palsy [29].

Management of cerebral palsy is primarily directed towards maintaining or regaining muscle length. Use of BTX-A here is based on the concept that muscle fibres in children with spastic cerebral palsy are short, and weakening the muscles with BTX-A injections might allow them to be stretched and thus grow in length. Thus the progression from dynamic to fixed deformity may be delayed [30] and any orthopaedic surgical procedures might be postponed or reduced in frequency [31]. Although muscle growth following BTX-A injection has also been found in animal models [9,32], the findings of our study did not support this in children with cerebral palsy in a short-term follow up. As there is no direct way to measure muscle length in humans, muscle length calculations are based on mathematical models of the lower extremities and on measurements of ankle, knee, hip and pelvis kinematics. It is obvious that such mathematical modelling approaches have limitations, but the method is well accepted in the literature [25,33] because it provides additional information which can help us to better understand the biomechanics of a spastic muscle. We speculate that a period of ten weeks is too short to expect any measurable gain in muscle length by muscle growth. However, we observed significant improvements in ankle kinematics and kinetics. We interpret the beneficial changes in kinematics and kinetics observed in this study as an effect of BTX-A on intrafusal muscle fibres [34]. Therefore, the application of BTX-A may have a more significant influence on muscle spasticity than on muscle growth. The BTX-A is effective in reducing the over-activity of the spastic muscle and improving its function [35]. Moreover, even if the main effect of BTX-A might be to weaken the muscle, we observed no decrease in power generation in terminal stance, which is the most important factor for forward propulsion in walking. Again, this could be explained by different influences of BTX-A on intrafusal and extrafusal muscle fibres. Such differences in outcome might be attributed to the variety of methods used to calculate muscle lengths in the various studies.

In conclusion, this study demonstrated beneficial functional changes in ankle kinematics and kinetics after the application of BTX-A when combined with serial casting. We did not observe any change in the peak muscle-tendon length of the gastro-soleus muscle complex. Further studies are needed to address the question of muscle growth after BTX-A application in children with spastic cerebral palsy.

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STUDY 4

Detecting postoperative change in children with cerebral palsy:

Net nondimensional versus body mass oxygen normalization

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Abstract

The aim of the study is to investigate if the net nondimensional oxygen utilization scheme is able to detect postoperative improvement in the energy cost of walking of children with cerebral palsy and compare it to body mass normalization scheme. We evaluated ten children with spastic cerebral palsy pre- and nine months after equinus deformity surgery. Participants walked at a given speed of 2 $km.h^{-1}$ and 3 $km.h^{-1}$ on a treadmill. Oxygen utilization was measured, mass relative VO_2 and net nondimensional VO_2 were calculated. Coefficient of variation was used for the description of variability among subjects. Postoperatively, gait kinematics normalized and the mass relative VO_2 and net nondimensional VO_2 showed significant improvement. Net nondimensional VO_2 is able to detect postoperative improvement with smaller variability among subjects than body mass related normalization in children with cerebral palsy.

Introduction

Cerebral palsy (CP) describes a group of permanent disorders of the development of movement and posture causing activity limitation, that are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain. Deformity, impaired function and limitation in mobility are key features of many of the children with cerebral palsy, which lead to referral to an orthopaedic surgeon (Young & Wright, 1995). Measuring physical function is more difficult than measuring deformity, and measures of deformity are often used as surrogate measures for function (Graham et al., 2004).

Normal gait depends on an efficient use of energy, something that kinematic and kinetic gait data cannot directly measure. Conservation of energy is a typical aspect of walking. Children with cerebral palsy have been shown to expend greater energy during walking than their typically developing peers when walking at self-selected economical (Rose et al., 1990) as well as a given speed (Rose et al., 1993). Many factors influence energy expense while walking, including spasticity, bony deformity, strength and selective motor control. Thus the energy cost of walking reflects the cumulative effect of many factors (Waters & Mulroy, 1999).

To quantify the energy cost of walking, oxygen consumption was used in previous studies (Dahlback & Norlin, 1985; Piccinini et al., 2007; Rose et al., 1989), because it gives an objective view of the overall efficacy of the patient's gait. Schwartz pointed out the need to have a utilization measure independent of children's age, weight and height in order to allow efficiency to be compared between subjects. Therefore Schwartz introduced a nondimensional normalization scheme for oxygen utilization data (Schwartz et al., 2006). Nondimensionalisation is a form of normalization that renders parameters independent of original units of measurement. It decreases or even removes the impact of key anthropometric and physiological variables on the energy variables. This is important when comparing the energy cost of walking in children of different ages and morphology (Thomas et al., 2009). The nondimensional normalization scheme was found to be superior to body mass normalizaion, because it is only about one-fourth as sensitive to mass, height and age changes, and therefore provides a much more robust, reliable and properly interpreted measure of efficiency. (Schwartz et al., 2006). However, this scheme has not yet been used in assessing the effectiveness of soft-tissue surgery in children with cerebral palsy.

The aim of this study is to investigate if the nondimensional oxygen utilization scheme is able to detect postoperative change in the energy cost of walking in children with cerebral palsy and to compare it to body mass normalization scheme.

Methods

Subjects

Ten children (4 girls, 6 boys) were included in this prospective study. The average age of our group of patients at the time of surgery was 8.4±3.1 years (mean±SD). Six children had a diplegic and 4 hemiplegic topographic distribution of spasticity. Partial body weight support reduces the oxygen cost of walking (Unnithan et al., 2006). Therefore patients who were not able to walk on a treadmill without continuous holding of the bars were excluded. They were only allowed to touch the bars momentarily to maintain their balance. Two of the children used forearm crutches as their normal walking aid, but all participants were able to fulfill the above mentioned criteria.

Children were community ambulators and were designated for gait corrective surgery for fixed equinus deformity. Two patients underwent additional adductor longus lengthening. All the other contractures were assesed as dynamic (rectus femoris, hamstrings) and were addressed by conservative treatment. Patients suitable for corrective bony procedures and patients treated with botullinum toxin in the last six months were excluded. Parents' approval of the study was given in written form and children agreed verbally. This study was approved by the local Ethical committee.

Study design

Children were evaluated preoperatively and nine (9.33±0.64) months postoperatively. Based on our previous study, the nine months period is a sufficient time for functional recovery after a surgery in children with cerebral palsy (Švehlík et al., 2008). Based on the findings that reliable physiologic and metabolic variables may be collected in subjects with mild cerebral palsy after one treadmill walking practice session (Maltais et al., 2003), exercise testing and gait analysis were carried out in two sessions at the same time of the day on separate days.

The first session (introductory visit) consisted of a physical examination, assessment of joint contractures, weight, height, lower limbs lengths and body adiposity assessment. Leg length was measured as the distance between the spina illiaca anterior superior and the tip of the medial malleolus using the medial condyle of the femur as a reference point if the knee extension was restricted.

Familiarization with walking on a treadmill (Marquette T2000) and respiratory gas analysis system (Oxycon beta, Jaeger, Hoechberg, Germany) was performed during 15 minutes of

walking. Ability of participants to walk without holding on to bars at a given speed of 2 $km.h^{-1}$, 3 $km.h^{-1}$ and the velocity of comfortable walking speed were tested.

The second session took place a day after the introductory visit and selected parameters were recorded at rest and at a given speed of 2 $km.h^{-1}$ and 3 $km.h^{-1}$ at 0% gradient on treadmill. The monitored parameters were: oxygen consumption (VO_2) and carbon dioxide production (VCO_2). The derived parameters were gross oxygen consumption relative to body mass (mass relative VO_2), nondimensional VO_2 (Schwartz et al.) normalized to leg length (Equtation 1) and respiratory exchange ratio (RER). The steady state was defined as a variation of both VO_2 and respiratory exchange ratio lower than 10% (McArdle et al., 2006) and was expressed as the average of the last 30 seconds of the four minutes stage. The steady state was reached in all patients.

Gait analysis was used to provide an objective measure of the postoperative improvement. Instrumented gait analysis was performed with eight-camera video based motion capture system (Proreflex 240Hz, Qualysis, Sweden), and provided three-dimensional kinematic and temporal-spatial parameters. Changes in the joint angles over the gait cycle were calculated using a seven-segment model using Euler angles (Visual3D, C-motion, USA).

Postoperatively, below-knee plaster casts were applied to all patients for six weeks. Physiotherapy followed the same protocol for all the patients and continued on an out-patient basis for the whole period of our study.

Statistical analysis

Statistical analysis was done using STATISTICA 6 (StatSoft, Tulsa, USA). Descriptive data are referred as mean±SD. Student's paired t-test for dependent samples was used to compare pre- and postoperative values. *P*-value of less than 0.05 was considered statistically significant. Coefficient of variation (computed as standard deviation / mean) for the both normalization methods was computed. Spearman rank correlation coefficient was used to assess the relation between the two methods.

Results

The average height of our patients increased by mean 3.8 cm (range 1.7-5.3 cm), leg length increased by 2.6 cm (range 1.2-3.5 cm) and body weight increased by 1.9 kg (range 0.2-4.1 kg) during the postoperative period. The average comfortable walking speed (2.025 $km.h^{-1}$) did not change after the surgery. The level of Gross Motor Function Classification system was II-III (Palisano et al., 1997).

Gait analysis demonstrated normalization of the ankle motion. The ankle position at the initial contact changed from 9.6˚ of plantarflexion to 1.7˚ of dorsiflexion (p=0.001) and the maximal dorsiflexion during the stance phase of the gait cycle increased from 1.7˚ to 12.2˚ (p=0.022). Knee flexion angle at initial contact decreased from 33.2° to 21.1° (p<0.0001). During single limb support, the knee flexion angle also decreased by 14.5° (p<0.0001).

There was no difference between pre- and postoperative resting VO_2 and respiratory exchange ratio. Oxygen utilization data are shown in Figure 1. There were significant decreases in both mass relative and nondimensional VO_2 parameters at 2 $km.h^{-1}$ as well as at 3 $km.h^{-1}$. Preoperative and postoperative coefficients of variation of the net nondimensional VO_2 were notably lower at both speeds when compared to mass relative VO_2 (Table 1). The Spearman rank correlation revealed a significant association between mass relative and net nondimensional VO_2 at both speeds preoperatively, as well as postoperatively.

	Mass relative and Net nondimensional VO_2 correlation	
	Spearman R	***P*-level**
2 $km.h^{-1}$ Pre OP	0.8788	0.0008
2 $km.h^{-1}$ Post OP	0.7697	0.0092
3 $km.h^{-1}$ Pre OP	0.8666	0.0024
3 $km.h^{-1}$ Post OP	0.7000	0.0358

Table 2. Spearman rank correlation of the body mass relative and net nondimensional VO_2 normalization.
(Pre OP = preoperatively, Post OP = postoperatively)

Discussion

Energy expenditure as measured by oxygen consumption can be considered as an objective tool for assessment of functional ability. It is an objective tool because it does not rely on parental or patient report and is also functional because its interpretation provides an indication of endurance, fatigue and ability to accomplish the routine daily task of locomotion. Fixed selected speeds of 2 $km.h^{-1}$ and 3 $km.h^{-1}$ should be comparable to speeds perceived in the patient's everyday life (Unnithan et al., 1996). While resting oxygen consumption remained unchanged, gross mass relative oxygen consumption decreased and thus efficiency of walking increased. Postoperatively, the gait kinematics also considerably improved.

Most of the oxygen, especially during locomotion or other exercise is used in energy metabolism. Lower oxygen consumption point out the lower energy demand and thus longer time till fatigue occurs. In contrast to healthy persons, the major reason for the increased energy cost of locomotion in children with cerebral palsy is ineffective gait, due to simultaneous activation of antagonist muscles (Unnithan et al., 1996; Unnithan et al., 1999). The preoperative values of VO_2 in our group of children with cerebral palsy are considerably higher in comparison to values of the population of healthy children but similar to previously published data on children with cerebral palsy (Rose et al., 1993). Morover, the comfortable walking speed is more than double in healthy subjects (Waters & Mulroy, 1999).

