Gait deviations in children with cerebral palsy: 
a modeling approach

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Gait deviations in children with cerebral palsy:  
a modeling approach

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1.1. Introduction

Humans can walk with an agility and versatility that may seem self-evident at first sight. Yet, when parts of the neural or musculoskeletal system are affected due to injury or chronic disorders, walking can become most difficult, or even impossible. The complexity of human bipedal gait also becomes apparent when trying to synthesize gait in computer simulations or physical robot models.

The ability to walk is essential for many daily-life activities. Many children with cerebral palsy (CP) experience problems with walking, due to disorders affecting the neuromuscular control and musculoskeletal structure of the lower extremities. This can restrict their functional performance and affect their capacity to take part in daily-life activities and social situations.

In order to help patients and improve their ability to walk, a good understanding of gait in general and pathological gait in particular is essential. This thesis aims to study some aspects of the underlying causes of gait deviations in CP.

This general introduction will first introduce the background of CP, the many impairments that can occur in CP, and the gait deviations that are most commonly seen. A short overview will be given on determining the underlying causes of gait deviations, and the problems that arise with this. Next, the aim, approach, and outline of this thesis will be described.

1.2. Cerebral palsy

Definition, prevalence, and causes

Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy, and by secondary musculoskeletal problems (Rosenbaum et al., 2007).

There is no explicit upper age limit for the onset of the disorder specified in this definition, although the first two or three years of life are most important in the timing of disturbances resulting in CP. In general, disturbance resulting in CP is presumed to occur before the affected functions (e.g. walking, manipulation) have developed (Rosenbaum et al., 2007). In the Netherlands, a specific age limit for the onset of CP of one year of age is adopted (Becher, 2002).

CP is the most frequent cause of motor disability amongst children in Europe (Himmelmann et al., 2005). The prevalence of CP in Europe has been rather stable over the last 30 years, and ranges between 1.5 and 3.0 per 1000 live births (McManus et al., 2006). In a large long-term
Swedish study on prevalence and etiology of CP a prevalence of 1.92 was reported for the most recent birth-year period (1995-1998) (Himmelmann et al., 2005). In the Netherlands, the prevalence has been reported to lay around 2 per 1000 live births (Wichers et al., 2005). The origin of CP can be prenatally (~28%), peri-/neonatally (~39%), post-neonatally (~5%), or unknown (~28%) (Himmelmann et al., 2005). The risk of CP increases with premature birth. The prevalence of CP amongst children born before 28 weeks of gestation is 77 per 1000 (Himmelmann et al., 2005). Several other factors are associated with the development of CP, including multiple birth, chorioamnionitis, maternal and fetal infection, or fetal anoxic events (Koman et al., 2004).

Classifications
There are three main groups of motor disorders in CP (Cans et al., 2004; McManus et al., 2006). All include abnormal patterns of posture and/or movement.

- Spastic CP is characterized by increased tone (not necessarily constantly) and/or pathological reflexes (hyper-reflexia or pyramidal signs).
- Ataxic CP is characterized by loss of orderly muscular coordination, so that movements are performed with abnormal force, rhythm and accuracy.
- Dyskinetic CP is characterized by involuntary, uncontrolled, recurring, occasionally stereotyped movements of affected body parts. Dyskinetic CP can be further classified into dystonic CP, dominated by both hypokinesia and hypertonia, and choreo-athetotic CP, dominated by both hyperkinesia and hypotonia.

Spastic CP accounts by far for the largest group, occurring in 85% (including mixed types) of the European birth cohort. Of this cohort, 6.6% had dyskinetic and 4.1% ataxic CP (McManus et al., 2006).

The localization of the CP can be unilateral or bilateral. Unilateral CP is also referred to as hemiplegia, in which the limbs on one side of the body are involved. Bilateral CP can be either diplegia, in which the legs are more involved than the arms; or quadriplegia, in which all four limbs are involved.

Children with CP can also be classified according to their level of gross motor functioning, by means of the Gross Motor Function Classification System (GMFCS) (Palisano et al., 1997). The GMFCS classifies children into five groups, depending on their functional limitations, the need for hand-held mobility devices (such as walkers, crutches, or canes), or wheeled mobility. For the age-range of 6 to 12 years, children in level I can walk without assisting devices and perform gross motor functions such as running and jumping, but speed, balance, and coordination can be limited. Children in level II can also walk without devices but with limitations, while children in level III can walk only with assisting devices. Children in level IV have independent mobility but with limitations, requiring physical assistance or powered mobility in most settings. Children in level V have no independent mobility, or may at best achieve self-mobility using a powered wheelchair with extensive adaptations.
The present thesis will focus on children with spastic CP, in the age range of 6-12 years, with hemiplegia or diplegia, and GMFCS level I or II.

**Functioning**

The current theoretical framework used in rehabilitation medicine for measuring health and disability is the International Classification of Functioning, Disability, and Health (ICF) (WHO, 2001). Recently, the ICF for children and youth (ICF-CY) has been developed, to describe the specific manifestations of functioning, disability, and health in children and adolescents (Figure 1.1) (WHO, 2007). Within this framework, functioning is categorized into three domains: body functions and structures, activities, and participation. The ICF places these domains in the context of environmental and personal factors, since these interact with the health condition.

![Diagram of ICF-CY framework]

**Figure 1.1:** The International Classification of Functioning, Disability, and Health for Children and Youth (ICF-CY) (WHO, 2007). The gray box indicates the focus of this thesis, and is further specified in Figure 1.2.

Body functions are the physiological and psychological functions of the body, and body structures are the anatomical parts. Abnormalities in body functions (e.g. muscle tone or power) or structures (e.g. bone or joint malformations) are referred to as impairments. Activities describe the execution of tasks or actions by an individual person, such as walking. Problems with performing activities are called limitations. Participation describes the involvement in life situations, i.e. taking part in society. Problems experienced in this domain are referred to as restrictions (Rauch et al., 2008).

The aim of pediatric rehabilitation medicine is to restore (potentially) disturbed interaction with the environment and to reach optimal autonomy and social participation (Meihuizen-de Regt et al., 2003). Although the ultimate aim is thus to improve functioning at the level of
participation, rehabilitation medicine uses a holistic approach, in which functioning is assessed at all domains of the ICF, including environmental and personal factors (Rauch et al., 2008). Furthermore, rehabilitation uses a patient-oriented approach, in which care is guided by patient-relevant problems. These concern mostly limitations in daily-life activities and restriction in participation. Impairments in body structure or function often underlie these limitations in activity. In order to address these problems, intervention can then be targeted at one or more of the domains of the ICF, depending on the assessment outcomes, individual goals and modifiability of the ICF category (Rauch et al., 2008).

Gait problems in CP are primarily caused by impairments in body structures and functions (Gage, 2004). This thesis will therefore focus mainly on the domain of body functions and structures and on impairments in CP. Specifically, impairments in the sub-domains of neuromusculoskeletal and movement-related structures and functions are expected to play a role and will be discussed in this thesis. Reference is also made to the domain of activity, i.e. limitations of walking in CP in terms of speed, energy cost, or stability.

In the following, first the main movement-related impairments as observed in CP will be discussed, followed by a description of common gait deviations in CP. Third, a short overview will be given of how impairments and gait deviations are related.

### 1.3. Impairments in CP

#### Primary versus secondary impairments

Impairments of the neuromusculoskeletal system in CP occur as a direct or indirect result of the upper motor neuron syndrome (Gage, 2004). Direct, or primary, impairments are of neurological nature. Injury to upper motor neurons decreases cortical input to the reticulospinal and corticospinal tracts, which produces abnormal muscle control and decreases the number of effective motor units, leading to weakness. The upper motor neuron injury also decreases the descending inhibitory input through the reticulospinal tract, which increases the excitability of alpha and gamma neurons, producing excessive muscle activity, such as spasticity and hypertonia (Koman et al., 2004). These primary impairments due to the upper motor neuron syndrome can lead, in the longer term, to adaptations in the musculoskeletal system, which are known as secondary impairments. Secondary impairments lay mostly at the orthopedic level.

Impairments in muscle activity are also commonly subdivided into excess (or positive) and deficit (or negative) symptoms. Excess symptoms are characterized by increased levels of involuntary muscle activity, whereas deficit symptoms indicate decreased voluntary muscle activity compared to normal (Pandyan et al., 2005).

Figure 1.2A-B gives an overview of the main primary and secondary neuromusculoskeletal impairments in CP, which are briefly described below.
Spasticity

Spasticity is one of the most frequently observed impairments in children with spastic CP. There is no consensus in the literature on the definition of spasticity. The most common definition in the literature is that of Lance (1980): ‘Spasticity is a motor disorder characterized by a velocity dependent increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome’. This definition thus restricts spasticity to those elements of the excess symptoms that are velocity-dependent and result from hyper-excitability of the (phasic) stretch reflex. Following this definition, spasticity has also been more practically expressed as a ‘velocity-dependent increased resistance to passive movement’ (Albright, 1996).

A more recent definition as proposed by the Support Programme for the Assembly of database for Spasticity Measurement (SPASM) consortium reads as follows (Pandyan et al., 2005): ‘Spasticity is disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles’. This definition thus includes all primary excess symptoms of the upper motor neuron system.

The difference between the two definitions is thus whether the term spasticity is reserved for one specific aspect of the excess symptoms or as a broader definition including several different symptoms. In this thesis, the first definition as introduced by Lance is adopted, since it is the most widespread and most specific. As such, this definition allows studying a single aspect of the upper motor neuron lesion, rather than a complex of symptoms.

Spasticity can be assessed by passively stretching the muscle and measuring the resulting resistance. This can be done with clinical tests such as the Spasticity Test (SPAT) (Scholtes et al., 2007a), the (Modified) Ashworth Score (Bohannon and Smith, 1987), or the (Modified) Tardieu score (Boyd and Graham, 1999). Spasticity can also be tested quantitatively using instrumented tests, such as the Instrumented SPAT (Van den Noort et al., 2008). In this thesis, the clinical SPAT will be used for the assessment of spasticity.

Other primary impairments

Other primary excess tone disorders include hypertonia and enhanced reflexes other than the stretch reflex. Hypertonia is defined as not-velocity-dependent increased resistance to passive movement, caused by an exaggerated response of the tonic stretch reflex, leading to tonic (continuous) activity (Becher et al., 2006). In the lower extremities, especially the extensor muscles show hypertonia.

Enhanced reflexes may include enhanced postural reflexes (Becher et al., 2006) and released flexor reflexes in the lower limbs (Mayer, 1997).

Selectivity (or selective motor control) is the ability to move an individual joint independently from the other joints in the same limb and to use only the correct muscle groups during movement (Desloovere et al., 2006). Selective motor control is often poor in CP, but it has hardly been studied in the literature. It is generally attributed to a persistence of primitive
control strategies, that normally diminish during childhood (Lin, 2004; Fowler and Goldberg, 2009). Usually, distal joints are more severely involved, as are bi-articular muscles (Gage, 2004). Poor selective control can lead to a loss of dexterity, synergistic movement patterns such as flexion or extension synergies, excessive co-contraction, or mirror movements (Gage, 2004; Fowler and Goldberg, 2009).

*Paresis*, as a primary result of the upper motor neuron syndrome, is caused by a decrease of cortical input to the reticulospinal and corticospinal tracts, which decreases the number of effective motor units, leading to weakness (Koman et al., 2004).

**Secondary impairments**

Secondary impairments arise over time as a result of abnormal usage or loading of muscles and bones, and can be due to primary impairments or to abnormal movement or posture.

*Muscle contractures* or ‘short muscles’ are common in CP, and measured by reduced range of motions in physical examination. The reduced muscle-tendon length is thought to be caused mainly by reduction of the muscle belly length, but the exact mechanisms underlying the

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**Figure 1.2:** Overview of the main impairments in CP, some of the main gait deviations, as well as their mutual dependency. This figure may be seen as a further specification of the grey box in Figure 1.1. Primary impairments (A; + excess; - deficit symptoms) are a direct result of the upper motor neuron lesion (UMNL), while secondary impairments (B) develop over time as a result of primary impairments and altered loading. Both primary and secondary impairments affect muscle behavior during gait (C), which in turn leads to deviations in joint and segment kinematics and kinetics (D). Tertiary impairments (i.e. compensation strategies) and dynamic factors during gait (E) can also affect the joint and segment motions. Gait deviations can lead to limitations in walking performance (F), such as reduced walking speed, which may in turn alter muscle behavior, joint/segment kinematics and gait dynamics. Underlined are aspects that are studied in this thesis. Primary and secondary impairments can be measured with physical examination; gait deviations at the muscle level with electromyography and musculoskeletal modeling; gait deviations at joint/segment level with 3D motion analysis.
shortening of muscles are unclear. Reduction of the number of sarcomeres in series or in parallel (Mohagheghi et al., 2008), and muscle atrophy (Shortland et al., 2002) have all been mentioned. These may be due to reduced use of the muscle (Lieber et al., 2004).

Related to muscle contracture is increased muscle stiffness. Intrinsic muscle properties of spastic muscles has been reported to change over time, resulting in changes in fiber structure and increased stiffness of muscle cells (Lieber et al., 2004; Foran et al., 2005). Furthermore, due to changes in the extracellular matrix, intermuscular connections and myofascial force transmission may be altered in spastic muscles, both between synergistic and antagonistic muscles, which may be one of the factors that lead to reduced range of motion and increased overall stiffness of muscles in CP (Lieber et al., 2004; Huijing, 2007). Furthermore, changes in muscle fiber size and fiber type distribution have been reported to occur in spastic muscles, which may also affect the fatigability of muscles (Lieber et al., 2004).

Bony or joint deformities can arise secondary to increased or altered loading. Hip subluxation, excessive tibial torsion, femoral anteversion, and foot deformities are all common in CP. Bony deformities can lead to lever-arm dysfunction, in which the leverage of muscles is distorted due to the deformity (Gage, 2004).

Muscle weakness can, apart from being a primary effect of the upper motor neuron syndrome (paresis), also arise as a secondary impairment, for example due to inactivity or reduced use of specific muscles.

1.4. Gait pathology in CP

Classification and terminology of gait patterns

About 70% of children with CP are able to walk, either with or without assisting devices, at the age of five (Beckung et al., 2008). Although the gait patterns of these ambulatory children can differ vastly from patient to patient, several characteristic patterns can be observed. Different classification schemes are proposed in the literature to describe these common gait deviations in CP.

The classification most commonly used in the Netherlands is that by Becher et al (2002), which is illustrated and explained in Figure 1.3. In the international literature, a different terminology is most common, although the exact definitions and classified groups vary (Dobson et al., 2007). The following gait deviations are most common and most relevant for this thesis (listed in Figure 1.2D).