Because of the strong dependence of gross oxygen consumption on anthropometrical data there is a need for normalization. Gross oxygen consumption relative to body mass ($ml.kg^{-1}$) is the most widely used parameter representing the intensity of physical effort during exercise. The mass relative normalization scheme provides widely accepted but not entirely effective normalization and its performance decreases with increasing height, mass and age (Schwartz et al., 2006). The net nondimensional normalization scheme, using the nondimensional variables described by Hof (Hof, 1996), is essentially independent of all three above mentioned factors. This scheme was shown to perform better than standard mass relative normalization in able-bodied children (Schwartz et al., 2006) and it was also used to evaluate gait efficiency in children with cerebral palsy (Brehm et al., 2008). To our knowledge, it has not been used to detect the improvement of gait efficiency after the soft-tissue surgery in children with cerebral palsy.

Our results demonstrate that the net nondimensional normalization enables a better pre- and postoperative comparison of energy consumption in children with cerebral palsy of different ages and anthropometrical parameters. This was reflected by the lower variability of the net nondimensional VO_2 at both speeds when compared to mass relative VO_2. Moreover, the Spearman rank correlation pre- and postoperatively indicates a close association between the two normalization methods.

In conclusion, the net nondimensional VO_2 was able to detect the postoperative improvement of gait efficiency with smaller variability compared to standard mass relative normalization. Because of its independance on age, weight and height it might be recommended in studies concerning children with cerebral palsy.

Velocity	Parameter	*P*-level	Pre OP	CV Pre OP	Post OP	CV Post OP
2 km.h^{-1}	Mass relative VO_2	0.0498	19.4±4.3	1.4108	17.8±2.7	0.9010
	Net nondimensional VO_2	0.0068	0.57± 0.17	0.0531	0.50± 0.11	0.0375
3 km.h^{-1}	Mass relative VO_2	0.035	24.0± 3.3	0.9971	22.5± 2.5	0.8508
	Net nondimensional VO_2	0.0333	0.76± 0.18	0.0556	0.70± 0.10	0.0351

Table 1. Comparison of *P*-levels, coefficients of variation and confidence intervals of mass relative VO_2 ($ml.min^{-1}.kg^{-1}$) to nondimensional VO_2. (Pre OP = preoperatively, Post OP = postoperatively, CV = coefficient of variation).

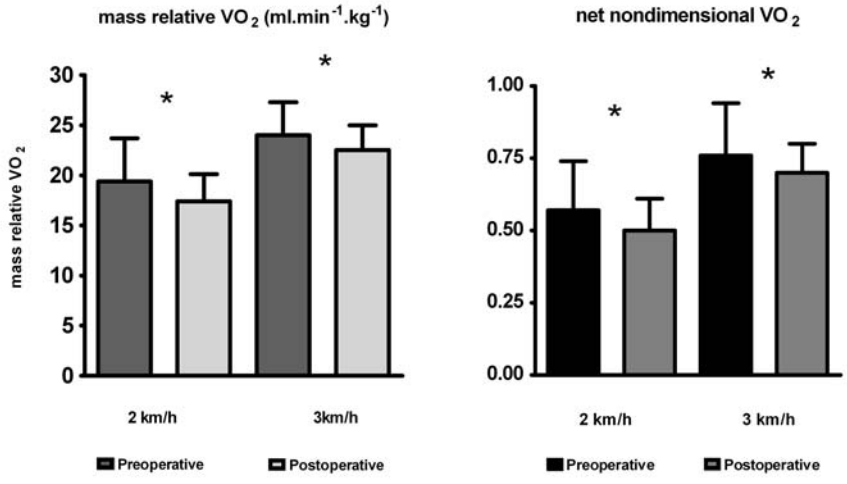

Figure 1. Comparison of pre- and postoperative oxygen consumption results using two different normalization schemes (* indicates the statistical significance at the *P*<0.05).

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STUDY 4

Energetická náročnost chůze u pacientů s dětskou mozkovou obrnou: proč a jak ji měřit.

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Abstract

One of the main goals of therapy in cerebral palsy (CP) is improvement of quality of life and making activities od daily life easier. Key role in both is improvement of independence and physical functioning which depends on quality of ambulation and fatigability to great extent. Both these factors depend on energy cost of walking. Energy cost may be assesed with exercise test. Parameters of oxygen utilization and heart rate are measured and computed. If available, should we prefer oxygen utilization parameters over heart rate measurement and treadmill over free surface testing. The most sensitive parametersare oxygen cost (VO_2 / speed in $m.min_{-1}$) and Physiological Cost Index (heart rate acceleration /speed). The advantage of oxygen utilization data is a good reproducibility. Measurement of heart rate is easy and feasible in field. Reference values are available. Energy cost of walking reflects postoperative development and may be used in prescription of assistive devices.

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Energetická náročnost chůze u pacientů s dětskou mozkovou obrnou: proč a jak ji měřit.

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❒ Souhrn

Cílem konzervativní i chirurgické léčby pacientů s dětskou mozkovou obrnou (DMO) je udržení kvality života a zařazení pacienta do kolektivu. Nedílnou součástí je zlepšení samoobslužnosti, na kterou má zásadní vliv kvalita chůze a unavitelnost při běžných aktivitách. Oba tyto faktory do značné míry závisejí na energatické náročnosti chůze. Energetickou náročnost můžeme odhadnout zátěžovým vyšetřením. Při testu měříme a následně počítáme parametry související se spotřebou kyslíku nebo s tepovou frekvencí. Je-li dostupné vybavení, testujeme na běhátku, měříme spotřebu kyslíku a následně počítáme parametr O_{2cost}, což je kyslík spotřebovaný na přemístění se o 1m. Výhodou je nastavení intensity a dobrá reproducibilita. V opačném případě měříme tepovou frekvenci a počítáme parametr Physiological Cost Index (PCI), což je vzestup tepové frekvence potřebný s přemístěním o 1m. Testovat můžeme na volné podlaze. Výhodou je jednoduchost měření a možnost testovat v terénu. Pro oba zmíněné parametry jsou k dispozici referenční hodnoty. Energetická náročnost chůze dobře odráží zlepšení v pooperačním průběhu a lze ji využít také při výběru ortopedických pomůcek.

❒ Summary

Slabý, K., Švehlík, M., Trč, T., Radvanský, J.: Energy cost of walking in children with cerebral palsy.

One of the main goals of therapy in cerebral palsy (CP) is improvement of quality of life and making activities od daily life easier. Key role in both is improvement of independence and physical functioning which depends on quality of ambulation and fatigability to great extent. Both these factors depend on energy cost of walking. Energy cost may be assesed with exercise test. Parameters of oxygen utilization and heart rate are measured and computed. If available, should we prefer oxygen utilization parameters over heart rate measurement and treadmill over free surface testing. The most sensitive parameters are oxygen cost ($\dot{V}O_2$ / speed in $m.min^{-1}$) and Physiological Cost Index (heart rate acceleration / speed). The advantage of oxygen utilization data is a good reproducibility. Measurement of heart rate is easy and feasible in field. Reference values are available. Energy cost of walking reflects postoperative development and may be used in prescription of assistive devices.

Úvod

Dětská mozková obrna (DMO) je zastřešujícím názvem pro skupinu poruch motorického vývoje dítěte, které vznikají jako důsledek neprogresivní léze nezralého centrálního nervového systému v perinatálním období. Porucha motorického vývoje je často provázena poruchami smyslového vnímání, čití, komunikace, chování, epilepsií a sekundárními deformitami pohybového aparátu (17). Tato definice zahrnuje širokou škálu klinických manifestací, které se mohou lišit

v etiologii, projevech i tíži postižení. Incidence je 1,5–2,5 na 1000 živě rozených dětí (9), tzn. že v České republice žije asi 30 000 dětí do 15 let věku s diagnostikovanou dětskou mozkovou obrnou. Nejčastější formou DMO je forma spastická, která se dále dělí dle topografické distribuce postižení. Všichni pacienti s hemiparézou, 90% s diparézou a asi 50% pacientů s tetraparetickým postižením jsou schopni chůze (18). Spasticitu můžeme definovat jako patologickou mimovolní svalovou aktivitu, která se vyskytuje na podkladě postižení centrálního motoneuronu (3). Spasticita se projevuje abnormálním svalovým napětím, zvýšenými šlachovo-okosticovými reflexy, přetrváváním primitivních reflexů ale také změnou stavby a růstu příčně pruhované svaloviny. Ve svalových biopsiích pacientů s DMO se nachází predominance prvního typu svalových vláken současně s fibrotizací svalu a jeho tukovou atrofií (16). Proto je jedním ze základních problémů vedoucím k poruchám hybnosti snížená síla maximální svalové kontrakce a porucha selektivní kontroly hybnosti. Na myších modelech bylo prokázáno, že spastický sval dorůstá pouze 55% délky kosti (25), což vysvětluje spolu se změnami architektury svalu vznik kontraktur vedoucí k omezení hybnosti jednotlivých kloubů.

Lidská chůze je cyklicky se opakující a energeticky velmi úsporný proces lokomoce, kdy vzpřímená postura je střídavě zajišťována oběma dolními končetinami. Tento způsob bipedální lokomoce je typický pouze pro člověka. Základní předpoklady normální chůze byly definovány Gagem (4):

1) zajištění stability stojné končetiny a celého trupu v gravitačním poli
2) dostatečná elevace nohy během švihové fáze
3) správné nastavení nohy během závěru švihové fáze
4) adekvátní délka kroku
5) minimalizace energetické náročnosti chůze

U dětí s DMO se nachází porucha stereotypu chůze ve všech výše uvedených bodech. Příčinou je špatná selektivní kontrola hybnosti, spasticita, svalová dysbalance a přítomnost svalových kontraktur a deformit dolních končetin. Pacienti s DMO mají nižší rychlost chůze ve srovnání s vrstevníky a neekonomičnost jejich pohybu vede k brzké únavě. Bylo prokázáno, že ko-kontrakce (současná aktivace agonistické i antagonistické skupiny svalů) je hlavním faktorem zvýšené energetické náročnosti chůze (23). Pacienti s DMO spotřebovávají až třikrát více energie při běžné chůzi než jejich vrstevníci (1). Jejich pohyb se dá přirovnat svou náročností chůzi zdravého člověka v hlubokém sněhu. Proto je minimalizace energetické náročnosti chůze jedním ze základních cílů léčby DMO.

Přestože je vyšetření energetické náročnosti chůze důležitou součástí sledování a léčby pacientů s DMO, není jedinou indikací k zátěžovému vyšetření. Standardní zátěžový test lze provést z běžných indikací jako u ostatní populace. Zátěžový test se dále provádí u sportovců s DMO k určení zdatnosti, což obnáší jak zjištění maximální aerobní kapacity, tak anaerobní testy.

Způsob zatížení

Kde testovat

Vzhledem k tomu, že se u DMO při testování snažíme co nejvíce přiblížit podmínkám v běžném životě pacientů, je potřeba pečlivě zvažovat i způsob zatížení. Pro testování chůze se nejčastěji používá pohyblivý pás (běhátko) nebo samostatná chůze, buď po rovném povrchu na dlouhé chodbě, nebo v terénu.

Při vyšetření na pohyblivém pásu lze přesně nastavit intensitu zátěže, ale testovat lze pouze v laboratoři a ne každá zátěžová laboratoř je vybavena vhodným běhátkem. V redukované podobě lze testování provést i na rehabilitačních přístrojích, které bývají k disposici v lázních. Charakter chůze na pohyblivém pásu není plně shodný s chůzí na pevné podložce. Přirozené tempo

volné chůze ani její rychlost nebývají rovnoměrné. Pokud má pacient senzorický deficit, problémy s rovnováhou, čím je mladší nebo méně spolupracující, tím větší je pravděpodobnost, že se vyšetření nepodaří dokončit. Nicméně, na základě literárních údajů (14, 15, 22) i z vlastní zkušenosti (21) soudíme, že lze testovat děti s DMO přibližně od 5 let věku. Před měřením je potřebná zacvičovací návštěva, kde je pacient seznámen s technikou chůze na běhátku i se samotným průběhem měření. Mnoho pacientů v ČR má již s chůzí na pohyblivém pásu předchozí zkušenost z lázní. Literatura uvádí, že stačí jedna zacvičovací návštěva den před plánovaným vyšetřením k tomu, aby výsledky následujících měření byly již beze změny (11). Pro přiblížení se běžným životním podmínkám testujeme často pacienty s DMO při jimi zvolené komfortní rychlosti. Tuto rychlost musíme určit dopředu, nejlépe na závěr zacvičovací návštěvy. Případné měření komfortní rychlosti bezprostředně před vlastním zátěžovým vyšetřením by mohlo zkreslit výsledky testu.

Podmínky pro vyšetření chůze po pevné podložce bývají obdobné jako např. při známém šestiminutovém chodeckém testu (6MWT). Používá se dlouhá světlá rovná chodba. Takové podmínky nebývá problém zajistit, jen je třeba se pokusit o striktní standardizaci, kdy pacient není nadměrně pobízen ani jinak ovlivňován v průběhu testu. Při striktním provedení by ho ani neměl nikdo doprovázet, což může mírně zvýšit riziko pádu. U hůře spolupracujících nebo úzkostných pacientů se v neznámém prostředí s rostoucí vzdáleností od doprovázející osoby obvykle snižuje pocit jistoty a zhoršuje se soustředění. Další nevýhodou je obtížné dodržení stanovené rychlosti. Toto omezení odpadá při testování pacientem zvolenou (komfortní) rychlostí. Naopak nespornou výhodou je možnost testovat za použití různých pomůcek.

Měření v terénu je obdobou chůze po pevné zemi s tím, že je možné testovat pacienty v jejich důvěrně známém prostředí při specifických činnostech, které nás mohou zajímat. Takové vyšetře-

ní je potřeba sestavit pacientovi „na míru“, což automaticky znamená nestandardní podmínky a výsledky nejsou aplikovatelné na jiné situace nebo pacienty.

Intenzita zátěže

Pacienty s DMO je vhodné testovat konstantní zátěží, protože při kontinuálně zvyšované zátěži by hrozilo riziko pádu. Toto riziko lze omezit použitím bezpečnostního závěsného aparátu nebo oporou o madla běhátka, ale takové odlehčení není vhodné, protože snižuje energetickou náročnost chůze (24), a tudíž interferuje s vlastním měřením.

Z důvodu lepší zaměnitelnosti běhátka a terénu (zejména při testování s pomůckami) volíme konstantní sklon 0% a intensitu zátěže regulujeme pomocí rychlosti.

Rychlost je možné dopředu určit, nebo lze pacienta nechat zvolit si jemu pohodlnou (komfortní) rychlost (CWS). Při určení rychlosti je třeba brát v úvahu, že příliš nízká rychlost je subjektivně nepříjemná, vzrůstá práce statického charakteru a naměřené hodnoty potom mají vysoký rozptyl a málo odpovídají nastavené intenzitě zátěže. Naopak při příliš vysoké rychlosti hrozí předčasná únava nebo nedokončení testu. Nejčastěji používané rychlosti jsou 2 a 3 km/h, popř. 4 km/h. Pevně stanovená rychlost je výhodná pro opakovaná měření v průběhu sledování, kdy změna měřených parametrů přímo ukazuje na změnu schopnosti vypořádat se se zátěží. Chcemeli srovnávat pacienty různého věku, a tedy s odlišným vzrůstem a délkou kroku, může být vhodnější rychlost volená relativně, např. k maximální rychlosti chůze. Nevýhodou takového postupu je nutnost další návštěvy ke stanovení dalšího parametru nebo alespoň dlouhého zotavení, což prodlužuje čas potřebný k provedení testu. Komfortní rychlost je považována za rychlost s nejmenšími energetickými nároky, každý pacient si ji volí sám a není potřeba další měření. Nevýhodou je, že se při opakovaných návštěvách může lišit z jiných důvodů, než je změna energetické náročnosti (směrodatná odchylka referenčních hodnot CWS pro děti a adolescenty je přibližně 10m/min (15)).

Měřené parametry

Bioenergetická náročnost daného pohybu je závislá na objektivních faktorech (např. rychlost chůze) a na faktorech vnitřních (např. biomechanické podmínky, složení pracujícího svalu, koordinace zapojování jednotlivých svalů). Zjednodušeně nahlíženo, objektivní faktory určují vnější (fyzikální) podávaný výkon, zatímco vnitřní faktory určují metabolickou energii spotřebovanou na dosažení a udržení tohoto výkonu. U chůze je, narozdíl od některých jiných typů pohybu (např. bicyklový ergometr), poměrně obtížné stanovit fyzikální výkon. Mnohem jednodušší je vztažení spotřebované energie k objektivnímu parametru. Nejčastěji je takovým parametrem rychlost chůze nebo celková vzdálenost. Také přímé měření spotřebované energie (přímá kalorimetrie) je obtížně proveditelné, a proto měříme parametry se známým vztahem ke spotřebované energii (nepřímá kalorimetrie). Nejčastěji měříme spotřebu kyslíku nebo tepovou frekvenci. Přestože v tomto případě lze vypočítat energetický výdej v kilojoulech, bývá často jednodušší a srozumitelnější změřenou hodnotu nepřevádět na odpovídající energii.

Tepová frekvence

Velmi jednoduchým a levným způsobem, jak odhadnout intensitu zátěže, je měření tepové frekvence. Při odhadu energetické náročnosti se využívá znalosti, že spotřeba kyslíku až do stresového prahu roste přibližně lineárně se vzestupem srdečního výdeje a že se v tomto rozsahu příliš nemění systolický objem. Měřením tepové frekvence se tedy odhaduje spotřeba kyslíku, ze které by se s určitou chybou odvodila spotřebovaná energie. Proto se přepočty nepoužívají a uvádí se přímo tepová frekvence nebo od ní odvozené parametry. Obdobně jako u zdravých připadají v úvahu tyto parametry: tepová frekvence (HR) a její vzestup (HR_{net}), relativní parametry jako procento maxi-

mální tepové frekvence (%HR_{max}) a procento tepového rozpětí (%HRR) nebo parametr nazvaný Physiological Cost Index (10) (PCI). Tento parametr vyjadřuje nárůst tepové frekvence vůči klidovému stavu potřebný pro překonání vzdálenosti 1m. PCI je podobný parametru Energy Expenditure Index, konkrétně verzi založené na tepové frekvenci (EEI_{HR}). Měření tepové frekvence je jednoduché, pro výpočet PCI dokonce postačí prosté počítání pulzů bezprostředně po skončení zátěže (14), a lze tedy uvažovat o měření v terénu samotným pacientem nebo jeho rodiči. Další jednoduchou možností je použití monitoru tepové frekvence (tzv. sporttesteru).

Odhad energetické náročnosti pomocí tepové frekvence má určité omezení v tom, že vztah mezi tepovou frekvencí a spotřebou kyslíku na malých intensitách a okolo stresového prahu není lineární, a navíc směrnice lineárního úseku (strmost vzestupu) není univerzálně platná a pro přesnější měření by bylo třeba ji individuálně zjišťovat.

Spotřeba kyslíku

Ze spotřeby kyslíku po dosažení rovnovážného stavu a znalosti metabolického ekvivalentu pro kyslík lze odhadnout energii potřebnou pro vykonání daného pohybu (udržení daného výkonu). Za předpokladu, že účinnost buněčného energetického metabolismu se u DMO příliš neliší od zdravé populace, můžeme rozdíly ve spotřebě kyslíku přisuzovat změněným biomechanickým podmínkám a změnám v architektuře svalu.

Základním měřeným parametrem je celková spotřeba kyslíku ($\dot{V}O_{2gross}$) při dané intensitě zátěže. Vzhledem k menší aktivní tělesné hmotě u pacientů s DMO je nižší i klidová spotřeba kyslíku ($\dot{V}O_{2rest}$), a při stejné $\dot{V}O_{2gross}$ by tedy byla energetická náročnost podhodnocená. V případě, kdy dominuje zvýšený svalový tonus, může být naopak $\dot{V}O_{2rest}$ zvýšená a energetická náročnost nadhodnocená. Pro citlivější hodnocení energetické náročnosti je proto nutné vypočíst čistou spotřebu kyslíku ($\dot{V}O_{2net}$), což je rozdíl mezi celkovou a klidovou spotřebou. Chceme-li porovnávat zátěž stejné subjektivní intensity (např. zátěž subjektivně pociťovanou jako lehkou), musíme mít na paměti, že absolutní intensita této zátěže je závislá na celé řadě faktorů (typ pohybu, tělesná hmotnost, tělesná výška, věk nebo zdatnost). Ve srovnání se zdravou dětskou populací je u pacientů s DMO odlišná jak absolutní maximální intensita zátěže (např. maximální rychlost chůze či běhu), tak srovnatelná relativní intensita zátěže (např. chůze pacientem spontánně zvolenou komfortní rychlostí). Intenzita zátěže je u dětí s DMO mnohem více závislá na typu pohybu než u zdravé populace.

Pro lepší ilustraci energetické náročnosti chůze při různých rychlostech se klinicky osvědčily ještě další vypočtené parametry, nejčastěji se udává spotřeba kyslíku vztažená k rychlosti (O_{2cost}). O_{2cost} vyjadřuje, nárůst spotřeby kyslíku oproti klidu, potřebný k překonání vzdálenosti 1m. Vypočítá se jako ($\dot{V}O_{2net}$ / rychlost) nebo ($\dot{V}O_{2net}$. celkový čas / celková vzdálenost). Testujeme-li při individuálně zvolené rychlosti, je O_{2cost} jedním z nejcitlivějších parametrů.

Diskuse

Stanovení energetické náročnosti chůze je objektivní metodou, vhodnou jak k zachycení funkčních změn po korekčních operacích, tak i k objektivizaci výsledů konzervativní léčby. Gage (4) zařadil minimalizaci energetické náročnosti jako jeden ze základních předpokladů lidské chůze. Jedná se o funkční parametr, který v sobě zahrnuje jak pathobiomechaniku chůze, tak poruchu řízení motoriky, a odráží se tak v běžném životě pacienta. Cílem rehabilitace, preskripce rehabilitační pomůcky nebo operačního řešení by neměla být „pouze“ korekce deformity, ale také umožnit pacientovi s DMO běžný život mezi vrstevníky. Proto je vhodné zařadit vyšetření energetické náročnosti chůze do schématu předoperačního plánovaní i pooperačního sledování pacientů s DMO.

Stanovení energetické náročnosti chůze pomocí spotřeby kyslíku není vždy dostupné a ne všichni pacienti jsou schopni dostatečné spolupráce. Naproti tomu monitorace tepové frekvence je lev-

ný a jednoduchý způsob hodnocení energetické náročnosti.

Absolutní hodnoty jako např. HR při určité rychlosti mají velký rozptyl a nejsou pro ně dostupné referenční hodnoty. To má za následek, že nelze stanovit, jaký rozdíl je již signifikantní. Nicméně je lze použít pro krátké sledování, kde potřebujeme pouze hrubě orientační hodnocení a nepředpokládáme velké změny stavu pacienta a jeho antropometrických parametrů.