Crouch gait is generally referred to as a gait pattern with excessive knee flexion in stance (Wren et al., 2005). Since this can include many different patterns in hip and ankle, sometimes the term is specifically used for gait patterns with excessive knee flexion in combination with ankle dorsiflexion in stance (Sutherland and Cooper, 1978; Miller et al., 1995; Rodda et al., 2004). Other studies reserve the term for limited knee extension in terminal swing or at initial contact, not focusing on the entire stance phase (Arnold et al.,
is referred to as a gait pattern with excessive ankle plantar flexion in stance (Miller et al., 1995; Rodda et al., 2004).

The term equinus is used to describe a toe-walking gait pattern with excessive knee flexion in mid-stance and heel rise in mid-stance arises. Depending on the degree of flexion in hip, knee, and ankle, this pattern can either be just a mild deviation, or a severe case, with risk of loss of walking ability.

In this thesis, the term crouch gait will be used in a broad manner, to identify all gait patterns with excessive knee flexion throughout stance.

Equinus gait is referred to as a gait pattern with excessive ankle plantar flexion in stance (Wren et al., 2005). Sometimes a distinction is made between true and apparent equinus, with true equinus indicating excessive ankle plantar flexion in stance, and apparent equinus indicating a toe-walking gait but with a neutral ankle angle (Miller et al., 1995; Rodda et al., 2004). In the present thesis, the term equinus is used to describe true equinus, so increased ankle plantar flexion in stance.

Stiff knee gait indicates a gait pattern with limited knee flexion in swing (Sutherland and Davids, 1993; Wren et al., 2005).

Jump knee gait is used to describe a toe-walking gait pattern with excessive knee flexion in early stance, with the knee extending to a variable degree in late stance (Rodda et al., 2004).

Knee recurvatum indicates knee hyperextension in mid or terminal stance (Sutherland and Davids, 1993).

Most classifications systems, including those described above, classify gait patterns based on gait deviations in the sagittal plane. These gait patterns typically coincide with deviations in the frontal and transversal plane. For example, hip endorotation and adduction in terminal swing and stance are often seen in CP. Transversal and frontal deviations of the pelvis motion during gait are also quite common, as well as abnormal motion and positioning of
the trunk, and various foot deformations (Perry, 1992). Furthermore, these deviations in
kinematics obviously coincide with deviations in kinetics, such as reduced push-off or
increased joint torques.

**Clinical gait analysis**

Several tools exist to quantitatively assess gait disorders, that are collectively referred to as
clinical gait analysis (Kirtley, 2006). Over the last decades the development of laboratories for
gait analysis has greatly contributed to the enhanced assessment of gait disorders, both for
scientific purposes and for clinical decision making and evaluation (Narayanan, 2007; Gough
and Shortland, 2008). The increasing impact of clinical gait analysis is reflected in the rapid
growth of European and American scientific societies that focus specifically on this
interdisciplinary field. The different tools typically used in clinical gait analysis are briefly
outlined below.

First, video recordings are used for clinical observation of gait. With the use of multi-media
technology, these video’s can be displayed in a comprehensible manner, and supplemented
with quantitative gait graphs (Harlaar et al., 1998; Out et al., 2006). Video gait data can be
semi-quantified using clinical scales, such as the Edinburgh GAIT scale (Read et al., 2003).

For the quantitative assessment of gait in three dimensions, optical 3D motion analysis
systems are used that record the motion of passive (light-reflecting) or active (light-emitting)
markers in time. These markers are placed on the body segments, either directly on
anatomical bony landmarks, or using a standardized marker set-up, or using technical
clusters of markers in combination with virtual anatomical markers (i.e. the position of the
anatomical landmark relative to the technical cluster is indicated in a single palpation). Using
an anatomical model of the human body, segmental coordinate frames are then defined,
describing the 3D orientation and position of the body segments in time relative to a global
reference frame. These are used to calculate segment and joint angles over time in three
dimensions, describing the motion of the subject during gait (Cappozzo et al., 2005).

The 3D kinematic data can be supplemented with ground reaction force data using floor-
mounted force plates, to calculate joint kinetics. In combination with data on segment mass
and inertia, net joint moments and powers can be calculated, that display the net result of all
muscle actions.

To gain insight into the role of individual muscles, muscle electromyography (EMG) signals
can be recorded using surface electrodes mounted on the skin. EMG signals show the
electrical activity of muscles, indicating the timing of muscle activity. This gives information
about the coordination of different muscle groups during gait, for example in terms of
prolonged or abnormal activity patterns, co-contraction between agonist and antagonist
muscles, and synergistic patterns between different flexor or extensor muscles.

The studies in this thesis make use of quantitative 3D kinematic analysis of gait, using an
active marker system with technical cluster methodology, in combination with analysis of
EMG activity.
1.5. Relationship between impairments and gait pathology

Interplay between impairments and gait parameters

As described in Paragraph 1.3 and illustrated in Figure 1.2, many impairments can (co-)exist in a child with CP that can all influence the gait pattern. This makes it difficult to determine what the precise underlying causes of a specific gait deviation are. Moreover, there are several other factors that complicate the determination of underlying causes of gait deviations, as outlined below.

First, the different impairments can interact, and can therefore be related (Figure 1.2 A⇒B). For example, spastic muscles will not allow stretch to the same degree as muscles with normal tone. As a result, muscle growth may not keep pace with bone growth, resulting in contracture (Gage, 2004). Furthermore, spastic muscles generally show higher intrinsic passive muscle stiffness (Lieber et al., 2004).

Second, an abnormal gait patterns may involve abnormal loading on muscles, joints, and bones, which can in turn lead to the development of secondary impairments (Figure 1.2 B⇒D). For example, heavy loading on joints and bones during standing and walking can lead to bony deformities such as foot deformations or rotational deformities of femur and tibia. Similarly, if a muscle is never stretched to its full length during gait or other activities, it tends to shorten structurally. A reduction in active life style due to activity limitations may result in reduced use and weakness. This makes it difficult to determine what the cause is, and what the effect.

Third, children may show abnormalities during gait that are not a direct effect of primary or secondary impairments, but that are ‘coping mechanisms’, to walk despite other problems. These compensations strategies or ‘tertiary impairments’ (Figure 1.2 D⇒E) are sometimes hard to distinguish from the primary and secondary disorders (Gage, 2004). For example, if knee flexion in swing is decreased, foot dragging can be prevented by vaulting to the contralateral side, or circumduction of the swing leg.

Finally, the gait pattern itself is a biomechanically complex task, in which nonlinear dynamics of a multi-linkage system need to be controlled (Figure 1.2 D⇒E). Deviations in one end of the chain of segments can affect joint and segmental motion within the entire body. For example, toe-walking by itself has been shown to affect entire lower limb and pelvis motion (Brummer et al., 2008). Moreover, a gait deviation in one part of the gait cycle may lead to deviations in subsequent parts of the gait cycle. For example, toe-walking has been hypothesized to lead to a stiff-knee gait pattern in swing (Kerrigan et al., 2001).

These factors make it difficult to determine cause and effect of impairments and gait deviations. Yet, in order to determine the best intervention for a patient and improve walking performance, it is essential to gain a good understanding of underlying causes of gait deviations.
Determining the causes of gait deviations

Many years’ experience in the evaluation and treatment of CP gait has resulted in a large amount of knowledge of gait deviations and treatment options (Perry, 1992; Gage, 2004). However, due to the factors as described above, the specific effect of underlying impairments on gait is often not clear. One of the main gaps in the current literature is that the clinical effect of spasticity, i.e. the effect of spasticity during functional tasks such as walking, has hardly been established (Lin, 2004).

A possible way to gain insight into the effect of specific impairments on the gait pattern is by correlating the severity of the impairment to specific gait deviations. Desloovere et al. (2006) investigated the relationship between a large set of gait analysis data, including kinematics, kinetics, and EMG, and clinical measurements, including range of motion, spasticity, alignment, strength, and selectivity. They found only fair to moderate correlations, and concluded that gait analysis data cannot be sufficiently predicted by a combination of clinical measurements.

Several other studies have related spasticity measures to typical gait parameters such as joint angles or angular velocities (Tuzson et al., 2003; Damiano et al., 2006; Ross and Engsberg, 2007). The results of these studies were ambiguous, and correlations between impairments and conventional gait parameters, if present, were often weak. The same was true for the correlations between passive ranges of motion as measured during physical examination and gait deviations (McMulkin et al., 2000). Muscle strength and selectivity appear to be more closely related to gait parameters than spasticity or range of motion measures (Desloovere et al., 2006; Ross and Engsberg, 2007), which may indicate that these are more important factors for good walking performance. However, these correlations do not necessarily point to a causative relationship, which shows for example from the fact that the effects of strength training to improve gait so far have been inconclusive (Dodd et al., 2002). Moreover, all studies mentioned above related physical examination parameters to joint and segment angles or angular velocities during gait (Figure 1.2 A/B). This step from impairments to kinematics, or even to more general gait parameters such as walking speed or walking economy (Figure 1.2F), is quite large, since most impairments lay at muscle rather than joint level.

In order to relate impairments to gait data, it may be more fruitful to study muscle function during gait. This allows evaluating gait at the level of the impairments, which lay mostly at the muscle or muscle-tendon level (Figure 1.2C). Specifically, the effects of spasticity and contractures may slow more clearly in muscle-tendon behavior during gait than in joint kinematics. For example, it could be hypothesized that spasticity in the hamstrings limits muscle-tendon stretch velocity or peak muscle-tendon length reached during gait, which in turn may lead to decreased knee extension in terminal swing, or pelvic posterior tilt, or possibly hip rotation. Studying this ‘intermediate’ muscle level may thus give further insight than segment or joint parameters alone. Furthermore, many interventions interfere at the muscle level; therefore evaluation at the muscle level is closely related to possible treatment options.
However, muscle behavior during dynamic tasks is not easily quantified. EMG data give information on muscle activity, but muscle length or force cannot be readily retrieved from conventional kinematic data alone. Muscle-tendon lengths are not directly related to joint angles, because many important muscles span more than one joint, and muscle moment-arms can vary with joint angle. In order to calculate muscle-tendon length during gait, 3D kinematic data therefore need to be combined with computer simulations using models of the musculoskeletal system. Developments in musculoskeletal modeling techniques and computer capacity over the last decades have made it possible to use musculoskeletal models on patient gait data (e.g. Delp et al., 1990; 1998; 2007; Klein Horsman et al., 2007).

A number of studies have investigated muscle-tendon lengths during gait in order to evaluate the effect of impairments, especially of muscle contractures, on gait (e.g. Delp et al., 1996; Thompson et al., 1998; 2001; Arnold et al., 2006a; 2006b). These studies have yielded valuable insights into whether or not muscles present short length or slow stretch velocities during gait (Delp et al., 1996; Thompson et al., 2001; Jonkers et al., 2006), which proved to be a good indicator for success of orthopedic lengthening of muscles (Arnold et al., 2006a; 2006b).

However, only a few studies used musculoskeletal modeling to study the effects of spasticity (Crenna, 1998; Cheung et al., 2003; Jonkers et al., 2006). Furthermore, a confounding factor in all modeling studies mentioned above is the lack of correction for differences in walking speed. In general, the effect of walking speed is still poorly understood, and has hardly been studied in children with CP. Yet, many patients walk slower than typically developing children. Slow walking speed may by itself lead to ‘gait deviations’, for example to short muscle length or slow stretch velocity during gait, and is therefore important to consider when studying gait data.

1.6. Problem statement

From the previous, it follows that:

- First, a thorough understanding of underlying causes of gait deviations in CP is lacking. Especially, the clinical significance of spasticity on gait has not been well established in the current literature, and little is known about the effects of walking speed.

- Second, an essential step towards understanding the underlying causes of gait deviations in CP is to study gait at the level of the impairment of interest. The use of musculoskeletal modeling allows studying muscle-tendon lengths and velocities during gait. This may give further insight than looking at joint or segment kinematics alone, since no one-to-one relationship exists between joint kinematics and muscle behavior, especially when bi-articular muscles are involved.
Finally, correlating impairment measures to gait parameters alone does not, or only moderately, give insight into cause and effect relationships. Therefore, isolating the role of specific impairments during gait will be a main theme of this thesis, as outlined below.

1.7. Aim of this thesis

The general aim of this thesis is to gain insight into the underlying causes of gait deviations in children with spastic CP.

More specifically, this thesis focuses on:

- the role of spasticity;
- the interplay between the effects of spasticity, muscle contractures and walking speed;
- and
- the role of dynamics.

In an attempt to unravel these specific aspects of the gait disorder (i.e. spasticity, contractures, speed, and dynamics), several tools, measurement techniques, and analyses will be applied. These include musculoskeletal modeling, the use of healthy subjects as a model, modulation of spasticity effects by varying walking speed, measurement of spasticity directly during gait, and forward dynamic modeling techniques.

1.8. Approach and outline

Use of musculoskeletal modeling

As described above, musculoskeletal models can be used to estimate muscle-tendon lengths during gait, which allows evaluating gait at the level of impairments. Several musculoskeletal models exist in the literature that can be used to calculate muscle-tendon lengths during dynamic activity such as walking, using 3D kinematic data as input. These models can be incorporated in commercial packages such as SIMM (Delp et al., 1990), AnyBody (www.AnyBodyTech.com), or more recently in open-source software packages such as OpenSim (Delp et al., 2007).

The SIMM musculoskeletal model (shown in Figure 1.4) is one of the most wide-spread models used for calculation of muscle-tendon lengths in the literature. This 3-dimensional model has been developed by Delp et al. (1990; 1995) and used previously for the calculation of muscle-tendon lengths in CP. The full body model contains 86 degrees of freedom, 117 joints, and 344 muscle-tendon actuators. The joints have anatomically accurate kinematics, for example the knee model includes the sliding and rolling of the tibia and patella on the femur. Muscle paths are modeled using anatomical via-point and wrapping surfaces. The
model can be scaled to individual subject sizes, and can be made to match measured patient gait kinematics, in order to calculate muscle-tendon length and velocity during gait.

In Chapter 2 of this thesis, three different models for the calculation of hamstrings length will be compared in a validation study. Their accuracy to calculate peak hamstrings length will be evaluated at a range of combinations of hip and knee angles. In Chapter 3 to 6 of this thesis, the SIMM lower extremity model, scaled to individual subjects sizes, will be used to calculate semitendinosus, biceps femoris, psoas, gastrocnemius, and soleus lengths during gait.

**Use of healthy subjects as a model**

One method to investigate specific aspects of pathological gait in isolation is to use healthy subjects as a model, by letting them simulate one specific aspect of pathological gait, or by imposing one specific ‘impairment’. This allows studying these particular aspects in isolation, not including any other impairments or gait deviations. Several studies have investigated the effects of voluntary toe-walking (Davids et al., 1999; Riley and Kerrigan, 2001; Romkes and Brunner, 2007), voluntary crouch walking (Harlaar, 2003), or of imposed shortened hamstrings length (Matjacic and Olensek, 2007; Whitehead et al., 2007) in healthy subjects.

Chapter 3 of this thesis will adopt this approach in a study on voluntary crouch gait. This chapter will address the question whether crouch gait per se coincides with short muscle-tendon length or slow stretch velocity of hamstrings muscles during gait. This could give indirect evidence for the possible effect of contractures (short peak length) and spasticity (slow peak velocity) on crouch gait. This chapter also addresses the relative effect of crouch gait and variation of walking speed on hamstrings length and velocity.

**Modulating the effect of spasticity by varying walking speed**

Another way to study the effect of a specific impairment on gait is to modulate this impairment and investigate the effect. Intervention studies use this approach, for example by reducing muscle excitation with botulinum toxin (Scholtes et al., 2007b), or increasing muscle strength with a strength training program (Dodd et al., 2002), and evaluating the effect on the gait pattern. However, these studies are time-consuming, and it is often difficult to interfere in only one specific impairment. Since spasticity is defined as a velocity-dependent phenomenon, the effects of spasticity in particular can also be modulated by imposing different walking speeds. Increasing walking speed could be expected to increase the velocity with which muscles are stretched, thereby enhancing the effects of spasticity.
Chapter 1

Chapter 4 to 6 of this thesis will use this approach to try and gain a better understanding of the specific effects of spasticity on gait, as well as of the effects of walking speed itself. Chapter 4 evaluates the relationship between hamstrings spasticity as measured during a clinical spasticity test and peak hamstrings length and lengthening velocity during gait, for a range of walking speeds. Chapter 5 evaluates muscle-tendon length and lengthening velocity during gait for the gastrocnemius and soleus muscles. Spastic calf muscles with and without contractures in children with CP will be compared to muscles in typically developing children.

**Measuring spasticity directly during gait**

Instead of correlating spasticity as measured during passive testing to gait parameters, it is also possible to study spasticity effects directly during gait. This can be done by studying the ‘velocity-dependent increase in muscle tone’ during gait, by relating muscle activity to muscle-tendon stretch velocity. This method, termed dynamic spasticity, has been proposed by Crenna (1998) and will be investigated in Chapter 6 of this thesis. In this chapter, the dynamic spasticity of the plantar flexor muscles is investigated, by relating phases of muscle-tendon stretch to muscle activity.

**Forward dynamic modeling**

A different approach that is particularly suitable to study specific aspects of pathological gait in isolation is the use of forward dynamic simulation of gait. Forward dynamic simulation follows the natural way of causality: the gait pattern is ‘synthesized’ by applying forces or moments to a biomechanical model of the human body and observing the resulting movement. This allows answering hypothetical ‘what if’ questions, by changing one or more of the parameters of the model and evaluating the resulting output. In the literature, two main approaches exist to forward dynamic modeling of gait: one using complex musculoskeletal models (similar to the abovementioned models for muscle-tendon length calculation), in which the gait data of subjects are ‘tracked’ to achieve a forward dynamic simulation based on inverse analysis, (e.g. Delp et al., 2007). Opposed to this more complex approach is the so-called dynamic walking approach, which uses simpler, conceptual models that allow studying basic principles of human gait in a more fundamental manner. Since these models synthesize a gait pattern that is repeatable (i.e. it can produce perpetual, stable gait), they are also called limit cycle walking models. In Chapter 7 of this thesis, the latter approach will be applied to study factors that may lead to a stiff-knee gait pattern. This chapter presents a forward dynamic model of normal and crouch gait. Using this model, the effects of a crouched posture, as well as the effects of push-off strength and hip torque on the dynamics of the swing leg are studied.

In Chapter 8 the main findings of this thesis will be summarized and discussed. This chapter provides an evaluation of the methods used, reflects on the fundamental and clinical implications of this research, and gives some recommendations for further study.
Chapter 2

Validation of hamstrings musculoskeletal modeling by calculating peak hamstrings length at different hip angles

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Caroline A.M. Doorenbosch
Jaap Harlaar
Abstract

Introduction. Accurate estimates of hamstrings lengths are useful, for example to facilitate planning for surgical lengthening of the hamstrings in patients with cerebral palsy. In this study, three models used to estimate hamstrings length (M1: Delp, M2: Klein Horsman, M3: Hawkins and Hull) were evaluated.

Methods. This was done by determining whether the estimated peak semitendinosus, semimembranosus and biceps femoris long head lengths, as measured in eight healthy subjects, were constant over a range of hip and knee angles.

Results. The estimated peak hamstrings length depended on the model that was used, even with length normalized to length in anatomical position. M3 estimated shorter peak lengths than M1 and M2, showing that more advanced models (M1 and M2) are more similar. Peak hamstrings length showed a systematic dependence on hip angle for biceps femoris in M2 and for semitendinosus in M3, indicating that either the length was not correctly estimated, or that the specific muscle did not limit the movement.

Interpretation. Considerable differences were found between subjects. Large inter-individual differences indicate that modeling results for individual subjects should be interpreted with caution. Testing the accuracy of modeling techniques using in vivo data, as performed in this study, can provide important insights into the value and limitations of musculoskeletal models.
2.1. Introduction

Modeling of muscle-tendon complex lengths has been an important object of study for a long time. For example, knowledge about (maximal) hamstrings length during physical examination and gait is essential to facilitate planning for surgical lengthening of the hamstrings in children with cerebral palsy (e.g. Delp et al., 1996; Thompson et al., 1998; Ma et al., 2006; Arnold et al., 2006b). Estimates of peak hamstrings lengths have also been used to study the effects of muscle stretching (Halbertsma and Goeken, 1994; Halbertsma et al., 1999) and the relationship between extensibility of hamstrings and low back pain (Halbertsma et al., 2001).

A number of musculoskeletal models to estimate hamstrings muscle-tendon length have been described in the literature (e.g. Brand et al., 1982; Delp et al., 1990; Hawkins and Hull, 1990; Visser et al., 1990; Van Soest et al., 1993; Klein Horsman et al., 2007). These models are all based on cadaver measurements. Some used geometrical rules to calculate muscle-tendon length from the origin and insertion on the skeleton (Brand et al., 1982), others calculated muscle-tendon length directly in cadaver muscle as a function of joint angle changes (Visser et al., 1990), or used indirect estimates based on data from other studies (Hawkins and Hull, 1990; Van Soest et al., 1993). Advanced models also used so-called via-points and wrapping surfaces (Delp et al., 1990; Klein Horsman et al., 2007). Due to differences in parameters and derivation methods used, these models may yield considerably different results, possibly influencing interpretations of the role of hamstrings.

One way to test the accuracy of hamstrings musculoskeletal modeling is to measure peak hamstrings length for different hip and knee angle combinations. It can be assumed that at force levels that are applied during common physical examination, peak hamstrings length is independent of the hip and knee angle combination in which it is measured. This independence is therefore an indication of the accuracy of the estimated hamstrings length. If, however, calculated peak hamstrings length depends systematically on hip and knee angle, this may indicate an erroneous estimation of hamstrings length.

Therefore, the goal of the present study was to compare peak hamstrings length calculated with three different musculoskeletal models, for a range of hip and knee angle combinations. It was hypothesized that all models would estimate similar and constant peak hamstrings length.
2.2. Methods

Subjects

Eight healthy adult subjects participated in this study. Their characteristics are presented in Table 2.1. All subjects signed informed consent forms.

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<th>Gender</th>
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<td>F</td>
</tr>
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<td>30</td>
<td>M</td>
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<tr>
<td>7</td>
<td>1.80</td>
<td>67</td>
<td>43</td>
<td>F</td>
</tr>
<tr>
<td>8</td>
<td>1.75</td>
<td>70</td>
<td>23</td>
<td>M</td>
</tr>
</tbody>
</table>

Mean 1.76 70.3 33.4
SD 0.10 11.4 9.1

Design

Physical examination of the hamstrings muscles of the right leg was performed in all subjects (Figure 2.1). The subjects were lying on their left side on a bench, with the right leg horizontally supported on a table, to exclude effects of gravity. The right hip was fixed by the researcher in angles of approximately 70°, 80°, 90°, 100°, 110°, and 120°, randomly sequenced. In each position, the knee was passively brought to extension in order to achieve maximal stretching of the hamstrings. All movements were performed at similar slow velocities so that maximal extension was reached in approximately 5 s. A hand-held dynamometer was used to measure the exerted force. The hamstrings were stretched until an external moment of approximately 20 Nm was applied at the knee, comparable to standard clinical passive muscle testing. During each trial the hamstrings were stretched three times, and three trials were carried out for each hip position. If full knee extension was reached before the hamstrings were maximally stretched, which occurred at smaller hip angles in more flexible subjects, the trial was excluded.

Kinematics

3D kinematic data were collected for the pelvis, thigh and shank of the right leg, using a motion capture system (Optotrak, Northern Digital). Technical clusters of three markers were attached to sacrum, back of thigh and shank, respectively. With the subject standing in anatomical position, the position of relevant bony landmarks was measured in order to anatomically calibrate the technical cluster frames (Cappozzo et al., 1995). The right anterior superior iliac spine was also probed in supine position for each hip angle, in order to optimally estimate the pelvic position during the examination.
Validation of hamstrings musculoskeletal modeling

Electromyographic (EMG) signals of the semitendinosus (ST) and biceps femoris (BF) long head were recorded. These data were used to control for possible involuntary activation of the muscles as a reaction to passive knee extension. Skin preparation and electrode placement were carried out according to SENIAM guidelines (Freriks et al., 1999). EMG data were collected at 1000 Hz, and off-line high-pass filtered at 20 Hz to remove artifacts.

Calculation of muscle-tendon lengths

3D kinematic data were analyzed with custom-made software (BodyMech, Matlab®, The Mathworks). Hip and knee joint angles were calculated according to the CAMARC anatomical frame definitions (Cappozzo et al., 1995).

Lengths of semimembranosus (SM), ST, and BF were calculated with the following models:

- M1: SIMM (Delp et al., 1990; 1995);
- M2: The Twente Lower Extremity Model (Klein Horsman et al., 2007); and

These models were chosen because they are based on different methods and/or datasets, representing a broad range of the available methods. For all models, ST, SM, and BF lengths were normalized to their length in anatomical position, defined as 100%, to exclude scaling effects. Characteristics of the models and their methods of calculating muscle-tendon length are shown in Table 2 and described here.

For M1, the SIMM standard generic model was used, scaled to the individual subject sizes using 3D co-ordinates of bony landmarks in the reference position. Next, 3D co-ordinates of the bony landmarks during passive movement trials were entered into the model and the lengths of the three hamstrings muscles were calculated.

Figure 2.1: Experimental setup. Additional information is indicated in the picture with arrows.
Chapter 2

M2 is based on a complete dataset of one cadaver. This is a two-legged model, in which 10 joints are crossed by 264 muscle elements. Moment arms of all muscle elements are simulated as a function of the corresponding joint angles. The model was scaled per segment, using pelvic width, thigh length, and shank length as scaling factors.

M3 was constructed to determine muscle-tendon length for 16 muscles, based on joint angles and easily measured anthropometric parameters. For various lower extremity joint flexion angle combinations in six subjects, Hawkins and Hull (1990) determined muscle origin and insertion locations, based on cadaver origin and insertion information (Brand et al., 1982) and individual anthropometric parameters. From these data, they derived regression equations with which normalized muscle-tendon lengths can be estimated from joint flexion angles only. For the three hamstrings muscles, the following equations were derived, with correlation coefficients of 0.98, 0.97, and 0.97, respectively:

\[
L_{SM} = 1.027 + 1.99E-3 \times \phi_{HIP} - 2.22E-3 \times \phi_{KNEE} \tag{1}
\]

\[
L_{ST} = 0.987 + 2.07E-3 \times \phi_{HIP} - 1.78E-3 \times \phi_{KNEE} \tag{2}
\]

\[
L_{BF} = 1.048 + 2.09E-3 \times \phi_{HIP} - 1.60E-3 \times \phi_{KNEE} \tag{3}
\]

with \(L_{SM}\), \(L_{ST}\), and \(L_{BF}\) being the lengths of ST, SM, and BF, respectively, as a percentage of thigh length, and \(\phi_{HIP}\) and \(\phi_{KNEE}\) the hip and knee flexion angles in degrees, with anatomical position being zero.

**Table 2.2: Model details**

<table>
<thead>
<tr>
<th>Model</th>
<th>Reference</th>
<th>Source of anatomical data</th>
<th>MTC length calculation method</th>
<th>Input</th>
<th>Scaling</th>
</tr>
</thead>
<tbody>
<tr>
<td>M1</td>
<td>Delp et al. (1990)</td>
<td>Brand et al. (1982), various other sources, own adaptations</td>
<td>Bone attachment sites, via point and wrapping surfaces</td>
<td>Anatomical landmarks</td>
<td>Built-in scaling using reference posture</td>
</tr>
<tr>
<td>M2</td>
<td>Klein Horsman et al. (2007)</td>
<td>One human cadaver</td>
<td>Bone attachment sites, via point and wrapping surfaces</td>
<td>Joint angles</td>
<td>Per segment</td>
</tr>
<tr>
<td>M3</td>
<td>Hawkins and Hull (1990)</td>
<td>Brand et al. (1982)</td>
<td>Regression equation based on hip and knee angle</td>
<td>Joint angles</td>
<td>Thigh length</td>
</tr>
</tbody>
</table>

**Data analysis**

Peak SM, ST, and BF lengths were calculated as outcome measures for all trials. First, muscle-tendon length was plotted versus the force exerted on the muscle. This passive muscle force was estimated by dividing the external moment of the dynamometer by the muscle moment arm, and by three, assuming that force was equally distributed over the hamstrings muscles. This was done for each muscle, for each model and for each trial. Figure 2.2 shows an
example of ST length versus force for a typical trial of one subject. As can be seen, the curves level off at high muscle force, and increasing force has little influence on peak muscle-tendon length. For this reason, and because of the assumptions that had to be made in calculating the muscle force, it was considered appropriate to calculate peak muscle-tendon length as outcome measure, independent of the exact muscle force that was applied in the trial.

Statistics
A linear regression analysis was performed to calculate the dependence of peak muscle-tendon length on hip angle for each subject and for each model. The slopes of the fitted lines were calculated and a Student’s t-test was performed to determine whether these differed significantly from zero. *P*-values less than 0.05 were considered to be statistically significant.

2.3. Results
All measurements were performed successfully. Peak hamstrings length was tested for all subjects over a range of about 40° difference in hip angles. Peak moment delivered by the hand-held dynamometer was 20.9 ± 3.0 Nm, and was constant over the range of hip angles. EMG signals were generally absent or low, and did not show any notable differences between the conditions.