Relativní parametry jako %HRR obecně nejsou příliš vhodné, protože chybí horní referenční hodnota. Je sporné, jestli touto hodnotou má být HR_{max}, protože velkou část pacientů s DMO ani nelze na běhátku zatížit do maxima. Predikovaná HR_{max} nebo nejvyšší dosažená tepová frekvence (HR_{peak}) také nejsou vhodné, protože do výpočtu vnášejí další chybu.

Nejlepším parametrem založeným na tepové frekvenci je PCI, ať už se test provádí při komfortní rychlosti nebo při fixních rychlostech. Důvodem je i to, že jako pro jediný z ukazatelů energetické náročnosti chůze založených na tepové frekvenci existují pro PCI referenční hodnoty (10, 14, 15). Referenční hodnoty od různých autorů se ale mezi sebou liší i o desítky procent, věkové složení souborů není zcela srovnatelné a autoři většinou uvádějí hodnoty dohromady pro všechny věkové skupiny. Vzhledem k tomu, že se s věkem klidová tepová frekvence snižuje, a navíc její měření v laboratorních podmínkách může být zatíženo chybou, je důvod domnívat se, že se s věkem mění přinejmenším rozptyl PCI. Některé práce z poslední doby navíc zpochybňují použití PCI jako takového (6, 8, 13). Pro orientační měření, a zejména tam, kde není k disposici měření spotřeby kyslíku, je nicméně odhad energetické náročnosti pomocí PCI vhodný. V takovém případě bychom volili referenční data podle Rose et al. (15), protože byla získána na přiměřeně velkém souboru (N=102) pokrývajícím věkové rozmezí 6–18 let a rychlost chůze od pomalé (37 ± 10m/min, průměr ± směrodatná odchylka) přes komfortní (70 ± 11) po rychlou (101 ± 13), a to jak na běhátku, tak na zemi. Naopak výhodou měření spotřeby kyslíku je dobrá přesnost odhadu spotřebované energie, což platí i pro okrajové intensity. Nevýhodou je finančně a technicky náročné vybavení a také omezená možnost testování v terénu, přestože lze použít přenosné přístroje. Důležitá je také spolupráce pacienta, ať už používáme masku nebo náustek.

Protože spotřeba kyslíku je na rozdíl od tepové frekvence více závislá na antropometrických ukazatelích, je třeba ji vhodně normalizovat. Nejčastěji se spotřeba kyslíku uvádí vztažená na kilogram tělesné hmotnosti, přestože tento způsob normalizace neodstraní veškerý trend (částečně zůstává závislost na věku nebo povrchu těla). Přesnější by bylo vztahovat spotřebu kyslíku např. na kilogram aktivní hmoty, ale chyba měření by v tomto případě byla příliš vysoká, a proto se tento způsob příliš nepoužívá.

Nový způsob bezrozměrné normalizace navržený Schwartzem (19) výše zmíněnými nedostatky netrpí. Při tomto způsobu normalizace se využívá jak nelineární korekce na hmotnost, tak na délku kroku. Vzhledem k tomu, že tato normalizace vychází z teorie dynamické rovnováhy (5) popisující pouze chůzi, je specifická pro chůzi a nelze ji uplatnit např. na běh. Výsledkem normalizace je bezrozměrné číslo, které ovšem není příliš intuitivně uchopitelné, a navíc jsou s tímto způsobem normalizace spotřeby kyslíku u DMO jen malé zkušenosti. Autoři nicméně tento způsob normalizace úspěšně použili u DMO (20) a zdá se, že výsledky jsou přinejmenším srovnatelné se standardní normalizací vztažením na kilogram tělesné hmotnosti. Výhodou bezrozměrné normalizace je lepší možnost srovnání interindividuální variability, porovnání skupin pacientů nebo sledování vývoje parametrů v čase u jednoho pacienta.

Ačkoliv není pochyb o celkově zvýšené energetické náročnosti chůze pacientů s DMO, zátěžové vyšetření samo o sobě nám nepomůže rozeznat jednotlivé faktory, které spotřebu energie při chůzi zvyšují. Proto je důležité, aby přístup k léčbě pacienta s DMO byl multidisciplinární. Měření spotřeby kyslíku může přinést zhodnocení míry disability a stanovení funkční rezervy pacienta. Navíc je zvýšení O_{2cost} přímo úměrné míře funkčního postižení (7) a míře spontánní fyzické aktivity během dne (12).

Při zhoršení funkčního stavu pacienta dochází během chůze ke snížení rychlosti, aby se udržela spotřeba kyslíku na stejné úrovni. To se ovšem projeví zvýšením O_{2cost} a dítě s DMO se dříve unaví i při nižších rychlostech chůze (2). Díky zvýšené energetické náročnosti chůze může i chodící pacient preferovat pohyb na vozíku.

Závěry

Zátěžové vyšetření je možné i u dětských pacientů s DMO, záleží na spolupráci pacienta stejně jako je tomu u běžné populace. Zátěžové vyšetření je užitečné zejména ke sledování vývoje po korekčních operacích a mohlo by být doplňkovým vyšetřením i při jejich plánování. Některé prameny také uvádějí použití zátěžového vyšetření se stanovením PCI při rozhodování o nejvhodnějším typu pomůcky. Nejvhodnější je testovat na pohyblivém pásu („běhátku"), protože oproti chůzi po volné podložce nebo v terénu se dá dobře volit intenzita zátěže. Relativní parametry je obtížné použít, protože chybí referenční bod, jako např. maximální zátěž. V pooperačním průběhu je optimální sledovat časový vývoj energetické náročnosti, a to i v poměrně krátkodobém horizontu 12 měsíců po korekčních operacích. Z více důvodů jsou vhodnější parametry založené na měření spotřeby kyslíku než na měření tepové frekvence, nicméně v podmínkách, kde nelze použít analyzátory výměny dýchacích plynů, může být parametr PCI důležitým ukazatelem energetické náročnosti pohybu. Nová bezrozměrná metoda normalizace spotřeby kyslíku zatím u DMO čeká na ověření.

Způsob hodnocení chůze u DMO je multidisciplinární. Kromě klinického vyšetření přináší důležitou informaci také kinematická a kinetická analýza chůze, dynamická polyelektromyografie a v neposlední řadě zátěžové vyšetření se zaměřením na energetickou náročnost chůze.

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STUDY 5

Evolution of walking ability after soft tissue surgery in cerebral palsy patients. What can we expect?

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Abstract
Eleven patients with spastic cerebral palsy were evaluated preoperatively, three and nine month postoperatively after soft tissue surgery. Evaluation included clinical examination, Functional Mobility Scale (FMS) questionnaire, instrumented gait and center of mass (COM) trajectory analysis. A decrease in time-distance parameters after three months was followed by progress in all parameters at nine months postoperatively. Push-off range of ankle motion (ROM) decreased after surgery and was not restored to preoperative level until nine months. The COM vertical displacement improved significantly. FMS showed gait improvement. Despite the normalization of ROM after surgery, there is an obvious period of functional gait deterioration in the early postoperative period and push-off ROM at ankle did not recover to preoperative level until nine months.

Evolution of walking ability after soft tissue surgery in cerebral palsy patients: what can we expect?

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Eleven patients with spastic cerebral palsy were evaluated preoperatively, and 3 and 9 months postoperatively after soft tissue surgery. Evaluation included clinical examination, the Functional Mobility Scale questionnaire, and instrumented gait and center of mass trajectory analysis. A decrease in time–distance parameters after 3 months was followed by progress in all parameters at 9 months postoperatively. Push-off range of ankle motion decreased after surgery and was not restored to preoperative level until 9 months later. The center of mass vertical displacement improved significantly. The Functional Mobility Scale showed gait improvement. Despite the normalization of range of motion after surgery, there is an obvious period of functional gait deterioration in the early postoperative period and the push-off range of motion at the ankle did not recover to preoperative level until 9 months later. *J Pediatr Orthop B* **17:107–113 © 2008 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

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Keywords: cerebral palsy, gait analysis, gait, kinematics, surgery

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Introduction

The gait performance in patients with spastic cerebral palsy (CP) is impaired compared with healthy children. These children walk with reduced velocity, shorter step length and increased cadence. Over time, a reduction in gait stability was noticed [1]. To improve joint motion, soft tissue surgery has been established as an appropriate method to treat the muscle contractures [2]. A great deal of evidence exists showing that walking ability can improve after the operation, lasting a long period of time. Instrumented gait analysis has proved to be a useful and objective tool for outcome assessment when soft tissue surgery is performed [3,4]. Some reports show improvement lasting a year, 2 or even 5 years [5–7]. Not enough evidence exists, however, to show the evolution of early stages of regaining the ability to walk soon after soft tissue surgery. Every surgeon observes some degree of deterioration in CP patients for a certain period of time after corrective surgery. We should ask ourselves some questions and expect the patients to ask the same as well. How long does it take for the patients to get better? What level of deterioration is to be expected? What should we prepare our patients for?

The aim of this prospective study was to show the evolution of different gait parameters over a period of 9 months following soft tissue surgery.

Methods

Patients' selection

Eleven patients (five females, six males) were included in this prospective study. Eighteen limbs were treated in total. All of the patients had a spastic form of CP (nine diplegia, two hemiplegia) and their level of Gross Motor Function Classification system was II–III [8]. The average age of our group of patients at the time of surgery was 9.07 ± 3.7 years. All the patients were community ambulators and were designated for gait-corrective soft tissue surgery. Patients suitable for corrective bone procedures were excluded. The overall set of procedures is given in Table 1. The average number of surgical procedures was 3.2 per patient. Parents' approval for the study was given in written form and the children agreed verbally. This study was approved by the local ethical committee of the University Hospital Motol, Prague, Czech Republic.

Patients' evaluation

Patients were evaluated three times – 2 days preoperatively and approximately 3 (3.74 ± 0.53) and 9 (9.33 ± 0.64) months postoperatively. Evaluation included clinical examination, instrumented gait analysis, video and photo documentation. The Functional Mobility Scale (FMS) questionnaire was chosen to describe functional mobility in our patient group and also to detect sensitively the change after surgical intervention [9]. Physical examination included the assessment of passive range of motion of the lower limb joints and was measured goniometrically. Instrumented gait analysis was assisted by an eight-camera, video-based, motion capturing system (Proreflex 240 Hz; Qualysis, Gothenburg, Sweden), and provided three-dimensional kinematic and time–distance parameters. Changes in the joint

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Table 1 Summary of corrective soft tissue procedures and their clinical indications

Procedure	Clinical criteria for operative procedure	Number of limbs treated
Hip adductor tenotomy	Passive hip abduction of less than 30°	12
Gastrocnemius recession (Strayer)	Equinus deformity not correctable under anesthesia, positive Silverskiöld test	11
Gastrocnemius recession (Baker)	Equinus deformity not correctable under anesthesia, partially positive Silverskiöld test	5
Proximal tenotomy of rectus femoris	Positive Duncan–Elly test, anterior pelvic tilt, hyperlordosis, hip flexion contracture	4
Prolongation of Achilles' tendon	Equinus deformity not correctable under anesthesia, not influenced by position of the knee	2
Prolongation of medial knee flexors	Increased popliteal angle, fixed flexion deformity under anesthesia	1

angles over the gait cycle were calculated on a seven-segment model using Euler angles (Visual3D; C-Motion, Inc., Germantown, Maryland, USA). Marker clusters were used for model segment definition [10]. Time–distance parameters were normalized to the leg length according to Hof's method [11]. Vertical center of mass (COM) excursions were measured as a trajectory of the sacral marker [12]. Patients walked barefoot along a 10-m walkway at a self-selected speed. Relevant sagittal plane kinematic parameters, COM vertical displacement, score FMS and physical examination were considered for outcome measures.