Peak knee angles reached during all trials were highly linearly related to hip angle (*r* = -0.98 ± 0.02, *p*<0.001, Figure 2.3). The ranges of hip angles and peak knee angles differed between subjects, due to differences in hamstrings flexibility. The slopes of the curves in Figure 2.3 were 1.22 ± 0.16, indicating that the knee moment arm was approximately 20% smaller than the hip moment arm.
Figure 2.3: Peak knee angles reached during all trials versus hip angle for all subjects (indicated with S1–8), including regression lines.

Figure 2.4 shows accompanying peak SM, ST, and BF lengths, normalized to length in anatomical position, versus hip angle for all three models, including regression lines. Table 3 shows values for the slopes of the regression lines shown in Figure 2.4. M3 estimated shorter relative peak muscle-tendon lengths for all muscles, compared to M1 and M2 (p=0.001). Peak hamstrings lengths as estimated by the three models were not constant over hip angles in all cases: M3 estimated a systematic decrease in SM length with increasing hip angle (p<0.001), whereas M2 estimated a systematic increase in BF length with increasing hip angle (p=0.01).

Table 2.3: Slope of regression lines of peak semimembranosus (SM), semitendinosus (ST) and biceps femoris long head (BF) length versus hip angle as shown in Figure 2.4, expressed in % of reference length per degree of hip flexion; for M1 (Delp), M2 (Klein-Horsman), and M3 (Hawkins & Hull).

<table>
<thead>
<tr>
<th>subject</th>
<th>SM M1</th>
<th>ST M1</th>
<th>BF M1</th>
<th>SM M2</th>
<th>ST M2</th>
<th>BF M2</th>
<th>SM M3</th>
<th>ST M3</th>
<th>BF M3</th>
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<td>0.01*</td>
</tr>
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<td>0.04</td>
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<td>-0.01</td>
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<tr>
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<td>p</td>
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<td>0.00**</td>
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<td>0.01*</td>
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</table>

* p-values < 0.05 denote a significant difference from zero.
Large differences were found between subjects. For example, M1 estimated decreasing muscle-tendon lengths at greater hip angles, as seen in subjects S2 and S5 (Figure 2.4). Peak muscle-tendon length estimates also showed systematic dependence on hip angle over all muscles and over all models for four of the eight subjects (Figure 2.4, Table 3, rightmost columns).

Figure 2.4: Peak length of semimembranosus (SM, top row), semitendinosus (ST, middle row) and biceps femoris long head (BF, bottom row) reached during all trials versus hip angle for all subjects (indicated with S1–8), including regression lines; for M1 (Delpl), M2 (Klein Horsman), and M3 (Hawkins and Hull).
2.4. Discussion

Our evaluation of the modeling techniques revealed important differences. M3 estimated shorter peak muscle-tendon lengths than M1 and M2; both M2 and M3 showed a systematic dependence on hip angle for one of the three muscles; and large differences were found between subjects. The difference in peak length between M3 and M1/M2 can be explained by differences between the models. First, different types of models were used. M1 and M2 both were advanced musculoskeletal models, calculating the muscle-tendon lengths from geometrical sources such as attachment sites, via-points and wrapping surfaces. These two models did not differ much in calculated relative muscle-tendon length. The third model used only regression equations to calculate muscle-tendon length, with constant moment arms. Second, different anatomical datasets were used (Table 2).

Although M1 and M3 are both based on data from Brand et al. (1982), they still yield quite different results. This may be due to adaptations that were made to make the model more accurate (Delp and Loan, 1995), or to the simplifications of the model made by Hawkins and Hull (1990). Finally, in M3 only 2D flexion and extension angles for hip and knee were used for the modeling of hamstrings length, while for M1 and M2 3D data were used. However, this should not have much influence on the results, since care was taken to perform the trials in the sagittal plane. To understand the dependence on hip angle of BF length in M2 and of SM length in M3, the modeled moment arms need to be examined. In general, peak muscle-tendon length depends mainly on hip and knee angle and accompanying moment arms, although M1 and M2 use somewhat more complex ways of calculating muscle-tendon lengths. In formula

\[ L_{MUS} = R_{HIP} \times \phi_{HIP} + R_{KNEE} \times \phi_{KNEE} + C \]  

where \( L_{MUS} \) the length of one of the hamstrings muscles; \( R_{HIP} \) and \( R_{KNEE} \) the moment arms of the muscle around hip and knee respectively that can vary with joint angle; \( \phi_{HIP} \) and \( \phi_{KNEE} \) the joint angles of hip and knee in radians, and \( C \) a constant. From Figure 2.3, it can be derived what the ratio of hip and knee moment arms should be for each subject, in order to find a constant peak muscle-tendon length. Since hip and knee angle were linearly related, a constant ratio of moment arms, equal to the slopes in Figure 2.3, would have lead to constant peak muscle-tendon length estimates:

\[ \frac{R_{HIP}}{R_{KNEE}} = 1.22 \pm 0.16 \]  

with 1.22 ± 0.16 being the average slope ± SD of the peak knee angle versus hip angle relationship (Figure 2.3). Thus, from our data it can be derived that, in the range of joint angles evaluated and for those muscles that limit the movement, the hip moment arm is approximately one-fifth larger than the knee moment arm, with considerable interindividual differences.

Figure 2.5 shows example moment arms for hip (Figure 2.5A) and knee (Figure 2.5B) for one subject, derived from the three models. Moment arms for the other subjects were very similar. If, in one of the models, the ratio between hip and knee moment arm differs too
much from the value predicted by the data, the estimated muscle-tendon length will not be constant. This effect is most obvious in M3. As derived from Eqs. [1]–[3], M3 predicts moment arm ratios of 0.90, 1.16, and 1.30 for SM, ST, and BF, respectively. The moment arm ratio for SM differs most from the value estimated from our data, leading to a peak length of SM that is not constant over the range of hip angles. Similar effects occur in M2 for BF: M2 estimates decreasing moment arms with hip flexion for BF, and increasing moment arms in the knee with flexion. Thus, the moment arm ratio ranges from approximately 3.5 at the smallest (most extended) hip angles where the knee is almost fully extended, to about 1 at more flexed positions. For SM and ST, the moment arms are more constant for this range of knee angles, leading to more constant ratios between 1 and 2.

These results can indicate that either the moment arms are not correctly estimated, or that the specific muscle does not limit the movement. Although it is likely that all hamstrings muscles are close to maximal in all positions, only the length of the muscle that yields the highest passive force will restrict the movement and determine the hip angle to knee angle relationship. It is difficult to know which muscle is limiting the movement, since it is not known how the external moment is divided over the three muscles. To get an indication of what is happening, we need to look at all three muscles. M3 estimates very constant peak ST and BF lengths for almost all subjects, indicating that, according to this model, these two muscles limit the movement, and that their moment arm ratio is correctly estimated. M2 shows large inter-individual differences for SM and ST, similar to M1 for all muscles (Figure 2.4, Table 3). It seems that in these cases there may be individual errors in moment arm ratio that are contradictory, leading to large individual dependences of muscle-tendon length on

![Figure 2.5](image_url)

**Figure 2.5**: A Hip and B knee moment arms versus joint angle for one representative subject of semimembranosus (thin dark lines), semitendinosus (thick dark lines) and biceps femoris long head (thick gray lines); for M1: Delp (solid), M2: Klein Horsman (dashes), and M3: Hawkins and Hull (dash/dot).
hip angle, but not to systematic deviations. No systematic deviations were found in any of the models for ST, which indicates that this muscle is most likely to restrict the movement.

For four of the eight subjects, a systematic dependence of peak hamstrings length on hip angle was found in all three models (Figure 2.4, Table 3, rightmost columns). This indicates that none of the models estimated hamstrings length correctly for these four subjects. Since these four subjects were all female, there could be some systematic difference between the generic models that were based on male data, and our female subjects. Therefore, some caution is needed when interpreting modeling results for individual subjects, especially when they are different from the population on which a model is based.

Two other factors that may have influenced our results should be mentioned. First, some of the model parameters were extrapolated, because hamstrings length was only modeled up to hip angles of approximately 120°. In order to calculate peak hamstrings lengths for all hip angles measured, the equations were extrapolated for high hip flexion angles. Specifically, M1 estimates decreasing muscle-tendon lengths with increasing hip angles for subjects 2 and 5, who were more flexible. This may be due to erroneous extrapolation in this area, in which the moment arms become very small (Figure 2.5).

Second, an important assumption in our study is that in vivo peak muscle-tendon length is constant, and that differences can be attributed to measurement or modeling errors. In other words, muscles are modeled as simple ‘strings’, with length solely depending on the hip and knee angles, weighted by their anatomical lever arm. However, recent findings in rat muscles show the presence of intermuscular connections that are not incorporated in these models (Huijing and Baan, 2003). These intermuscular connections may play a role, since part of the external force might not only be used to stretch the muscle, but also to stretch other tissue. The dependency of muscle-tendon length on relative position of the muscle (Huijing and Baan, 2003) may thus have contaminated our results. In the absence of sufficient knowledge about the quantitative effects of intermuscular connections for human hamstrings, these possible effects could not be accounted for.

Despite these factors, this study shows that validation of modeling using in vivo data can provide important insight into the value of musculoskeletal models. Our results show that peak knee angle is linearly related to hip angle, with the hip moment arm approximately one-fifth greater than the knee moment arm. Calculated peak hamstrings length depends on the model that is used, even when length is normalized. More advanced models (M1 and M2) show more similarity. Peak hamstrings length showed a systematic dependence on hip angle for BF in M2 and for SM in M3, indicating that either the muscle-tendon length was not correctly estimated, or that the specific muscle did not limit the movement. No systematic deviations were found for M1, indicating that this model represents our average data well and seems appropriate to be used for group comparisons. Inter-individual differences indicate that modeling results for individual subjects should be interpreted with caution.
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Chapter 3

Muscle-tendon length and lengthening velocity in voluntary crouch gait

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Abstract

Introduction. The purpose of this study was to explore how origin-insertion length and lengthening velocity of hamstring and psoas muscle change as a result of crouch gait. The second purpose was to study the effect of changes in walking speed, in crouch, on muscle-tendon lengths and velocities.

Methods. Eight healthy female subjects walked on a treadmill both normally and in crouch. In the crouch condition, subjects walked at three different walking speeds. 3D kinematic data were collected and muscle-tendon lengths and velocities were calculated using musculoskeletal modeling.

Results. Voluntary walking in crouch resulted in shorter psoas length compared to normal, but not in shorter hamstrings length. Moreover, crouch gait did not result in slower muscle-tendon lengthening velocities compared to normal gait. Decreasing walking speed clearly reduced muscle-tendon lengths and lengthening velocities.

Interpretation. These results do not support the role of hamstrings shortness or spasticity in causing crouch gait. Patients with short or spastic muscles are more likely to respond by walking slower than by walking in crouch. Also, differences in walking speed should be avoided as a confounding factor when comparing patient groups with controls.
3.1. Introduction

Crouch gait, a gait pattern characterized by excessive knee flexion in terminal swing and stance, is a frequently observed gait deviation among children with cerebral palsy (CP). No unambiguous definition of crouch gait is present in the literature. According to Perry (Perry, 1992), ‘excessive knee flexion’ in loading response is present when knee flexion is greater than 25° and ‘inadequate extension’ in mid stance and terminal stance when knee flexion remains greater than 10°. Oftentimes, a threshold value is used of 20° of minimal knee flexion during stance (Delp et al., 1996) or at initial contact (IC) (Arnold et al., 2006a; 2006b). Crouch gait in children should be subjected to treatment, as knee and hip flexion angles tend to increase with age due to increasing body weight, ultimately resulting in the loss of independent walking (McNee et al., 2004).

It is not well understood why many children with CP walk with excessive knee and hip flexion. Possible causes of crouch gait are short or spastic muscles. Especially contracture and spasticity of the hamstring muscles are often mentioned as possible causes of a flexed-knee gait pattern (Baumann et al., 1980; Perry, 1992; Crenna, 1998; Tuzson et al., 2003). However, contractures or spasticity in other muscles such as the psoas may also contribute to crouch gait (Delp et al., 1996).

So far, studies of the role of hamstrings length and velocity in crouch gait have been inconclusive. In a modeling study, Delp et al. (1996) found that in only 3 of 14 children with spastic CP walking in crouch, the hamstrings operated at shorter maximal muscle-tendon length than in healthy children. Thus, functional hamstrings length was adequate in most children, indicating that hamstrings contracture may not be the cause of crouch gait. In all children, the psoas muscle was shorter than normal during crouch walking (Delp et al., 1996). This could indicate that hip contractures may be a primary cause of crouch, although these could also be the result of compensation. In 1998, Thompson et al. (1998) had similar findings, showing that only 4 of 18 limbs had shorter hamstrings length during crouch gait than normal. Arnold et al. (2006a) also found that 35% of 152 CP patients walking in crouch had short hamstrings during gait. However, another 30% had not short but ‘slow’ hamstrings compared to normal. Surgically lengthening these short and/or slow muscles improved knee extension in terminal swing in most cases (Arnold et al., 2006a; 2006b), indicating that a relationship between hamstrings length and knee extension does exist. Thus, the exact relation between hamstrings length, hamstrings velocity and the severity of crouch gait remains unclear.

Moreover, all studies that have investigated muscle-tendon lengths and velocities during crouch gait show a number of compromising factors. First, muscle-tendon lengths and velocities in children with CP are compared to those in healthy children, walking at comfortable walking speed. As children with CP tend to walk slower than healthy children, walking speed in itself may have a large effect on hamstrings lengths and velocities. Second, a lot of variation usually exists among the gait patterns of CP patients. For example, in the studies of Arnold et al. (2006a; 2006b) both children with ‘jump knee’ gait and with persistent
crouch during stance were included. Third, variations may have existed between patients in their clinical history and in previous surgical procedures. All these factors obscure a clear view on the relation between hamstrings length and crouch gait.

In order to look at the effects of crouch gait without any interfering factors, healthy subjects walking in crouch can be used as a model. In this way walking speed can be controlled, and there are no side effects of pathologies or uncontrolled compensations. It also eliminates inter-subject variance, as all conditions can be performed by the same subjects. Healthy subjects have previously been used successfully as a model for pathological gait in children with CP (Thomas et al., 1996; Duffy et al., 1997; Harlaar et al., 2004; Matjacic and Olensek, 2007; Romkes and Brunner, 2007), but with different purposes.

The purpose of the current study was to examine how muscle-tendon lengths and velocities change as a result of walking in crouch. We focused on three muscles that are commonly suspected to cause crouch gait in children with CP: psoas, medial and lateral hamstrings (semitendinosus and biceps femoris). We hypothesized that these muscles would show shorter than normal peak lengths and slower than normal peak lengthening velocities during crouch gait. Second, we studied the effect of different walking speeds, in crouch, on muscle-tendon lengths and velocities. We expected that decreasing walking speed would decrease muscle-tendon lengths and lengthening velocities.

### 3.2. Methods

#### Subjects

Eight healthy female subjects were included in this study. The characteristics of the subjects (mean ± SD) were as follows: age 22.1 ± 1.1 years; weight 61.8 ± 6.5 kg; length 1.70 ± 0.06 m. All subjects provided informed consent.

#### Design

The subjects walked on a treadmill both normally and in crouch. Normal gait (NORM) was performed at self-selected comfortable walking speed (CWS), which was measured beforehand while walking over ground on a 50 m track. Crouch gait was performed at this same speed with 20° of knee flexion at midstance (CWS20) and with 30° of knee flexion at midstance (CWS30). Deep crouch (30° flexion) was also performed at walking speeds of 0.8, 1.1, and 1.4 m/s (SLOW30, MID30, and FAST30). The knee flexion angle was set with a goniometer while standing, while a rope was tightened mediolaterally in front of the subjects, at eye-height. During walking, subjects were instructed to keep this rope in front of their eyes, to keep the trunk upright and not to walk on their toes. Other than that they were free to walk as they preferred. They walked in each condition for 4 min, in order to accommodate to the new situation. The recordings of the final minute were used for analysis.
Voluntary crouch gait

Kinematics

3D kinematic data were collected for the pelvis, thigh, shank, and foot of the right leg, using a motion capture system (Optotrak, Northern Digital, Waterloo, Canada). A technical cluster of three markers was attached to each segment. While standing in an anatomical position, fifteen anatomical landmarks were indicated in order to anatomically calibrate the technical cluster frames (Cappozzo et al., 2005). These were for the pelvis: the left and right anterior and posterior superior iliac spines; for the thigh: the greater trochanter and the medial and lateral epicondyle; for the shank: the tibial tuberosity, the head of the fibula, and the medial and lateral malleolus; and for the foot: the calcaneus and the first, second, and fifth metatarsophalangeal joints.

Data analysis

3D kinematic data were analyzed using custom made software (BodyMech, Matlab®, The Mathworks). Initial contact (IC) and toe-off (TO) values were calculated using the forward foot velocity, and defined as the instants where this velocity became lower (IC) or higher (TO) than 20% of its maximal value. This method was derived comparing kinematic data with force plate data in previous experiments. Kinematic data were divided into strides from IC to TO. The first 10 strides of the final (fourth) minute of each trial were taken for further analysis. Joint and segment angles were calculated following the CAMARC anatomical frame definitions (Cappozzo et al., 1995). The spatio-temporal parameters stride length, stride frequency, and the percentage of stance time in the total gait cycle were also calculated.

The data were normalized to time per stride, yielding normalized time of 0–100% of gait cycle, and averaged over 10 strides for each subject. Mean knee angles during 0–50% of the gait cycle, covering loading response, midstance, and terminal stance, and excluding rapid knee flexion in pre-swing, were calculated and used as a measure of crouch.

SIMM modeling software (Delp et al., 1990) was used to calculate muscle-tendon complex lengths for three muscles. These were the main muscles that are often spastic or contracted in children walking in crouch, i.e. psoas, semitendinosus, and biceps femoris (long head). Semimembranosus was not analyzed as this muscle operates at similar muscle-tendon length.
as the semitendinosus during walking (Schutte et al., 1997; Arnold et al., 2001). The SIMM standard generic model was used and scaled to the individual subject sizes, using 3D kinematic data from the anatomical landmarks. Muscle-tendon lengths were differentiated to obtain muscle-tendon velocities. Both muscle-tendon lengths and velocities were normalized to time and averaged over 10 strides for each subject. Peak muscle-tendon length and lengthening velocities were calculated and statistically analyzed for all muscles.

Statistics

A repeated measures analysis of variance (ANOVA), with Bonferroni adjustment for multiple comparisons as post hoc test, was used to investigate the effects of crouch gait and walking speed on kinematics, spatio-temporal parameters, muscle-tendon lengths and velocities. *P*-values less that 0.05 were considered statistically significant. If the sphericity assumption was not met, a Huynh-Feldt adjustment was used.

3.3. Results

Effects of walking in crouch

Figure 3.1 shows a schematic illustration of one subject walking both normally and in CWS30. For a proper interpretation of the muscle-tendon length data, we performed two checks on the measured data. First it was investigated whether the subjects performed crouch gait with knee flexion as intended. Figure 3.2 shows segment and joint angles for NORM, CWS20, and CWS30, averaged over all subjects. CWS was 1.41 ± 0.14 m/s.

Figure 3.2: Average sagittal A pelvis, B hip, C knee, D ankle, and E foot angles for normal gait (NORM), 20° (CWS20), and 30° (CWS30) crouch gait. Pelvis and foot angles are relative to global. *Indicates a significant difference between the conditions.
Voluntary crouch gait

Mean knee angles averaged over 0–50% of gait cycle were 12.6 ± 2.8°, 18.2 ± 4.7°, and 27.3 ± 6.7° for NORM, CWS20, and CWS30, respectively (p<0.001), which shows that knee angles increased with crouch as imposed. Hip flexion and ankle dorsiflexion during the stance phase increased significantly with crouch as well (p=0.001 for both hip and ankle), whereas sagittal pelvis and foot angles relative to the global reference frame remained constant (p=0.23 and p=0.79, respectively).

Second, the effects of crouch gait on spatiotemporal parameters were investigated. Table 1A shows stride length, stride frequency and the percentage of stance time during the gait cycle. No significant differences were found for any of these parameters between NORM, CWS20, and CWS30. Therefore, possible differences in muscle-tendon lengths or velocities can be attributed to differences in kinematics, not to differences in stride length or duration.

Muscle-tendon lengths of the psoas, semitendinosus, and biceps femoris muscles during normal and crouch gait are shown in Figure 3.3A–C. The psoas acted at significantly shorter length during the crouch conditions compared to normal, due to differences in hip angle. The bi-articular hamstring muscles on the other hand, showed hardly any differences between normal and crouch gait. Although peak semitendinosus length showed a tendency to increase, it did not change significantly with crouch (p=0.12). The same was true for the peak length of the biceps femoris longus (p=0.06). Muscle-tendon velocities of the same three muscles during normal and crouch gait are shown in Figure 3.3D–F. Most surprisingly, peak semitendinosus and biceps femoris longus lengthening velocity increased with crouch gait (p<0.001). Peak psoas lengthening velocity did not change with crouch (p=0.15), while the average lengthening velocity during stance increased (p=0.012).

Effects of different walking speeds during crouch gait

Again, it was first examined whether the SLOW30, MID30, and FAST30 conditions were performed as intended. Knee flexion angles during stance for the three conditions were 31.1 ± 9.3°, 26.6 ± 7.2°, and 26.4 ± 8.8°, respectively. Thus, knee flexion in SLOW30 was approxi-
mately 5° higher than in the other two conditions ($p=0.02$). Table 1B shows spatio-temporal parameters for the SLOW30, MID30, and FAST30 conditions. As could be expected, both stride length and stride frequency decreased with decreasing walking speed ($p<0.001$). Also, stance percentage increased with decreasing walking speed ($p<0.001$).

Figure 3.4A–C shows muscle-tendon lengths for the psoas and hamstring muscles during the three different walking speeds in crouch. The excursion (maximal length minus minimal length) of all muscles decreased with decreasing walking speeds. Peak length of semitendinosus ($p=0.04$), biceps femoris ($p=0.03$), and psoas ($p=0.004$) all decreased with decreasing walking speed. Figure 3.4D–F shows the muscle-tendon velocities during the three different walking speeds in crouch. Not surprisingly, all velocities clearly decreased with decreasing walking speed.

3.4. Discussion

The main goal of this study was to investigate how muscle-tendon lengths and velocities change as a result of crouch gait. These findings might contribute to the discussion on why cerebral palsy patients walk in crouch. It was hypothesized that hamstrings and psoas would operate at shorter lengths and/or slower lengthening velocities during crouch, because this would indicate that muscle-tendon shortness and/or spasticity could be the cause of crouch gait. However, only psoas functioned at shorter length during crouch, and none of the muscles showed slower lengthening velocities in crouch.
Additionally, the effect of different walking speeds, in crouch, on muscle-tendon lengths and velocities was studied. It was shown that both hamstrings and psoas muscles functioned at shorter muscle-tendon lengths and at lower lengthening velocities when walking speed was decreased.

We used healthy subjects as a model, which enabled us to look specifically at the effects of a crouched position during gait. In comparison to crouch gait in patients with CP, simulated crouch gait is not influenced by inter-individual differences and has no side effects of pathologies or uncontrolled compensations. Furthermore, the use of healthy subjects made it possible to control for walking speed. On the other hand, there may have been differences in the way healthy subjects performed crouch gait and the way patients typically walk in crouch. Our subjects walked at relatively high walking speed (CWS of 1.41 ± 0.14 m/s), whereas most patients walk substantially slower. These high walking speeds required large steps, resulting in knee angles at IC that were smaller than 20° in some cases. According to the definition of Arnold et al. (2006a; 2006b) this would not be considered crouch gait. However, the crouched conditions still showed considerably more flexed knees than normal (Figure 3.2), which allows for a meaningful comparison between the two gait types.

There may also have been a difference between our subjects and crouch walking patients in the positioning of the pelvis. Although our instructions to the subjects did not include a standardization of pelvic positioning, subjects appeared to walk in crouch with an unchanged pelvic position. If we assume that patients walk with spastic or short hamstrings, they may show posterior pelvic tilt in terminal swing, to prevent further lengthening of the hamstrings. It is also known that many patients walk in crouch with anterior pelvic tilt,
especially in crouched gait patterns with equines (Perry, 1992). Therefore, our results cannot be generalized to all crouch walking patients with CP, because patients are not uniform in their performance of crouch gait with respect to pelvic positioning. Our results do show however, that hamstrings length is not influenced by the degree of crouch gait, and thus that anterior or posterior tilting of the pelvis will influence hamstrings length independently of walking in crouch.

Furthermore, as all three muscles under study showed higher than normal muscle-tendon lengthening velocities in crouch, crouch gait does not seem to be a way to prevent spastic responses, i.e. velocity dependent hyper reflexes (Lance, 1980). Based on the results of this study, only the psoas muscle functions at a shorter than normal length during crouch gait. Pure contracture of the psoas could therefore be hypothesized to be a cause of crouch gait, while pure contracture of the hamstrings could not.

These results seem contradictory to the results of a study by Matjacic and Olensek (2007), who studied artificially induced crouch walking in healthy adults by means of a psoas and hamstrings contraction emulation system. They found that when hamstrings or psoas or both were ‘contracted’, a flexed gait pattern arose, although with differences between the conditions. However, although their artificial, exoskeletal hamstring was said to be constructed in such a way as to act in parallel with hamstring muscles, no data on moment arms were given. From their pictures it can be derived that the moment arm about the knee is substantially larger than that about the hip, whereas anatomical studies show a smaller hamstrings moment arm about the knee (Chapter 2; Delp et al., 1990; Visser et al., 1990). These differences may explain the apparent contradictory results with our study.

Our results also show that reducing walking speed has a much larger effect on muscle-tendon lengths and lengthening velocity than walking in crouch. This indicates that lowering walking speed acts as an effective adaptation strategy to short or spastic muscles. The peak velocities of all muscles decreased almost linearly with walking speed, even though subjects walked with slightly higher knee flexion in the slowest walking condition. This result reinforces the results of others that differences in walking speed are an important confounding factor when comparing patient groups with controls (Stansfield et al., 2001a; 2001b; Hof et al., 2002).

The strong dependency on walking speed might explain why the results of the studies of Arnold et al. (2006a; 2006b) seem contradictory to our results. In their study, hamstrings length and velocity before and after surgery were compared for a large group of children with CP walking in crouch. They found that lengthening of those hamstrings that operated at short length or slow velocity pre-operatively resulted in increased length and velocity post-operatively in most cases, indicating that procedures were functionally effective. However, since no correction was made for walking speed, their results may, at least partly, be explained by differences in walking speed, rather than differences in flexed-knee gait.

A very important assumption in this study as well as in many other modeling studies (e.g. (Delp et al., 1996; Thompson et al., 1998; Arnold et al., 2006a; 2006b) is that muscles are modeled as simple strings, acting between origin and insertion. Geometrical origin-insertion
length might be a valid estimate for the total length of the muscle–tendon complex, especially when wrapping points are accounted for. However, recent findings have shown that intermuscular connections exist, which result in myofascial force-transmission between muscles and the surrounding connective tissue (Huijing and Baan, 2003). These cross-bindings influence muscle behavior and may cause that distal hamstring lengthening or shortening around the knee is not directly related to hamstrings lengthening or shortening around the hip. In a flexed-knee gait pattern, both hip and knee are more flexed than during normal gait, and hamstring muscles might therefore act at a different relative length around hip and knee while having constant geometrical origin-insertion length. Future research to investigate such effects will require new, non-conventional, approaches in experimental and modeling techniques.

This study focused on describing muscle kinematics during crouch gait in healthy subject, in order to explore whether changes at this level could explain crouch gait in children with CP. Factors related to the kinetics of walking are ignored in such an approach. For instance, weakness of the calf muscles is often mentioned as a main cause of crouch gait (Perry, 1992; Chambers, 2001). More comprehensive modeling of human gait, using forward simulation, will be needed to explore factors and their mutual dependency that fully explain crouch gait in CP.

3.5. Conclusions

It can be concluded that walking in crouch does not necessarily coincide with shorter than normal hamstrings length. Moreover, crouch gait does not result in lower muscle-tendon lengthening velocities compared to normal gait. In contrast, decreasing walking speed in crouch has a much larger effect on both muscle-tendon lengths and velocities. Therefore, patients with short or spastic muscles are more likely to respond by walking slower than by walking in crouch. Differences in walking speed should be considered when comparing patient groups with controls. Comprehensive musculoskeletal modeling is required to further investigate possible causes of crouch gait.

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Chapter 4

The effect of walking speed on hamstrings length and lengthening velocity in children with spastic cerebral palsy

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Abstract

Introduction. Children with cerebral palsy often walk with reduced knee extension in terminal swing, which can be associated with short length or slow lengthening velocity of hamstrings muscles during gait. This study investigated the role of two factors that may contribute to such short and slow hamstrings: walking speed and spasticity.

Methods. 17 children with spastic cerebral palsy and 11 matched typically developing children walked at comfortable, slow, and fast walking speed. Semitendinosus muscle-tendon length and velocity during gait were calculated using musculoskeletal modeling. Spasticity of the hamstrings was tested in physical examination.

Results. Peak hamstrings length increased only slightly with walking speed, while peak hamstrings lengthening velocity increased strongly. After controlling for these effects of walking speed, spastic hamstrings acted at considerably shorter length and slower velocity during gait than normal, while non-spastic hamstrings did not.