Postoperative rehabilitation

Postoperatively, below-knee plaster casts after correction of fixed equinus deformities were applied to all patients for 6 weeks. The average duration of hospital stay was 5–7 days. After cast removal, the patients underwent physiotherapy twice a day on an inpatient basis. They were discharged as soon as they could walk with reasonable confidence. Parents were educated in the home-training program. Physiotherapy continued on an outpatient basis for the whole period of our study.

Statistical analysis

Preoperative and 3 and 9 months postoperative results were compared using repeated-measures analysis of variance. Fisher's test was used as a post hoc analysis. FMS score was evaluated using Friedman's analysis of variance. A P value < 0.05 was considered statistically significant.

Results

Time–distance parameters

We found a decrease in time–distance parameters after 3 months, but this was followed by progress on all parameters at 9 months postoperative gait analysis. Step length decreased at first follow-up ($P = 0.046$) and by the time of the second follow-up, it increased to a higher value than preoperatively. Velocity followed the same pattern and the increase from the first to the second follow-up was statistically significant ($P = 0.0013$). Cadence did not decrease significantly after operation, but increased at 9 months ($P = 0.0263$) compared with the 3-month follow-up. We did not find any differences in stride width and double support time over the whole period of our study (Table 2)

Ankle joint

We could find improvements at the ankle joint during the whole period of gait cycle at both follow-ups. At initial contact the position of the foot changed from an average 8.28° plantar flexion to a neutral position at the first follow-up and was maintained until the last gait analysis ($P = 0.0085$). The mean ankle angle in the single support phase of the gait cycle also changed from 4.04° of plantar flexion to 5.53° of dorsiflexion at 9 months after surgery ($P = 0.0412$). Push-off range of ankle motion significantly decreased after surgery ($P = 0.0075$), but was restored to the same level as preoperatively after the 9-month period. During the swing period of the gait cycle, the mean ankle angle was shifted to the neutral position at the first follow-up and very mild dorsiflexion at the second follow-up ($P = 0.0022$) (Fig. 1, Table 2).

Knee joint

We observed an improvement in knee joint movement toward extension at the stance phase with the maintenance of knee flexion in swing, resulting in increased knee range of motion. Knee angle at initial contact decreased from 32.13° to 22.96°, which was highly significant ($P < 0.0001$). During single support, the knee angle also declined by 15.75° ($P < 0.0001$) and this change persisted until the last follow-up. At the swing phase, we observed a clinically slight but statistically significant decrease in maximal knee flexion at 3 months ($P = 0.0019$), followed by an increase in preoperative value after 9 months (Fig. 2, Table 2).

Hip joint

Overall hip joint motion had a tendency toward a more extensive pattern. No change in hip joint angle at initial contact was found, but we observed a significant decrease in hip flexion during the single limb support phase of the gait cycle at both follow-ups ($P = 0.0003$). Maximal hip extension over the gait cycle reached positive values and the change was highly significant ($P = 0.0053$) compared with preoperative values. Maximal hip flexion during a swing was not changed over the period (Fig. 3, Table 2).

Center of mass vertical displacement

The center of mass vertical excursion decreased markedly during three postoperative months, but did not reach

Table 2 Summary of results

	PREOP mean	3M mean	9M mean	PREOP SD	3M SD	9M SD	PREOP versus 3M *P* level	PREOP versus 9M *P* level	3M versus 9M *P* level
TDP									
Cadence (s)	13.73	13.08	14.10	3.08	2.25	2.71	0.3221	0.1986	**0.0263**
Velocity (m/s)	0.25	0.23	0.28	0.08	0.08	0.10	0.0837	0.0967	**0.0013**
Step width (m)	0.27	0.28	0.27	0.11	0.08	0.14	0.4709	0.8240	0.3470
Step length (m)	0.53	0.51	0.59	0.13	0.12	0.13	**0.0460**	0.1014	**0.0006**
Double phase support (s)	1.47	1.61	1.38	1.77	1.05	0.56	0.3699	0.7781	0.2409
Ankle									
Initial contact (°)	−8.28	−0.97	0.79	13.19	5.61	6.30	**0.0309**	**0.0085**	0.5794
Toe-off (°)	−14.36	−0.86	−3.80	23.42	5.52	8.94	**0.0125**	**0.0446**	0.5767
Mean of single support phase (°)	−4.04	4.71	5.53	18.11	3.95	6.08	0.0617	**0.0412**	0.8476
Mean of swing phase (°)	−9.95	1.51	2.55	17.19	4.57	4.55	**0.0040**	**0.0022**	0.8169
Ankle push-off ROM	17.51	10.74	16.56	12.15	6.71	8.56	**0.0075**	0.4670	**0.0407**
Knee									
Initial contact (°)	34.13	22.26	22.96	10.67	8.94	8.62	**0.0000**	**0.0000**	0.3360
Toe-off (°)	42.05	36.11	38.49	11.65	8.43	7.06	**0.0305**	0.2720	0.2601
Mean of single support phase (°)	26.74	10.99	12.96	14.85	9.85	7.45	**0.0000**	**0.0000**	0.2766
Maximal knee flexion (°)	62.57	55.80	59.60	8.08	9.32	9.16	**0.0019**	0.1463	0.0674
Hip									
Initial contact (°)	38.55	36.74	35.98	10.95	8.36	12.24	0.2514	0.2653	0.9728
Toe-off (°)	12.42	12.29	7.56	6.56	10.20	11.62	0.8940	0.1121	0.1435
Mean of single support phase (°)	18.87	11.43	9.68	8.05	4.44	8.07	**0.0003**	**0.0000**	0.5120
Maximal extension (°)	5.01	2.26	−1.95	4.82	5.42	9.12	0.1662	**0.0053**	0.1248
COM									
COM vertical excursion	1.1181	1.0872	1.076	0.0763	0.0478	0.0607	0.0879	**0.0297**	0.5853
Gonio									
Hip flexion (°)	116.56	124.00	129.11	12.26	9.69	11.13	0.0743	**0.0004**	**0.0385**
Hip extension (°)	15.39	19.69	18.89	7.77	6.04	7.31	**0.0493**	**0.0343**	0.8677
Hip abduction (°)	32.83	40.44	33.56	10.08	9.63	6.20	**0.0097**	0.8626	**0.0063**
Hip adduction (°)	19.94	22.63	22.33	5.32	4.82	4.07	0.1711	0.3793	0.6138
Hip internal rotation (°)	50.28	53.81	48.56	12.53	13.72	11.50	0.8404	0.2323	0.1653
Hip external rotation (°)	48.78	48.13	49.89	12.35	9.16	10.25	0.3594	0.8605	0.4570
Knee flexion (°)	143.50	148.31	148.22	9.08	5.50	5.13	0.1020	**0.0365**	0.6196
Knee extension (°)	−0.56	−0.50	1.33	4.89	3.30	3.88	0.8692	**0.0083**	**0.0125**
Ankle plantarflexion (°)	60.00	52.75	50.78	11.68	6.50	6.93	**0.0010**	**0.0004**	0.7361
Ankle dorsiflexion (°)	−7.44	8.19	6.67	10.99	3.75	3.22	**0.0000**	**0.0000**	0.6143
FMS									
FMS 5	4.72	5.11	5.67	1.02	1.02	0.49	0.0858	**0.0015**	**0.0117**
FMS 50	4.06	4.22	5.28	1.21	1.73	0.96	0.6051	**0.0010**	**0.0058**
FMS 500	3.39	3.56	4.06	1.58	1.62	1.59	0.1088	**0.0281**	0.1549

Significant changes are shown in **bold.**
COM, centre of mass; FMS, Functional Mobility Scale; Gonio, goniometry; 3M, 3 months postoperatively; 9M, 9 months postoperatively; PREOP, preoperatively; SD, standard deviation; TDP, temporal-distance parameters.

statistical significance. This improvement became significant at 9-month follow-up ($P = 0.0298$) (Fig. 4).

Physical examination

A gradual increase in hip flexion ($P = 0.0004$) and extension ($P = 0.0343$) was measured in a passive range of motion after 9 months. We also observed a significant increase in hip abduction 3 months postoperatively ($P = 0.0096$), but it decreased to the preoperative level after 9 months. No changes in hip rotations were found. Passive knee flexion increased. The most dramatic changes were seen at the ankle: passive dorsiflexion increased from 7.44° of plantar flexion to 8.18° of dorsiflexion after 3 months and it remained the same throughout the whole follow-up period. On the basis of the described results, clinical tests for muscle contractures became negative (Silverskiöld, Patrick's sign, Phelp's sign).

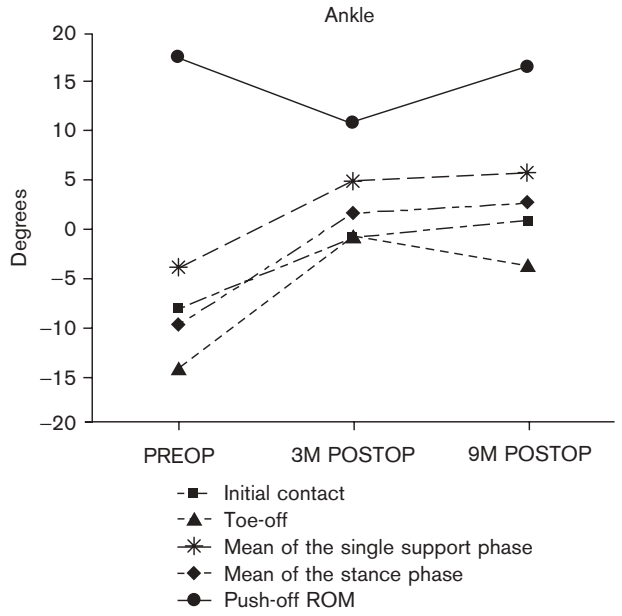
Functional mobility scale

No difference in any of the FMS distances was observed after the 3-month period, but there was a significant change after 9 months. Improvement was seen at distances of 5 m ($P = 0.0015$) and 50 m ($P = 0.001$). This result indicates better mobility indoors after 9 months postoperatively in our patients' group (Table 2).

Discussion

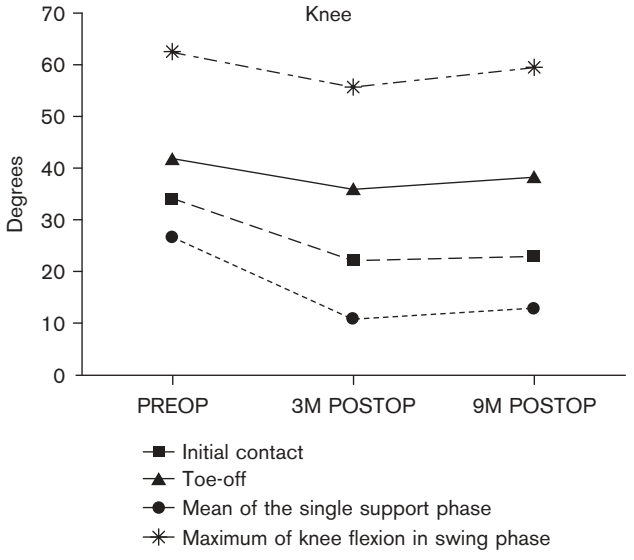
Sutherland [13] identified the major determinants of normal mature gait as velocity, step length, cadence and duration of single limb support in stance. Although the gross motor function according to motor development curves [14] should remain stable after motor development is complete, progressive deterioration in the walking ability of children with CP in childhood and adolescence has been documented [1]. Natural progression of their gait tends to deteriorate over time in time–distance parameters and kinematics, if there is no surgical intervention [15]. This might be a result of the inability of length changes in spastic muscle to keep up with bone growth [16]. Another factor that leads to gait deterioration could be weight gain [15]. While evaluating our surgical results in the CP population, we have to be

Fig. 1



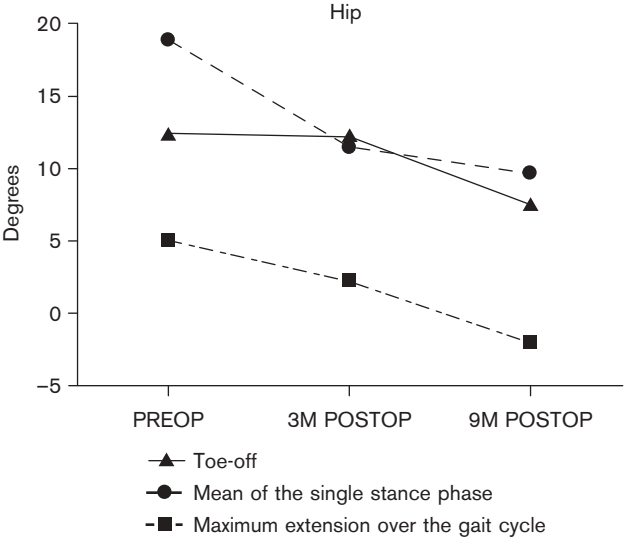
Postoperative ankle kinematic parameter development. Postoperative development shows normalization of kinematic ankle parameters. At initial contact, the equinus position of the foot changed to neutral. Mean ankle angle in single support phase of gait cycle also improved. Push-off range of ankle motion (ROM) significantly decreased after surgery, but was restored to the preoperative level at 9-month follow-up.

Fig. 2



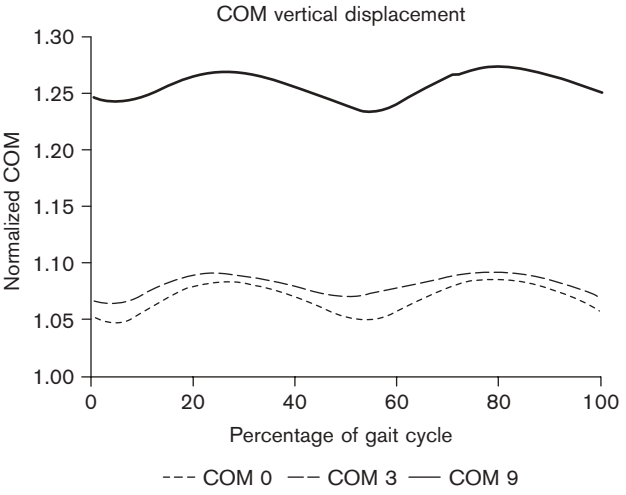
Postoperative ankle kinematic parameters development. Knee angle at initial contact and during single support significantly improved with maintenance of maximal knee flexion. These improvements result in an increase in the knee range of motion at the second follow-up.

Fig. 3



Postoperative hip kinematic parameters development. Overall hip joint motion had a tendency toward a more extensive pattern. A significant decrease is seen in the hip flexion during single limb support, and maximal hip extension improved compared with preoperative values.

Fig. 4



Postoperative development of center of mass (COM) vertical displacement. A remarkable decrease in COM vertical displacement is seen, which reached statistical significance at 9 months postoperatively and indicates better walking efficacy. COM vertical displacement is normalized to the leg length of each patient and expressed as a dimensionless number. COM 0, center of mass vertical displacement preoperatively; COM 3, center of mass vertical displacement 3 months postoperatively; COM 9, center of mass vertical displacement 9 months postoperatively.

cautious about underestimating the immediate effect of surgical intervention, which results in the stabilization of a child's gait pattern [17]. The aim of our study was to

prove the following clinical observation: although we can surgically help our patients to stabilize their walking level or even improve their gait in the long run [5,6,18], there is a period of time up to 6 months, just after the surgery [19], when the walking ability deteriorates.

For a long time, physical examination with range of motion measurement together with radiological imaging has been a single indication for corrective surgery in children with CP. Only fair-to-moderate correlation was found between static clinical measurements and gait analysis data [20]. Also, clinical tests determining isolated muscle contracture might be misleading because of spasticity, if performed in the awake state [21]. In our study, a passive range of motion increased at the hip, knee and ankle. These changes were gradual over time. On the contrary, we observed a statistically significant decrease in step length 3 months after surgery. Other time–distance parameters also deteriorated, but the changes did not reach statistical significance, although there was a clear tendency toward a postoperative decrease in velocity ($P = 0.0837$). These findings are in agreement with those by Abel *et al.* [5], but they did not provide kinematic description at 3 months postoperatively.

The indications for the selection of surgical procedures are often discussed in the literature. It is well documented that the surgical lengthening of adductor muscles may be inappropriate in an attempt to reduce excessive internal rotation of the hip [22], but contracture of adductors may be associated with hip lateralization or even subluxation or luxation of the femoral head in spastic CP. Specifically, the adductor longus has been noted to have the greatest effect in causing hip dislocation [23]. This pathological sequence may be prevented by surgical release of adductor muscles [24]. In agreement with other authors [6,25], we consider the restriction of abduction by more than 30° as a contracture of adductors and we suggest adductor muscle tenotomy as preventive hip surgery because adductor tenotomy reduces the trend toward lateral displacement of the hip joint [26]. Hip flexor contracture is believed to result in gait deviations including restricted stride length, excessive anterior pelvic tilt, increased lumbar lordosis and limited hip extension in stance [27]. Proximal rectus femoris release was initially proposed to reduce hip flexion contracture and lumbar lordosis while improving knee flexion in swing [28]. Inappropriate activity of the rectus femoris during swing is believed to contribute to stiff-knee gait in patients with CP [29]. Distal rectus femoris transfer posterior to the knee was proposed by Perry [30] to treat this gait deviation. This procedure was thought to convert the rectus femoris from knee extensor to knee flexor, but it shows that the beneficial effects of rectus femoris tendon transfer are derived from reducing the effects of the rectus femoris muscle as a knee extensor rather than from converting the muscle to knee flexor [31]. Our aim was to address anterior pelvic tilt, hyperlordosis and reduced hip extension by proximal rectus femoris release. Contrary to the results of some authors [27], we have good clinical experience with this procedure, which we have already published [32].

At the first follow-up, we found a trend toward a greater extension pattern at the hip and the knee, which is pointed out as an important mechanism in patients with CP [17]. These changes allow normal weight acceptance through the ankle and knee. An improvement in the single support stance phase and maximal hip extension took place, although maximal hip flexion during swing was not changed. At the knee there was a dramatic improvement toward extension at initial contact and during the single support stance phase, but maximal knee flexion in swing was decreased. This might be because of decreased walking velocity and power generation during push-off [33]. Foot clearance was not a problem in our group of patients. The most dramatic changes were seen at the ankle joint, where the overall joint motion was shifted toward dorsiflexion. The push-off range of motion was decreased, which goes along with slower velocity and step length and implicates a reduction in plantar flexor power. The normalization of foot position in swing, as was observed in our group of patients, is in agreement with Reimers [34], who states that antagonist function improves when spasticity of the agonist is reduced by tendon lengthening.

At the second follow-up (9 months postoperatively) an overall improvement was present. Velocity, step length and cadence increased. Increased cadence was an opposite finding to that of some authors [18,35], but in agreement with others [36], our patients used both strategies to increase speed postoperatively – increased step length and cadence. Even though the parents of our patients reported improvement in stability, we did not find any significant change in double support time or step width. This discrepancy might be explained by the small sample size of our study. At the hip the increased extension pattern during the stance phase was still present and the change in maximum hip extension reached statistical significance. Contrary to the findings of Abel [5], the maximum knee flexion in swing increased back to the preoperative level, although the improved knee extension in initial contact and stance remained. Positive results at ankle joint ROM seen after the first follow-up were still present at 9-month follow-up. Significant improvement was seen in push-off range of movement compared with the first follow-up. This parameter returned to the preoperative level and indicates better propulsion. This finding is consistent with those of Steinwender *et al.* [35] who did not find any changes in push-off range of motion after surgery, and the energy generated at push-off was significantly increased.

Children with CP typically expend two to three times more energy in submaximal walking than age-matched controls without CP [37]. They have 60% greater COM excursion and poorer phasic relation between potential and kinetic energies. Analysis of COM vertical movement seems to be a good biomechanical factor in monitoring the walking efficacy [38]. We observed a decrease in COM vertical displacement at first follow-up, but it did not reach statistical significance. A significant improvement in COM vertical excursion was seen at second follow-up ($P = 0.0298$), which indicates better walking efficacy of our patients (Fig. 4).

Measuring physical function is more difficult than measuring deformity. FMS proved to be an excellent tool in demonstrating the difference between preoperative and postoperative states and in detecting improvement and deterioration in the walking ability of children with CP [9]. The FMS was filled out by the parents of our patients. Subjective perception of parents of their children's gait improvement goes together with other kinematic data. No improvement in FMS was seen after the 3-month period, but there was a highly significant increase in parents' rating of children's walking ability after 9 months.

We can conclude that the children increased their walking ability and became better community ambulators compared with the preoperative state. We have to keep in mind that all the described improvements are not only a result of surgery, but that physiotherapy also played a great role in increasing their walking ability.

Conclusion

We found soft tissue surgery to be an effective method for the correction of contracted muscle deformities. Although a 6-month period is reported to be sufficient time for recovery after soft tissue surgery [5], some of the important gait parameters (push-off range of motion at ankle joint) did not recover to their preoperative levels until 9 months postoperatively. Despite the normalization of range of motion immediately after operation, there is an obvious period of deterioration after a corrective surgery in patients with CP and we have to inform our patients about this fact in advance. Our results also highlighted the importance of postoperative physiotherapy and the necessity of muscle and gait training programs after soft tissue gait-improvement surgery.

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STUDY 5

Počítačová 3D analýza chůze hodnotí objektivně pooperační vývoj korekce equinozity u pacientů s dětskou mozkovou obrnou

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Abstract

Equinus deformity is a common problem in children with cerebral palsy (CP) and significantly influences their gait. Corrective surgery at the level of gastrocnemius aponeurosis is one of the treatment options. Computerized 3D gait analysis was used to objectify the results of the surgery and to explore the development of the ankle motion over the gait cycle. Postoperatively, the ankle and also the knee motion were normalized. Because of the postoperative triceps surae muscle weakness the push-off phase of the gait cycle was deteriorated and did not improve until 9 months after the surgery. Prolongation of the aponeurotic part of the gastrocnemius muscle is an effective and safe way of the equinus correction in children with CP. Computerized gait analysis is a good tool to objectively assess postoperative results of CP patients and should become part of the common care in CP patients.

Počítačová 3D analýza chůze hodnotí objektivně pooperační vývoj korekce equinozity u pacientů s dětskou mozkovou obrnou

Computerized 3D analysis of gait objectively assesses the postoperative development of equinus deformity correction in children with cerebral palsy

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Souhrn:

Pes equinus neurogenes je u pacientů s dětskou mozkovou obrnou (DMO) častou deformitou, která výrazně alteruje chůzový stereotyp. Operační korekce deformity zásahem v aponeurotické části musculus gastrocnemius je jedním z možných řešení. Využili jsme metody počítačové 3D analýzy chůze k objektivizaci výsledků operativy a stanovení vývoje dynamiky pohybu hlezna během chůzového cyklu. Pooperačně se upravuje nejen postavení a pohyb hlezna během celého chůzového cyklu, ale zároveň se normalizuje i pohyb kolena. Kvůli oslabení musculus triceps surae dochází pooperačně ke zhoršení odrazové fáze hlezna, která se upravuje až 9 měsíců po operaci. Prolongace aponeurotické části musculus gastrocnemius je efektivní a bezpečnou variantou korekce equinózního postavení nohy u pacientů s DMO. Počítačová analýza chůze je ideálním prostředkem k objektivizaci operačních výsledků pacientů s DMO a v budoucnu by se měla stát součástí péče o pacienty s DMO.