Interpretation. These data are important as a reference for valid interpretation of hamstrings length and velocity data in gait analyses at different walking speeds. The results indicate that the presence of spasticity is associated with reduced hamstrings length and lengthening velocity during gait, even when controlled for walking speed.
4.1. Introduction

Children with cerebral palsy (CP) often walk with reduced knee extension in terminal swing. This reduction in knee extension leads to a decrease in step length and reduced walking speed, which may limit functional performance. Reduced knee extension in terminal swing is often associated with reduced hamstrings length and lengthening velocity during gait (Delp et al., 1996; Thompson et al., 1998; Arnold et al., 2006a; 2006b). Arnold et al. (2006a) showed that about 35% of subjects walking with reduced knee extension had short and in most cases also slow hamstrings during gait; 30% had slow but not short hamstrings and another 35% had neither short nor slow hamstrings. They also showed that surgically lengthening of muscles that were short or slow, resulted in improved length and velocity during gait and in increased knee extension in terminal swing (Arnold et al., 2006b). This suggests that hamstrings length and velocity during gait are important measures to consider in treatment planning. Moreover, since many of the underlying impairments as well as treatment options lay at muscle rather than joint level, considering muscle-tendon behavior during gait seems to have added value over conventional gait analysis techniques alone that examine joint kinematics and kinetics.

However, the underlying causes of short or slow hamstrings during gait are not well understood. Several factors may cause hamstrings to be short or slow during gait, two of which are walking speed and spasticity. First, children with CP generally walk slower than typically developing children. Walking speed has been shown to influence hamstrings lengths and velocity in healthy subjects walking in crouch (Chapter 3). However, no studies have investigated the effect of walking speed on hamstrings length and velocity in CP patients. Therefore, it is not known to what extent differences in hamstrings length or velocity between patients and control subjects can be attributed to differences in walking speed.

Second, hamstrings spasticity may contribute to short and slow hamstrings during gait. Cheung et al. (2003) compared spastic threshold velocity as measured during physical examination with muscle-tendon lengthening velocity during gait in spastic hamstrings and quadriceps muscle, and found a significant correlation. Jonkers et al. (2006) found that peak rectus femoris length and lengthening velocity were reduced in spastic muscles in stiff knee gait, with peak lengths during gait decreasing with increasing rectus femoris spasticity scores. These studies suggest that muscle-tendon length and velocity during gait are related to spasticity, but evidence is limited.

Moreover, walking speed and spasticity effects may interact, first because the presence of spasticity may limit walking speed, and second because walking speed may influence the effects of spasticity, due to its velocity-dependency (Lance, 1980). Therefore, studying speed effects in spastic children is particularly relevant.
The aims of this study were:

1) to investigate the effect of walking speed on hamstrings length and lengthening velocity during gait in children with CP;

2) to investigate to what extent spasticity as measured in physical examination is related to hamstrings length and lengthening velocity during gait; and

3) to study the interacting effects of walking speed and spasticity.

### 4.2. Methods

#### Subjects

17 children with spastic CP and 11 typically developing (TD) children, matched in age, height, and weight, participated in this study. Characteristics of the children with CP (mean ± SD) were: age 8.9 ± 2.1 years (range 6-12); height 136 ± 13 cm; and weight 33 ± 10 kg; and of the TD children: age 8.2 ± 1.8 years (range 6-12); height 134 ± 12 cm; and weight 32 ± 13 kg. All children with CP were clinically diagnosed with spastic CP (13 bilateral, 4 unilateral), were able to walk independently without walking aids, were classified on the gross motor function classification scale (GMFCS) as level I-II (Palisano et al., 1997), had no prior orthopedic surgery, rhizotomy or baclofen treatment, and had no prior botulinum toxin treatment within the previous 16 weeks. All children and their parents provided informed consent. The study protocol was approved by the Medical Ethics Committee of the VU University Medical Center.

#### Design

The children walked along a 10m walkway, first at self-selected comfortable walking speed (CWS), and subsequently at 70 ± 5% (SLOW) and 130 ± 5% (FAST) of CWS, in random order. Walking speed was recorded online and controlled by giving instant feedback to the children. After sufficient practice attempts, three successful trials were collected for each speed condition. The children were measured twice in two separate sessions, in order to obtain a large and reliable data sample per child, and as part of a larger study. This resulted in a total of six trials per condition. The sessions took place at the same time of day, 17.6 ± 11.6 days apart, without any interventions in between the two sessions. For logistic reasons, two children could be measured only once.

3D kinematic data were collected for the trunk, pelvis, thigh, shank, and foot, using a motion capture system (Optotrak, Northern Digital, Waterloo, Ontario). Data on the right leg were collected in the TD group and on both legs in the CP group. A technical cluster of three markers was attached to each segment. While standing in anatomical position, bony landmarks were indicated in order to anatomically calibrate the technical cluster frames (Cappozzo et al., 2005).
All children with CP underwent a standard physical examination in one of the two sessions, all performed by the same person. Spasticity was measured in the hamstrings with a standardized clinical spasticity test (SPAT) (Scholtes et al., 2007a), which is based on the Modified Tardieu Scale (Boyd and Graham, 1999). In this test muscles were stretched at slow and very fast speed. Based on these measurements, the muscles were grouped according to the level of spasticity: SPAS2 = severe spasticity (presence of a clear catch at fast stretch, SPAT score 2 or 3); SPAS1 = mild spasticity (increase in muscle resistance somewhere in the range of motion at fast stretch, without a catch, SPAT score 1); SPAS0 = no spasticity.

Analysis

3D kinematic data were analyzed with open-source software (www.BodyMech.nl; MatLab®; The Mathworks). Initial contact (IC) was calculated from the forward foot velocity, and defined as the instants at which this velocity became lower than 20% of its maximal value (Chapter 3). One successful stride (IC to IC), for each leg separately, was selected for each trial, and left and right legs were included independently. It is acknowledged that muscle behavior may be influenced by other muscles in the ipsilateral leg, or even by muscles in the contralateral leg. However, since it was not possible to account for any such influence, muscles of all legs were evaluated independently. For one patient, data on only one leg were available for technical reasons, resulting in a total of 33 legs in the CP group and 11 legs (all right legs) in the TD group.

Actual walking speed during the successful stride was calculated as the average forward velocity of the pelvis markers over the full stride, and nondimensionalized by $l_{leg} \cdot \sqrt{g \cdot l_{ref}}$ (Hof, 1996), with $l_{leg}$ the leg length, calculated as the summed length from trochanter major to lateral epicondyle to lateral malleolus. Stride length was calculated as the forward progression of the pelvis markers over the stride and nondimensionalized by $l_{ref}$. Semitendinosus (ST) was considered representative for the hamstrings, and ST length was calculated with SIMM musculoskeletal modeling software (Delp et al., 1990; Delp and Loan, 1995). This model has been validated (Arnold et al., 2001; Chapter 2) and previously used for calculation of hamstrings length in CP (Delp et al., 1996; Thompson et al., 1998; Arnold et al., 2006a; 2006b). The standard generic model was used and scaled to the individual subject sizes, using 3D kinematic data from the anatomical landmarks. Muscle-tendon length was filtered using an 8 Hz low-pass symmetric filter and differentiated in order to obtain muscle-tendon velocity. Both muscle-tendon lengths and velocities were time-normalized to 100% gait cycle, and nondimensionalized by $l_{ref}$ and $\sqrt{g \cdot l_{ref}}$, respectively, with $l_{ref}$ the anatomical reference length with all joint angles set at zero, calculated with SIMM. Peak muscle-tendon length and lengthening velocity, as reached in (terminal) swing, were calculated for all trials.
Statistics

A repeated measures analysis of variance (ANOVA), with Bonferroni adjustment for multiple comparisons, was used to investigate whether walking speed and stride lengths differed between the three speed conditions and between subgroups.

A linear generalized estimating equation (GEE) analysis was applied to investigate the relationship of spasticity and walking speed with the outcome measures peak muscle-tendon length and peak muscle-tendon lengthening velocity (SPSS v15.0.0; exchangeable working correlation structure and robust estimation of the covariance matrix). This analysis accurately controls for differences in walking speed, and estimates the individual contributions of the independent variables to the outcome measures. Three independent variables were included in the model: (1) spasticity group as categorical variable (TD, SPAS0, SPAS1, and SPAS2); (2) walking speed as continuous variable; and (3) the interaction of group and walking speed. Walking speed was centered around the mean nondimensional walking speed of 0.40, by subtracting this value from the measured walking speed. Centering allowed for a meaningful interpretation of main effects when interaction was present in the model (Aiken and West, 1991), in which case the main effect could be interpreted as the effect of spasticity group at nondimensional walking speed of 0.40. This resulted in the following model:

\[
\text{Outcome} = B_0 + B_1 (\text{group}) + B_2 \times (\text{walking speed} - 0.40) + B_3 (\text{group}) \times (\text{walking speed} - 0.40)
\]

with \(B_0\) the value of the outcome measure in TD (reference group), at a walking speed of 0.40; \(B_1\) the difference between the CP groups and TD at a speed of 0.40 (main effect of group); \(B_2\) the slope of the outcome measure versus speed curve for TD (main effect of speed); and \(B_3\) the difference in slope between groups (interaction). Post-hoc analyses were performed with SPAS0 and SPAS1 as reference groups to determine the significance of all pair-wise comparisons between groups. \(P\)-values of less than 0.05 were considered to be statistically significant.

4.3. Results

Based on the physical examination, 6 of the 33 semitendinosus muscles were assigned to SPAS0, 15 to SPAS1 and 12 to SPAS2. Of the ‘sound’ limbs of the unilaterally involved children, 3 muscles were assigned to SPAS0 and 1 to SPAS1.

Nondimensional walking speed differed significantly between the three conditions (\(p<0.001\), Table 4.1). The CP group walked slower than the TD group (\(p<0.001\)); CWS in the CP group was close to SLOW in the TD group (\(p=0.25\)), and FAST in the CP group was close to CWS in the TD group (\(p=0.85\)). Walking speed in SPAS2 was significantly slower than in SPAS0 and SPAS1 (\(p<0.05\)). Stride length was lower in the CP group than in the TD group, and decreased with increasing levels of spasticity (\(p<0.001\), Table 4.1).

Peak ST length and lengthening velocity both increased with walking speed (Figure 4.1 & 4.2, Table 4.2: \(B_2,p_2\)). The effect of walking speed on ST length was small (Figure 4.2A). \(B_5\) which
### Table 4.1: Non-dimensional walking speed and stride length (mean ± SD)

<table>
<thead>
<tr>
<th>Condition</th>
<th>TD</th>
<th>CP-ALL</th>
<th>SPAS0</th>
<th>SPAS1</th>
<th>SPAS2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absolute speed (m/s)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLOW</td>
<td>0.36 ± 0.03 **</td>
<td>0.26 ± 0.06 *</td>
<td>0.30 ± 0.02 *</td>
<td>0.28 ± 0.05 **</td>
<td>0.23 ± 0.06 **</td>
</tr>
<tr>
<td>CWS</td>
<td>0.51 ± 0.04 **</td>
<td>0.39 ± 0.07 **</td>
<td>0.42 ± 0.03 **</td>
<td>0.41 ± 0.06 **</td>
<td>0.35 ± 0.08 **</td>
</tr>
<tr>
<td>FAST</td>
<td>0.66 ± 0.05 ***</td>
<td>0.51 ± 0.07 **</td>
<td>0.56 ± 0.03 **</td>
<td>0.53 ± 0.05 **</td>
<td>0.47 ± 0.08 **</td>
</tr>
<tr>
<td>Relative speed (speed / CWS)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLOW</td>
<td>0.71 ± 0.03</td>
<td>0.68 ± 0.06</td>
<td>0.73 ± 0.02</td>
<td>0.67 ± 0.07</td>
<td>0.67 ± 0.05</td>
</tr>
<tr>
<td>CWS</td>
<td>1.00 ± 0.00</td>
<td>1.00 ± 0.00</td>
<td>1.00 ± 0.00</td>
<td>1.00 ± 0.00</td>
<td>1.00 ± 0.00</td>
</tr>
<tr>
<td>FAST</td>
<td>1.29 ± 0.03</td>
<td>1.34 ± 0.13</td>
<td>1.33 ± 0.10</td>
<td>1.30 ± 0.12</td>
<td>1.37 ± 0.14</td>
</tr>
<tr>
<td>Stride length (cm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLOW</td>
<td>1.60 ± 0.13 **</td>
<td>1.27 ± 0.28 *</td>
<td>1.46 ± 0.12 **</td>
<td>1.32 ± 0.28 **</td>
<td>1.14 ± 0.31 **</td>
</tr>
<tr>
<td>CWS</td>
<td>1.87 ± 0.12 **</td>
<td>1.46 ± 0.25 **</td>
<td>1.61 ± 0.04 **</td>
<td>1.58 ± 0.18 **</td>
<td>1.28 ± 0.31 **</td>
</tr>
<tr>
<td>FAST</td>
<td>2.11 ± 0.13 **</td>
<td>1.68 ± 0.23 **</td>
<td>1.85 ± 0.08 **</td>
<td>1.76 ± 0.12 **</td>
<td>1.39 ± 0.30 **</td>
</tr>
</tbody>
</table>

**TD:** typically developing; **CP-ALL:** all cerebral palsy patients grouped together  
SPAS0, 1 and 2: increasing levels of spasticity in CP; **CWS:** comfortable walking speed  
*and** indicate significant difference (p<0.05) of subgroup to TD (*, **), SPAS0 (**), SPAS1 (**), and SPAS2 (**) respectively. All speed conditions were significantly different from each other (p<0.001)

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![Semitendinosus muscle-tendon length (top row) and velocity (bottom row) versus gait cycle, for the typically developing group (TD; n=11) and the cerebral palsy (CP) groups: SPAS0 (n=6), SPAS1 (n=15), and SPAS2 (n=12), indicating increasing levels of spasticity. CWS: comfortable walking speed. Data at comparable walking speeds (CP CWS and TD SLOW; CP FAST and TD CWS; see Table 1) are plotted together for better comparison.](image-url)

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indicates the slope of the peak length versus walking speed curve, was 0.029 ± 0.012 in the reference group TD. This means that peak ST length increased with 2.9% of reference length for each unit of nondimensional walking speed. Or in more meaningful terms: as walking speed almost doubled from slow (0.36) to fast speed (0.66), peak length increased with approximately 1% of reference length, which came down to about 3.5mm. The effect of walking speed on peak lengthening velocity was more pronounced (Figure 4.2B). B2 was 0.174 ± 0.022 for the TD group. Thus, as walking speed almost doubled from slow to fast speed, peak ST velocity increased with approximately 0.050, or 40%. Peak ST length and lengthening velocity were lower in more spastic muscles (Figure 4.1 & 4.2, Table 4.2: B1, p1). Both SPAS1 and SPAS2 had shorter peak length and slower peak velocity than TD. Non-spastic muscles in CP were not shorter or slower than normal. At the average nondimensional walking speed of 0.40, peak length in TD was 7.6 ± 0.4% longer than reference length (B0). Peak length in SPAS2 was 2.9 ± 0.7% of reference length shorter than in TD (B1). Peak lengthening velocity in SPAS2 was 0.028 ± 0.008 or about 20% lower than normal.