Klíčová slova: dětská mozková obrna, pes equinus neurogenes, analýza chůze, musculus gastrocnemius

Summary:

Equinus deformity is a common problem in children with cerebral palsy (CP) and significantly influences their gait. Corrective surgery at the level of gastrocnemius aponeurosis is one of the treatment options. Computerized 3D gait analysis was used to objectify the results of the surgery and to explore the development of the ankle motion over the gait cycle. Postoperatively, the ankle and also the knee motion were normalized. Because of the postoperative triceps surae muscle weakness the push-off phase of the gait cycle was deteriorated and did not improve until 9 months after the surgery. Prolongation of the aponeurotic part of the gastrocnemius muscle is an effective and safe way of the equinus correction in children with CP. Computerized gait analysis is a good tool to objectively assess postoperative results of CP patients and should become part of the common care in CP patients.

Key words: cerebral palsy, equinus deformity, gait analysis, gastrocnemius muscle

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Úvod

Equinózní deformita je jedním z nejčastějších problémů vyskytujících se u spastické formy dětské mozkové obrny (DMO). Chůze pacientů s touto deformitou je charakterizována sníženou stabilitou během stojné fáze, kratším krokem a obtížemi během švihové fáze chůzového cyklu (26). Tyto změny vedou k větším vertikálním výchylkám těžiště, a tedy i vyšší energetické náročnosti chůze.

Cílem naší prospektivní studie je zhodnotit vliv korekční operace pes equinus nerogenes na postoperační vývoj dynamických parametrů hlezenního kloubu při chůzi. Naše klinická zkušenost nás vede k hypotéze, že postoperační období po sejmutí sádrové fixace je provázeno zhoršením schopnosti chůze na úroveň nižší než před operací a pozitivní výsledky korekce deformity můžeme očekávat až v horizontu několika měsíců. Pro potvrzení naší hypotézy jsme použili kromě klasických metod (klinické vyšetření, goniometrie) také technicky sofistikovanou metodu analýzy chůze pomocí její počítačové simulace (11). Pokud je nám známo, jedná se o první použití této metody k evaluaci výsledků operační léčby DMO v českém písemnictví.

Soubor pacientů a metodika

Do prospektivní studie bylo zařazeno 8 pacientů (12 operovaných končetin) se spastickou diparetickou formou dětské mozkové obrny s průměrným věkem 7,32 ± 2,72. Jejich mobilita byla ohodnocena podle Gross Motor Function Classification System (17) na stupni I–III. Pacienti byli indikováni k operačnímu zásahu určenému ke korekci equinózního postavení hlezna závislém na postavení v kolenním kloubu. Operace byly provedeny buď metodikou dle Strayera (27), nebo Bakera (3). Rodiče podepsali písemný souhlas se zařazením dětí do studie a ústní souhlas ke spolupráci byl dán i samotnými

pacienty. Veškeré operace byly provedeny standardním způsobem dle indikace operatéra. Postoperační péče a rehabilitace byla srovnatelná u všech pacientů. Všem byla pooperačně naložena sádrová fixace na dobu 6 týdnů. Po sejmutí fixace pacienti rehabilitovali za hospitalizace do plné vertikalizace. Následná rehabilitace probíhala na denní bázi.

Pacienti byli vyšetřeni předoperačně a poté 3 a 9 měsíců po operaci. Při každé kontrole bylo provedeno klinické vyšetření (goniometrické vyšetření kloubů dolních končetin, antropometrické vyšetření, zjištění kontraktur a spasticity svalů dolních končetin, fotografická a videodokumentace) a počítačová trojdimenzionální analýza chůze. K vyšetření bylo použito 8 speciálních videokamer emitujících infračervené světlo (ProReflex, Qualisys, Švédsko). Pro vytvoření trojrozměrného počítačového modelu dolních končetin byly použity reflexní markery (7) (Obr. 1). Kinematická data byla vypočtena pomocí metodiky Eulerových úhlů (8) za použití softwaru Visual3D (C-motion, USA). Vertikální výchylky těžiště (COM=Center of Mass) byly stanoveny pomocí sledování trajektorie virtuálního markeru umístěného v oblasti druhého bederního obratle (23). Pacienti chodili spontánně zvolenou rychlostí po dráze 10 m. Byli vyšetřeni bez obuvi a ortéz. Pakliže běžně používali berle nebo jiné pomůcky, vyšetření bylo provedeno

Obr. 1 Pacient s aplikovanými reflexními markery během vyšetření kinematické analýzy chůze v biomechanické laboratoři CASRI, Praha. V pozadí je vidět infračervené kamery ProReflex240 (Qualisys, Švédsko)

s nimi. K analýze byl použit průměr minimálně 3 chůzových cyklů.

Výsledná data přístrojového vyšetření chůze byla normalizována pomocí délky dolní končetiny na bezrozměrné veličiny. Metoda navržena Hofem (14) pomáhá minimalizovat interindividuální antropometrické odlišnosti a umožňuje srovnání dětí různých věkových skupin a vzrůstu. Ke statistickému zhodnocení byla použita ANOVA s Fisherovým testem nejmenšího rozdílu jako post-hoc testem. Za signifikantní jsou považovány výsledky na hladině významnosti $p < 0,05$.

Výsledky

Klinické vyšetření

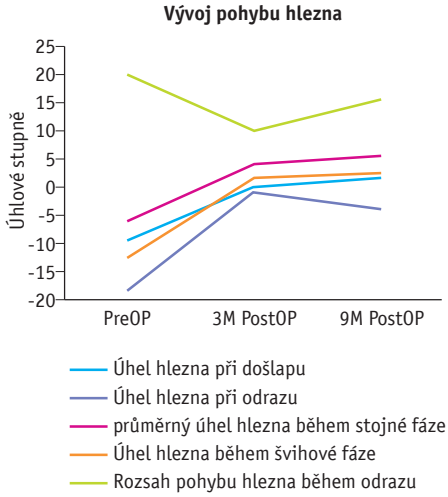
Pooperačně dochází k zvýšení goniometricky měřeného rozsahu pohybu hlezna z 53,5 ° na 61 ° tři měsíce pooperačně a toto zlepšení přetrvává i při poslední kontrole. Zároveň došlo k významnému zvýšení dorziflexe hlezna z předoperační hodnoty -7,66 ° na 8,25 ° ($p < 0,0001$), což vedlo k pooperační negativitě Silverskiöldova testu ($p = 0,0077$). Rozsah pohybu, maximální hodnoty extenze a flexe kolenního kloubu ani hodnota Bleckova popliteálního úhlu se pooperačně významně nezměnily.

Základní parametry chůze

Pooperační vývoj byl obdobný u všech základních parametrů chůze. Při kontrole po 3 měsících od operace dochází k patrnému zhoršení chůze, které je následováno zlepšením při závěrečné kontrole, a to i vůči předoperačnímu stavu. Výsledkem operace je zvýšená spontánní rychlost chůze, kterou dosahují díky prodloužení délky kroku i zvýšené kadenci. Tyto změny jasně dokumentují kladný výsledek operace, neboť přirozený vývoj těchto parametrů chůze u DMO má obrácený charakter (16).

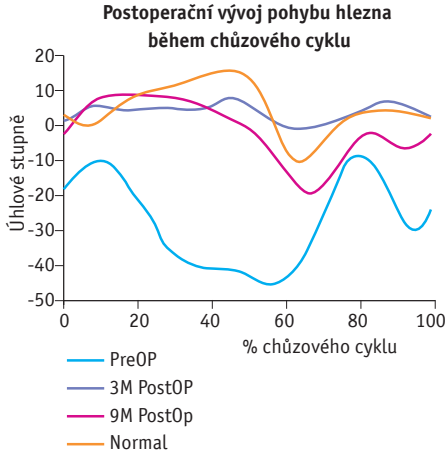
Kinematická data

Postoperační vývoj pohybu hlezenního kloubu ukazuje na výrazné zlepšení směrem k normálním hodnotám ve všech fázích chůzového cyklu. Předoperačně byl průměrný úhel hlezenního kloubu při došlapu 9,56 ° plantiflexe, tzn. v equinózním postavení nohy. Během stojné fáze zůstávala noha nadále v plantiflexi v průměru 6,18 °. Na křivce (Graf 1) můžeme vidět špatně diferencovanou fázi odrazu. Během švihové fáze zůstává hlezno v plantigrádním postavení 12,27 °, které funkčně prodlužuje dolní končetinu a je příčinou zvýšené flexe kolenního kloubu během švihové fáze a špatného přednastavení dolní končetiny pro fázi opory.



Graf 1 Porovnání pooperačního vývoje pohybu hlezna – porovnání křivek pohybu hlezna jednoho z našich pacientů během krokového cyklu předoperačně, 3 měsíce a 9 měsíců po operaci s křivkou zdravého dítěte. Je patrný postoperační posun k normálnímu rozsahu pohybu hlezna. (PreOP = předoperačně, PostOP 3M = kontrola po 3 měsících od operace, PostOP 9M = kontrola po 9 měsících od operace, Normal = zdravé dítě)

Při kontrole po 3 měsících dochází k signifikantnímu zlepšení postavení nohy a hlezna (Graf 2). Průměrný úhel hlezna při došlapu byl -0,22 ° ($p = 0,0287$). Během stojné fáze se normalizovala poloha hlezna do dorziflexe 4,11 °, ale změna ještě nedosáhla statisticky signifikantní hladiny ($p = 0,0613$). Operačním zákrokem byla alterována zejména fáze odrazu, kdy se hlezno dostává jen do velmi mírné plantiflexe 0,77 ° ($p = 0,0057$) a nemůže tak vyvinout dostatečnou

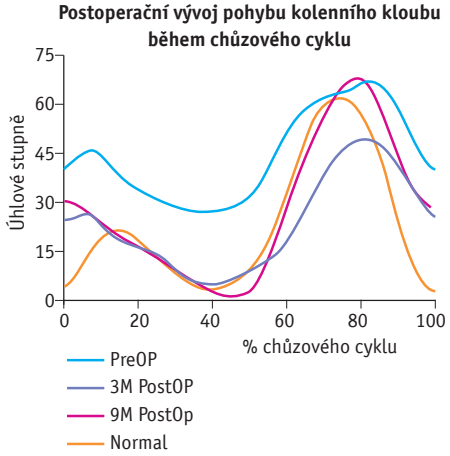


Graf 2 Pooperační vývoj pohybu hlezna – grafické porovnání parametrů pohybu hlezenného kloubu v průběhu prospektivní studie. (PreOP = předoperačně, PostOP 3M = kontrola po 3 měsících od operace, PostOP 9M = kontrola po 9 měsících od operace)

propulzní sílu (21). Tento nález svědčí pro operační oslabení plantiflexorů hlezna a je ve shodě se sníženou rychlostí a kratším krokem v období 3 měsíce po operaci. Během švihové fáze chůzového cyklu je již hlezno zcela normálně nastaveno do neutrální polohy 1,93° dorziflexe (p = 0,0041), která napomáhá lepšímu přednastavení nohy a kolena při došlapu.

Při poslední kontrole po 9 měsících se ještě zlepšilo postavení v hleznu při došlapu 1,74 ° (p = 0,0098). Během stojné fáze se zlepšila dorziflexe na průměrnou hodnotu 5,57 ° a dosáhla tak statisticky signifikantního rozdílu vůči předoperačnímu stavu (p = 0,0321). Během švihové fáze chůzového cyklu si udrželi normální postavení hlezna v mírné dorziflexi 2,79 °. Důležitým parametrem chůze je odrazová fáze hlezna, kdy síla plantiflexorů nohy dává tělu asi 50% propulzní energie (12). Při operačním zásahu na musculus triceps surae, zejména při prolongaci Achillovy šlachy, dochází k jeho oslabení (9). Pooperačně po třech měsících jsme pozorovali zmenšený rozsah pohybu hlezna při odrazu (p = 0,0025). Tento pokles byl následován normalizací na předoperační úroveň po 9 měsících od operace (Graf 2). Celkově se pohyb v hleznu pooperačně normalizoval.