Peak ST velocity showed a significant overall interaction effect of walking speed and spasticity, and a similar trend was seen for peak length (Table 4.2: B3, p3). Peak velocity increased more with walking speed in SPAS0 than in all other groups, but no difference was found between SPAS1 or SPAS2 and TD. Peak length increased more with walking speed in SPAS0 than in SPAS2.

**Figure 4.2:** A Peak semitendinosus (ST) muscle-tendon length and B lengthening velocity versus walking speed, for the typically developing (TD) group and the cerebral palsy groups: SPAS0 (n=6), SPAS1 (n=15), and SPAS2 (n=12), indicating increasing levels of spasticity. Symbols and thin lines represent means and standard deviations of measured data, bold lines represent modeled data from the generalized estimating equation analysis.
This study investigated the role of two factors that may contribute to hamstrings length and velocity during gait, i.e. walking speed and spasticity. When walking speed was reduced, both peak ST length and velocity decreased, with relatively small changes in peak length. Spastic muscles were shorter and slower during gait than non-spastic muscles, even after controlling for walking speed.

We studied the effect of walking speed in CP patients, since it can differ considerably between gait analyses pre- and post treatment, or between subjects, for many reasons. For good interpretation, it is important to understand the separate effects of walking speed on gait parameters. Our result that peak ST length increased only slightly with walking speed indicates that differences in peak length between patients and control subjects, or between pre- and post-treatment analyses mostly reflect deviations in pelvis or leg positioning in terminal swing, or differences in step length, that are not attributable to differences in walking speed per se. As derived from Table 4.3, stride length was indeed lower in the more spastic groups than in TD, even at constant walking speed (e.g. compare SLOW in TD with CWS in CP). This could partly explain the differences in ST length. Contrarily, the strong increase in peak ST velocity with walking speed indicates that differences between gait analyses in hamstrings velocity can to a large extent result from differences in walking speed. This reinforces the result of other studies that walking speed is an important factor to consider when interpreting gait data (e.g. Chapter 3; Stansfield et al., 2001b; Hof et al., 2002; Schwartz et al., 2008).
With our methodology the effects of walking speed could be separated from the effects of spasticity, showing that spastic muscles are shorter and slower than non-spastic muscles, irrespective of walking speed and over a range of speeds. Although no other studies controlled for walking speed in this way, our findings are in line with previous results on the relationship between spasticity and hamstrings length during gait in CP patients (Cheung et al., 2003). These results together indicate that spasticity may be an important contributor to short and slow hamstrings during gait. The close relationship between physical examination spasticity scores and muscle-tendon length and velocity during gait also strengthens the need to study gait at the muscle level, rather than joint and segment level alone, in order to better understand underlying causes of gait deviations in CP.

Of course hamstrings behavior during gait can be influenced by other factors than walking speed and spasticity alone. For example, short passive hamstrings length (contractures) or increased intrinsic muscle stiffness may restrict hamstrings length and velocity during gait (Cooney et al., 2006). Furthermore, muscle weakness, poor selective control or inadequate push-off can affect swing leg behavior, and thereby hamstrings length and velocity during gait (Perry, 1992). The difference between groups may therefore be caused not only by direct effects of spasticity, but also by secondary effects or other related impairments. Based on the concept of spasticity, changes in muscle-tendon velocities directly caused by spasticity are expected to evoke involuntary muscle (reflex-)activation (Crenna, 1998). Future study of electromyographic data will provide more insight into these effects. Moreover, further study is recommended on a large set of patient data, which would allow for multivariate analyses in which more of the possible underlying causes of short and slow hamstrings could be included, as well as further subgroup analyses.

Since spasticity has been defined as a velocity-dependent increase in muscle tone (Lance, 1980), it could be expected that effects of spasticity would increase with higher walking speeds. We did find interaction effects of spasticity and walking speed. First, children with more spastic ST muscles walked slower than children with less spastic muscles (Table 4.1), thereby avoiding high muscle lengthening velocities. Second, length and velocity in non-spastic muscles in CP increased more with walking speed compared with spastic and TD muscles. This may reflect a possible compensation strategy at faster speeds, to compensate for more involved muscles, for example to allow for a larger or faster step at the least affected body side in asymmetric gait. Further investigation of kinematics and detailed musculoskeletal modeling is necessary to study this and other possible compensation strategies to walk with slow and short muscles when faster walking speeds are required or imposed.

Spastic muscle length and velocity tended to increase somewhat less with walking speed than in non-spastic muscles on average, but this effect was small and only significant compared to non-spastic CP muscles. A factor to consider may be that we linearized the relation between peak hamstrings length or velocity and walking speed for the area under consideration. This simplification appeared reasonable, and non-linear approximations did not considerably improve the goodness of fit. However, peak ST velocity in the TD and
SPAS0 groups seemed somewhat non-linear, with lower slope at faster walking speeds. Future study with a broader range of walking speeds could reveal a more complex relationship of muscle-tendon lengths and velocities with walking speed, as has been shown for other kinematic, kinetic and electromyographic variables (Schwartz et al., 2008). This may also reveal more significant interaction effects of walking speed and spasticity.

4.5. Conclusions

This study investigated the separate effects of walking speed and spasticity on hamstrings length and lengthening velocity in children with CP. Peak hamstrings length increased only slightly with walking speed; therefore, differences in peak hamstrings length between patients and control subjects, or between pre- and post-treatment analyses will mostly reflect deviating pelvis or leg positioning in terminal swing, or differences in step length, and can only for a small part be attributed to differences in walking speed per se. Peak hamstrings lengthening velocity increased strongly with walking speed; therefore differences in hamstrings velocity can to a large extent result from differences in walking speed. These data are important as a reference for valid interpretation of hamstrings length and velocity data in gait analyses at different walking speeds. Even when controlled for walking speed, spastic hamstrings were considerably shorter and slower during gait than normal, while non-spastic hamstrings were not.
Chapter 5

Walking speed modifies spasticity effects in gastrocnemius and soleus in cerebral palsy gait

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Caroline A.M. Doorenbosch
Jules G. Becher
Jaap Harlaar
Abstract

Introduction. The calf muscles of children with cerebral palsy are often spastic, which can lead to an equinus gait pattern. Although spasticity is defined as a velocity-dependent increase in muscle tone, very little is known about the effect of walking speed on muscle-tendon behavior of spastic muscles during gait. The aim of this study was to investigate gastrocnemius and soleus length and lengthening velocity during gait in spastic muscles with and without static contractures compared to non-spastic muscles, as well as the effect of walking speed, and the interacting effect of walking speed and spasticity on muscle-tendon length and lengthening velocity.

Methods. 17 ambulatory children with spastic cerebral palsy and 11 typically developing children, aged 6-12, walked at comfortable, slow, and fast walking speeds. 3D kinematic data were collected and muscle-tendon lengths and velocities were calculated using musculoskeletal modeling. Spasticity and contractures of calf muscles were measured during standardized physical examination.

Results. Spastic calf muscles showed a deviating muscle-tendon length pattern with two peaks in stance, which was found to be irrespective of muscle contracture. This deviating pattern became more pronounced as walking speed increased. In swing, spastic calf muscles were stretched approximately one third slower than normal, while in stance, spastic calf muscles were stretched twice as fast as normal, with peak velocity occurring earlier in the gait cycle.

Interpretation. The increasingly deviating muscle-tendon length pattern at faster walking speed indicates a velocity-dependent spasticity effect. This impairs walking especially at faster speeds, and may therefore limit comfortable walking speed.
5.1. Introduction

Spastic paresis is the most common motor disorder in children with cerebral palsy (CP) (Himmelmann et al., 2005), and spasticity is one of the major problems. Spasticity has been defined as a velocity-dependent increase in the tonic stretch reflex (Lance, 1980), and is usually measured according to clinical scales during physical examination (Bohannon and Smith, 1987; Boyd and Graham, 1999). The calf muscles of children with CP are often spastic, which can lead to an equinus (‘toe walking’) gait pattern or early heel rise in stance. With growth, fixed contractures of the calf muscles can also develop, contributing to the equinus gait pattern (Wren et al., 2004). Several other factors, including muscle weakness, poor selective muscle control, or hyper-tonicity that is independent of velocity, may also contribute to an equinus gait pattern. All these factors may interact, and can lead to compensations that are hard to distinguish from the primary impairments.

In order to achieve a better understanding of the effects of calf muscle spasticity and contractures on gait in CP, it can be useful to study gastrocnemius (GM) and soleus (SO) muscle-tendon length during gait. Muscle-tendon behavior is closely related to underlying impairments, such as spasticity or contractures, as well as to possible treatment options, such as surgical lengthening, botulinum toxin treatment or stretching. Equinus gait has been shown to be accompanied by abnormally short GM and SO dynamic muscle lengths (Wren et al., 2004). Several studies have investigated muscle-tendon length of calf muscles during gait to assess the outcomes of botulinum toxin treatment (Eames et al., 1999; Bang et al., 2002) or surgical lengthening of calf muscles (Baddar et al., 2002; Orendurff et al., 2002; Wren et al., 2004). Furthermore, muscle-tendon length during gait has been related to static muscle-tendon length in physical examination, to assess the degree of ‘static’ versus ‘dynamic’ equinus during gait (Eames et al., 1999; Wren et al., 2004).

Since spasticity has been defined as a velocity-dependent increase in muscle tone (Lance, 1980), studying muscle-tendon velocity during gait can also increase our understanding of the effect of spasticity on gait. Stretch velocities can be expected to be reduced in spastic muscles, either due to direct effects of spasticity, or as a compensation strategy to prevent excessive spasticity to occur. For example, rectus femoris lengthening velocity during gait has been shown to be reduced in spastic muscles in stiff knee gait (Jonkers et al., 2006). Similarly, hamstrings peak stretch velocity has been shown to be reduced in most CP patients walking in crouch (Arnold et al., 2006a), and more reduced in hamstrings muscles with higher levels of spasticity (Chapter 4). However, very little is known about gastrocnemius or soleus muscle-tendon velocities during CP gait. Orendurff et al. (2002) showed that both shortening and lengthening velocities were impaired in children with CP, especially around push-off. However, peak values over the entire gait cycle were unchanged after tendon lengthening surgery, and not statistically compared to controls, which warrants further study. Furthermore, no distinction was made between different gait phases, only 12 sides were evaluated, and the data were compared to adult control data only.
These limitations highlight the need for standardized assessment of GM and SO muscle-tendon velocity during gait in spastic muscles, compared to the muscles of matched healthy controls. Moreover, the velocity-dependent effects of spasticity can presumably be modulated by imposing different walking speeds. Spasticity effects are expected to increase at faster walking speed, due to increasingly limiting effects of spasticity. Therefore, investigating the effects of walking speed on muscle-tendon behavior during gait will provide insight into the effect of spasticity on gait in patients with CP.

The aim of this study was to investigate GM and SO length and lengthening velocity during gait in spastic muscles compared to non-spastic muscles, as well as the effect of walking speed, and the interacting effect of walking speed and spasticity on GM and SO length and lengthening velocity during gait. In order to separate the effects of spasticity and contractures, spastic calf muscles of children with CP were classified into contractured and non-contractured muscles, and compared to the calf muscles of a group of matched typically developing (TD) children.

5.2. Methods

Subjects

17 children who were clinically diagnosed with spastic CP (13 diplegic; 4 hemiplegic; 6 boys, 11 girls) and 11 TD children (5 boys, 6 girls) volunteered to take part in this study. The CP and TD groups were matched in age, height, and weight. The mean characteristics of the children in the CP group were: age 8.9 ± 2.1 years (range 6-12); height 136 ± 13 cm; weight 33 ± 10 kg; and of the children in the TD group: age 8.2 ± 1.8 years (range 6-12); height 134 ± 12 cm; and weight 32 ± 13 kg. All children with CP were able to walk independently without walking aids, were classified as level I or II on the gross motor function classification scale (GMFCS) (Palisano et al., 1997), had no history of multilevel surgery, selective dorsal rhizotomy or baclofen treatment, and had received no botulinum toxin A treatment within the previous 16 weeks. All children and their parents provided informed consent. The study protocol was approved by the Medical Ethics Committee of the VU University Medical Center.

Design

The children walked along a 10m walkway at slow, comfortable, and fast walking speeds. First, they walked at self-selected comfortable walking speed (CWS), and subsequently at 70 ± 5% (SLOW) and 130 ± 5% (FAST) of this speed, in random order. Walking speed was recorded online, and controlled by giving instant oral feedback to the children. After sufficient practice attempts, data on a total of six successful trials were collected for each speed condition, divided over two sessions. These two sessions were part of a larger study, and resulted in a large and reliable data sample per child. The sessions took place 17.6 ± 11.6
Gastrocnemius and soleus length and velocity

days apart, at the same time of day, no interventions were performed in between the two sessions, and no systematic differences were present between the two sessions. Two children were measured only once for logistic reasons.

3D kinematic data were collected during the walking trials with a motion capture system (OptoTrak, Northern Digital, Waterloo, Canada). Data on the trunk and pelvis, and the thigh, shank, and foot of the right leg were collected in the TD group, and of both legs in the CP group. A technical cluster of three markers was attached to each segment. Anatomical landmarks were indicated in order to anatomically calibrate the technical cluster frames (Cappozzo et al., 2005).

The CP group underwent a standard physical examination to assess spasticity and static muscle-tendon length of the GM and SO muscles. Spasticity and contractures were assessed with a standardized clinical spasticity test (SPAT) (Scholtes et al., 2007a), which is based on the Modified Tardieu Scale (Boyd and Graham, 1999). In this test the ankle is dorsiflexed at slow and very fast speed, for GM with the knee extended, and for SO with the knee flexed at 90°. Muscles that did not show spasticity in this test were excluded from the analysis.

In order to study the separate effects of spasticity and muscle contracture, all spastic muscles were grouped based on static muscle length. Static muscle length was measured during the slow speed part of the SPAT test, by strongly dorsiflexing the ankle to the end of its range of motion, with the calcaneus in neutral position (no varus/valgus). The ankle angle was measured with standard goniometry. The presence of static contracture was defined as inability to passively dorsiflex the ankle to neutral (0°) or beyond. All spastic CP muscles were assigned either to the non-contractured group (SPAS-NC), or to the contractured group (SPAS-C). All tests were performed by the same researcher.