Musculus gastrocnemius je dvoukloubový sval a ovlivňuje tedy nejen hlezno, ale i pohyb v koleni. U pacientů s equinózní chůzí byla prokázána spojitost mezi plantární flexí hlezna a extenzí kolena (2). Popsaný souhyb je prodloužením aponeurotické části musculus gastrocnemius eliminován. Podobný postoperační vývoj pozorujeme i v souboru našich pacientů (Graf 3). Bez operačního zásahu v oblasti kolenního kloubu se významně zlepšila extenze kolenního kloubu jak při došlapu (p= 0,0002), tak během stojné fáze chůzového cyklu (p= 0,0002), přičemž hlezenní kloub jde během stojné fáze do extenze. Flexe kolena během švihové fáze nebyla nijak alterována. Rozpojení patologického souhybu hlezna a kolena přispívá ke snížení energetické náročnosti chůze. Pacienti s DMO spotřebovávají 2–3krát více energie na chůzi než jejich zdraví vrstevníci (20). Mechanická efektivita jejich pohybu je nižší než u normální populace. Mají až o 60 % vyšší vertikální výchylky trajektorie těžiště, a navíc se jejich kinetické a potencionální energie nepohybují po sinusoidě a nejsou vůči sobě ve fázi (5). Proto je trajektorie těžiště dobrým parametrem pro sledování biomechanických i funkčních změn po operaci a normalizace energetického výdeje je jedním z hlavních cílů léčby. Ve srovnání s předoperačním stavem dochází u našeho souboru pacientů pooperačně k symetrizaci vertikální trajektorie těžiště a zároveň ke zjevné tendenci ke zmenšení jeho vertikálních výchylek, která ovšem nedosáhla statistické signifikance (p = 0,0758).



Graf 3 Srovnání pooperačního vývoje kinematických parametrů kolene – porovnání křivek pohybu kolenního kloubu jednoho z našich pacientů během krokového cyklu předoperačně, 3 měsíce a 9 měsíců po operaci s křivkou zdravého dítěte. Pozorujeme pooperační vývoj směrem k extenzi ve stojné fázi chůzového cyklu bez ztráty maximální flexe během fáze švihové. (PreOP = předoperačně, 3M PostOP = kontrola po 3 měsících od operace, PostOP 9M = kontrola po 9 měsících od operace, Normal = zdravé dítě)

Diskuze

Sutherland (28) určil jako hlavní determinanty zralé chůze zdravého člověka její rychlost, délku kroku, kadenci a trvání fáze jedné opory během krokového cyklu. Ačkoliv dle vývojových křivek by úroveň hrubé motoriky měla zůstat stabilní v čase (22), jakmile pacient dosáhne vývojového maxima, existují práce, které prokazují postupné zhoršení schopnosti chůze pacientů s DMO během dětství a adolescence (16). Jejich vývoj směřuje ke zhoršení všech hlavních determinant chůze, snížení rozsahu pohybu kloubů dolních končetin a progresivnímu zhoršení kinematiky i kinetiky chůze, pokud není operačně zasáhnuto (4). Tento vývoj je zřejmě důsledkem neschopnosti spastických svalů růst stejnou rychlostí jako kost, což následně vede ke vzniku kontraktur (30). Proto nesmíme podcenit fakt, že i stabilizace kvality chůze je pozitivním výsledkem operační léčby (13).

Tato práce se jako první v české odborné literatuře zabývá využitím kinematické analýzy chůze v klinické praxi, ačkoliv v zahraničí se jedná o metodu používanou jak ve výzkumu, tak i klinicky. Pomocí přesných dat o trojrozměrném pohybu pacienta je sestaven počítačový model, na kterém se dají dále provádět například simulace délek svalů během chůzového cyklu (29) i predikce operačních výkonů (9). Slouží nejen jako objektivizační metoda operačních výsledků (21, 24, 26), ale pomáhá při plánování a indikaci operací u pacientů s DMO. Správná indikace operačního výkonu zůstává klíčovým okamžikem léčby. Bylo prokázáno, že statická klinická vyšetření mají jen střední korelaci s výsledky analýzy chůze (10). Díky spasticitě mohou být testy kontraktur jednotlivých svalů zavádějící, a někteří autoři proto doporučují provádět např. Silverskiöldův test v celkové anestezii, aby bylo možno dobře odlišit fixní a dynamickou kontrakturu (18, 26). Dynamická kontraktura může být dobře léčena pomocí aplikace Botulinum toxinu A (15), rehabilitace a redresního sádrování. Nesprávná indikace k prolongaci Achillovy šlachy při izolované kontraktuře musculus gastrocnemius přitom vede k významnému oslabení plantiflexorů hlezna až na 36 % jejich původní síly (9), což může mít za následek skrčeneckou chůzi nebo vznik hákovité nohy (25). Rizikovými faktory u diplegické formy DMO jsou zejména tíže postižení, věk pod 8 let a korekce provedená perkutální prolongací Achillovy šlachy (6). Bylo prokázáno, že prolongace aponeurální části musculus gastrocnemius nevede k oslabení propulzní síly musculus triceps surae, ale dokonce ke schopnosti generovat vyšší sílu během odrazové fáze chůzového cyklu (21). Prolongace svalu vede jednak ke snížení napínacího reflexu spastického musculus triceps surae během fáze zatížení a dále k výhodnější biomechanice svalu, který může pracovat ve střední části rozsahu své délky (21). Prolongace aponeurální části musculus gastrocnemius se proto zdá být bezpečnou a efektivní metodou operační korekce equinózní deformity pacientů s DMO, pokud jsou dodržena správná indikační kritéria.

Cílem naší studie bylo ukázat vývoj dynamické funkce hlezna po operační léčbě equinozity zásahem na musculus triceps surae. Potvrdili jsme naše klinické pozorování, že pozitivní efekt operace na chůzi pacienta nenastává ihned. Zpomalení chůze, zkrácení kroku, snížená kadence i odrazové fáze hlezna jsou v časné pooperační fázi alterovány.

	Průměrná hodnota			Směrodatná odchylka			Hladina významnosti p		
	PREOP	3M	9M	PREOP	3M	9M	PREOP vs. 3M	PREOP vs. 9M	3M vs. 9M
Základní parametry chůze									
Rychlost chůze	0,25	0,21	0,30	0,102	0,081	0,110	0,1233	0,2946	0,0142
Kroková frekvence	13,25	12,45	14,29	3,623	2,413	3,051	0,3609	0,2209	0,0398
Šířka kroku	0,30	0,30	0,31	0,125	0,085	0,156	0,7831	0,6112	0,8148
Délka kroku	0,54	0,49	0,61	0,164	0,131	0,144	0,0999	0,2572	0,0090
Délka fáze dvojí opory	1,78	1,89	1,44	2,156	1,182	0,641	0,7331	0,5176	0,3271
Vertikální výchylka těžiště	1,13	1,10	1,08	0,050	0,055	0,074	0,1128	0,0758	0,8205
Kinematické parametry									
Úhel hlezna při došlapu (°)	-9,56	-0,22	1,74	12,768	6,001	6,447	0,0287	0,0098	0,6232
Úhel hlezna při odrazu (°)	-18,27	-0,77	-3,41	24,019	6,386	9,105	0,0057	0,0153	0,6594
Maximální dorsiflexe hlezna (°)	1,67	9,69	12,15	14,782	5,598	4,319	0,0744	0,0220	0,5539
Maximální plantiflexe hlezna (°)	-22,05	-7,04	-7,90	22,455	4,389	7,244	0,0178	0,0190	0,9758
Průměrný úhel hlezna během stojné fáze (°)	-6,18	4,11	5,57	17,007	4,344	6,651	0,0613	0,0321	0,7518
Úhel hlezna během švihové fáze (°)	-12,27	1,93	2,79	17,575	4,941	5,224	0,0041	0,0030	0,8958
Rozsah pohybu hlezna během odrazu (°)	19,94	10,46	15,56	13,421	7,658	8,990	0,0025	0,0738	0,1316
Úhel kolene při došlapu (°)	33,24	21,62	23,12	12,648	10,028	9,760	0,0000	0,0002	0,1639
Průměrný úhel kolene během stojné fáze (°)	25,05	7,78	10,52	15,813	8,929	7,313	0,0000	0,0002	0,2647
Klinické vyšetření									
Plantární flexe hlezna (°)	61,17	52,67	53,17	10,701	6,286	6,464	0,0100	0,0145	0,8698
Dorsální flexe hlezna (°)	-7,67	8,25	7,67	8,435	4,434	2,807	0,0000	0,0000	0,8089
Flexe kolenního kloubu (°)	146,50	149,42	149,67	4,908	6,244	4,812	0,0832	0,0615	0,8778
Extenze kolenního kloubu (°)	1,17	0,67	2,50	3,010	2,309	4,011	0,4968	0,0789	0,0189
Blecků popliteální úhel (°)	47,50	45,00	43,00	25,840	14,334	10,287	0,7087	0,5029	0,7649
Siverskiöldův test							0,0077	0,0077	

Tabulka 1 Souhrnné výsledky – data jsou vyjádřena jako bezrozměrné veličiny pokud není uvedeno jinak. Tučným písmem jsou zvýrazněny statisticky signifikantní výsledky. (PREOP = předoperačně, 3M = kontrola po 3 měsících, 9M = kontrola po 9 měsících).

Stejné změny pozoroval ve své práci Abel (1). Postoperačního zhoršení chůze si musíme být vědomi a připravit na něj také naše pacienty. Příčinou může být dlouhá doba sádrové fixace, která vede k oslabení svalů dolních končetin. Dalším důvodem by mohla být změněná propriocepce a biomechanická situace, se kterou se pacient musí naučit pracovat. Ve shodě s pracemi ostatních autorů můžeme konstatovat, že pozitivní vliv operace se dostavuje přibližně za 6 měsíců (1, 19). V našem kolektivu se některé parametry chůze (odrazová fáze hlezna) dostaly zpět na předoperační úroveň dokonce až po 9 měsících po operaci. Naproti tomu úprava equinózního postavení nohy i normalizace rozsahu pohybu hlezna i kolena jsou zjevné již při první postoperační kontrole. Proto je nutné zdůraznit význam dlouhodobé rehabilitace v pooperační péči o pacienta s DMO.

Závěr

Prolongace aponeurotické části musculus gastrocnemius je efektivní a bezpečnou variantou korekce equinózního postavení nohy u pacientů s DMO. Je nutné však dodržet indikační kritéria izolované fixní kontraktury musculus gastrocnemius a polohové závislosti nohy na postavení kolenního kloubu. Jak ukazují naše výsledky, můžeme v pooperační fázi očekávat deterioraci chůze, která je ovšem následována rapidním zlepšením na úroveň vyšší než před operací. Po 9 měsících od operace je u všech pacientů patrný pozitivní vliv operačního výkonu na chůzi a úprava patologického pohybu hlezna i kolena. Kinematická analýza je ideálním prostředkem k objektivizaci operačních výsledků pacientů s DMO. Pomáhá nám lépe pochopit patokineziologii pohybu našich pacientů a lépe tak indikovat operační zákroky. Ve spojení s biomechanikou a počítačovou simulací nám může dát možnost vizualizace parametrů in vivo neměřitelných (např. dynamická délka svalů během chůze). Přístrojová analýza chůze by se do budoucna mohla stát přínosnou součástí indikačního schématu a pooperačního sledování pacientů s DMO.

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