Analysis

3D kinematic data were analyzed with custom-made software (BodyMech, MatLab®, The Mathworks). Knee and ankle angles were calculated following the CAMARC anatomical frame definitions (Cappozzo et al., 1995). Initial contact (IC) values were calculated from the forward foot velocity, and defined as the moments at which this velocity became lower (IC) or higher (TO) than 20% of its maximal value (Chapter 3). One successful stride (IC to IC) in each trial was selected for each leg separately. Left and right legs were included independently in the analysis. Actual walking speed during the successful stride was calculated as the average forward velocity of the pelvis markers over the full stride, and nondimensionalized by $\sqrt{\frac{g \cdot l_{leg}}{Hof}}$ (Hof, 1996), with $l_{leg}$ the leg length, calculated as the summed length from trochanter major to lateral epicondyle to lateral malleolus.

Muscle-tendon lengths for GM (medial head) and SO were calculated with SIMM musculoskeletal modeling software (Delp et al., 1990; Delp and Loan, 1995). This model has been validated for use in children with CP (Arnold et al., 2001), and has previously been used to calculate GM and SO lengths in children with CP (Wren et al., 2004). In the present study the SIMM standard generic model was used, scaled to the individual subject sizes.
using 3D kinematic data from the anatomical landmarks. Varus-valgus motion of the model’s knee was allowed, and the maximal ankle plantarflexion range was increased to 75° to cover the ankle motion as observed in CP subjects (Wren et al., 2004). Muscle-tendon lengths were low-pass filtered at 8Hz with a symmetric filter, and differentiated in order to obtain muscle-tendon velocities. Both muscle-tendon lengths and velocities were nondimensionalized by \( l_{\text{ref}} \) and \( \sqrt{g \cdot l_{\text{ref}}} \), respectively, with \( l_{\text{ref}} \) the anatomical reference length with all joint angles set at zero, calculated with SIMM.

Muscle-tendon length and velocity curves, as well as knee and ankle angles, were time-normalized to 100% gait cycle and averaged over the six strides for each subject, per speed condition. Next, average curves were calculated over all subjects per group, per speed condition, including standard deviations indicating the difference between subjects. As outcome measures for the statistical analysis, peak values for GM and SO length during the gait cycle were calculated for each selected stride separately, as well as peak lengthening velocity values in stance and in swing.

**Statistics**

A repeated measures analysis of variance (ANOVA) with Bonferroni adjustment for multiple comparisons was applied to investigate differences in walking speed between the three different speed conditions and between CP and TD children.

A linear generalized estimating equation (GEE) analysis was applied to investigate the separate effects of group (TD, SPAS-NC, and SPAS-C), walking speed, and their interaction on the outcome measures: peak muscle-tendon length, peak muscle-tendon lengthening velocity in stance, and peak muscle-tendon lengthening velocity in swing (SPSS v15.0.0; working correlation structure set at exchangeable and robust estimation of the covariance matrix). Walking speed was centered around the mean nondimensional walking speed of 0.40, by subtracting this value from the measured walking speed. Centering allows for a meaningful interpretation of main effects when interaction is present in the model (Aiken and West, 1991). In this way, the main effect of group could be interpreted as the effect at a nondimensional walking speed of 0.40, when interaction effects were present. This resulted in the following model:

\[
\text{Outcome} = B_0 + B_1(\text{group}) + B_2 \times (\text{walking speed} - 0.40) + B_3(\text{group}) \times (\text{walking speed} - 0.40)
\]

with \( B_0 \) the value of the outcome measure in TD, which was used as reference group, at a walking speed of 0.40; \( B_1 \) the difference between the CP groups and TD at a speed of 0.40 (main effect of group); \( B_2 \) the slope of the outcome measure versus speed curve for TD (main effect of speed); and \( B_3 \) the difference in slope between groups (interaction). This regression equation thus estimates the individual contributions of group effect, speed effect, and their interaction to the outcome measures peak length and peak velocity. If the interaction of group and walking speed was not significant, it was excluded from the model and a new
analysis was performed without the interaction term. P-values of less than 0.05 were considered to be statistically significant.

### 5.3. Results

Physical examination revealed spasticity in at least one calf muscle of all children with CP. In three limbs there was no spasticity in GM or SO, in one limb only in GM and in one limb only in SO. All muscles with no spasticity were excluded from the analysis, which yielded a total of 29 SO and 29 GM spastic muscles, in 30 limbs. Of these spastic muscles, 15 SO and 21 GM muscles also had static contractures.

Table 5.1 shows nondimensional walking speeds for the TD and CP subjects. Walking speed differed significantly between the three conditions, as imposed (p<0.001). The CP group walked slower than the TD group (p<0.001). Comfortable walking speed in the CP group was close to slow speed in the TD group (p=0.25). Fast speed in the CP group was close to comfortable walking speed in the TD group (p=0.85). Therefore, data at these comparable walking speeds will be plotted together for better comparison.

As a reference, Figure 5.1 shows knee and ankle angles for the CP subjects at comfortable walking speed, together with the angles of the TD subjects at (comparable) slow speed. The CP subjects showed limited knee extension in terminal swing and loading response, with variable alignment of the knee in mid-stance. The ankle joints showed increased plantar flexion in stance and swing, more so in the subjects with static contractures.

<table>
<thead>
<tr>
<th>Condition</th>
<th>TD</th>
<th>CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>SLOW</td>
<td>0.36 ± 0.03</td>
<td>0.26 ± 0.06</td>
</tr>
<tr>
<td>CWs</td>
<td>0.51 ± 0.04</td>
<td>0.39 ± 0.07</td>
</tr>
<tr>
<td>FAST</td>
<td>0.66 ± 0.05</td>
<td>0.51 ± 0.07</td>
</tr>
</tbody>
</table>

TD: typically developing; CP: cerebral palsy
CWs: comfortable walking speed
All speed conditions were significantly different (p<0.001)
CP subjects walked slower than TD subjects (p<0.001)
Figure 5.2 shows muscle-tendon length for GM and SO muscles as a function of the gait cycle, for all speed conditions. Muscle-tendon length in spastic GM and SO muscles was shorter during almost the entire gait cycle compared to the TD group, with SPAS-C muscles being shorter than SPAS-NC muscles. Peak GM and SO lengths are shown in Figure 5.3A,C.

In SPAS-C, peak SO length was approximately 5% of reference length shorter than in TD, and peak GM length was approximately 3% shorter (Figure 5.3A,C; Table 5.2: B1).

The pattern of muscle-tendon length over the gait cycle was different in all spastic muscles compared to TD (Figure 5.2). At comfortable walking speed, muscle-tendon length in spastic muscles showed two peaks, one in early stance and one in terminal stance, while in TD muscle-tendon length increased gradually up until terminal stance (Figure 5.2, second column). To quantify this difference in muscle-tendon length pattern, we calculated the ratio between the peak length as reached during the first half of stance (0-30%GC) and the peak length as reached during the second half of stance (31-60%GC). Thus, a ratio > 1 indicates that the first peak is larger than the second peak. As shown in Figure 5.3B,D and Table 5.2: B1, this ratio was larger in spastic muscles compared to TD and similar in SPAS-NC and SPAS-C muscles.

With increasing walking speed, this deviating double peak pattern became more pronounced. Specifically, the first peak increased with increasing walking speed, while the second peak decreased, leading to an increase in peak length ratio (Figure 5.3B,D; Table 5.2: B2). The effect was most pronounced in the SO muscles, where the deviating pattern increased more with walking speed in spastic muscles compared to TD, and similarly in SPAS-C and SPAS-NC muscles. Despite this change in muscle-tendon length pattern, overall peak length in TD and CP muscles hardly changed at all with walking speed (Figure 5.3A,C; Table 5.2: B2,B3).
Figure 5.2: Muscle-tendon lengths for gastrocnemius (top row) and soleus (bottom row). Muscle-tendon lengths normalized to reference length are shown as a function of the gait cycle for the three walking speed conditions. Lines represent the mean ± SD of all subjects in the corresponding group. Data at matched walking speed (CP CWS and TD SLOW; CP FAST and TD CWS) are plotted together for better comparison. CWS: comfortable walking speed. SPAS-NC: spastic CP muscles with no contracture (peak ankle dorsiflexion in physical examination > 0°; GM: n=8; SO: n=14); SPAS-C: spastic CP muscles with contracture (peak ankle dorsiflexion ≤ 0°; GM: n=21; SO: n=15). Note that contractured muscles act at shorter length than non-contractured muscles, but show the same pattern and change in pattern with walking speed.

Figure 5.3: A,C Peak muscle-tendon length and B,D peak length ratio as a function of nondimensional walking speed for gastrocnemius (top row) and soleus (bottom row). Peak length ratio: peak length in the first half of stance divided by peak length in the second half of stance. TD: typically developing (n=11); SPAS-NC: spastic CP muscles with no contracture (GM: n=8; SO: n=14); SPAS-C: spastic CP muscles with contracture (GM: n=21; SO: n=15). Symbols and thin lines represent means and standard deviations of measured data, bold lines represent modeled data from the generalized estimating equation analysis (Table 5.2). Note that spastic muscles have shorter peak length than TD muscles, and higher peak length ratio, indicating a deviating pattern.
Chapter 5

Figure 5.4 shows muscle-tendon velocities as a function of the gait cycle, for all walking speed conditions. Muscle-tendon velocities also showed a deviating pattern in spastic muscles compared to TD. Two stretch-velocity peaks were distinguished: one in stance and one in swing. Peak GM and SO stretch velocity in stance and swing are shown in Figure 5.5. In stance, peak stretch velocity in spastic muscles was approximately twice as fast as in TD (Figure 5.5A,C; Table 5.2: B), and occurred earlier in the gait cycle. As walking speed increased, peak stretch velocity in stance increased (Table 5.2: B), equally in all groups (Table 5.2: B).

Table 5.2: GEE results showing the effects of group, walking speed, and their interaction, as shown in Figure 5.3 and 5.5

<table>
<thead>
<tr>
<th>Outcome measure</th>
<th>Group</th>
<th>Intercept at v=0.40</th>
<th>Group effect</th>
<th>Speed effect</th>
<th>Group x speed effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>b0 ± SE</td>
<td>b1 ± SE</td>
<td>b2 ± SE</td>
<td>p1</td>
</tr>
<tr>
<td>Peak length ±</td>
<td>TO</td>
<td>1.012 ± 0.002</td>
<td>0 ± 0.000</td>
<td>-0.017 ± 0.000</td>
<td>0.007</td>
</tr>
<tr>
<td></td>
<td>SPAS-NC</td>
<td>id.</td>
<td>-0.020 ± 0.005</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>-0.029 ± 0.005</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak length ratio ±</td>
<td>TO</td>
<td>0.988 ± 0.002</td>
<td>0 ± 0.000</td>
<td>0.025 ± 0.005</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>SPAS-NC</td>
<td>id.</td>
<td>0.011 ± 0.003</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>0.015 ± 0.003</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak velocity in stance ±</td>
<td>TO</td>
<td>0.040 ± 0.002</td>
<td>0 ± 0.000</td>
<td>0.077 ± 0.009</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>SPAS-NC</td>
<td>id.</td>
<td>0.034 ± 0.010</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>0.037 ± 0.005</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak velocity in swing ±</td>
<td>TO</td>
<td>0.066 ± 0.004</td>
<td>0 ± 0.000</td>
<td>0.074 ± 0.010</td>
<td>0.000</td>
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<td>SPAS-NC</td>
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<td>-0.016 ± 0.007</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>-0.023 ± 0.005</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak length ±</td>
<td>TO</td>
<td>0.016 ± 0.003</td>
<td>0 ± 0.000</td>
<td>-0.006 ± 0.008</td>
<td>0.490</td>
</tr>
<tr>
<td></td>
<td>SPAS-NC</td>
<td>id.</td>
<td>-0.015 ± 0.004</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>-0.047 ± 0.007</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak length ratio ±</td>
<td>TO</td>
<td>0.979 ± 0.003</td>
<td>0 ± 0.000</td>
<td>0.054 ± 0.006</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>SPAS-NC</td>
<td>id.</td>
<td>0.029 ± 0.005</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>0.036 ± 0.005</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak velocity in stance ±</td>
<td>TO</td>
<td>0.049 ± 0.004</td>
<td>0 ± 0.000</td>
<td>0.130 ± 0.011</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>SPAS-NC</td>
<td>id.</td>
<td>0.054 ± 0.009</td>
<td>id.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
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<td>id.</td>
<td></td>
</tr>
<tr>
<td>Peak velocity in swing ±</td>
<td>TO</td>
<td>0.094 ± 0.007</td>
<td>0 ± 0.002</td>
<td>0.040 ± 0.012</td>
<td>0.000</td>
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<tr>
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<td>id.</td>
<td></td>
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<tr>
<td></td>
<td>SPAS-C</td>
<td>id.</td>
<td>-0.031 ± 0.009</td>
<td>id.</td>
<td></td>
</tr>
</tbody>
</table>

GEE: generalized estimating equations; according to: Outcome = b0 + b1(group) + b2(speed-0.40) + b3(group)x(speed-0.40)

E.g. GM peak length in SPAS-C at nondimensional speed of 0.50 is equal to: 1.012 - 0.029 × 0.50 + 0.015 × 0.50 = 0.910

P-values indicate significance of main effects of group (p1), walking speed (p2), and group x walking speed (p3).

If interaction was not significant (p3), it was excluded from the model. In this case the effect of walking speed was equal in all groups, and the main effect of group was valid over the full speed range

1 Peak length normalized to reference length
2 Peak length reached in first half of stance (0-30%GC) divided by peak length reached in second half of stance (31-60%GC)
3 Peak stretch velocity nondimensionalized to v(s)/v(p)
4 B0 and B1 values are identical (id.) in all groups; the difference between groups is indicated by B2 and B3

Set to zero because the typically developing (TD) group was used as reference group

SPAS-NC: spastic CP muscles with no contracture (peak ankle dorsiflexion > 0°); GM: n=8; SO: n=14
SPAS-C: spastic CP muscles with contracture (peak ankle dorsiflexion ≤ 0°); GM: n=21; SO: n=15

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Figure 5.4: Muscle-tendon velocities for gastrocnemius (top row) and soleus (bottom row). Muscle-tendon velocities are shown as a function of the gait cycle for the three walking speed conditions. Lines represent the mean ± SD of all subjects in the corresponding group. Data at matched walking speed (CP CWS and TD SLOW; CP FAST and TD CWS) are plotted together for better comparison. CWS: comfortable walking speed. SPAS-NC: spastic CP muscles with no contracture (GM: n=8; SO: n=14); SPAS-C: spastic CP muscles with contracture (GM: n=21; SO: n=15). Note that all spastic muscles show a high stretch velocity in early stance, and low stretch velocity in swing.

Figure 5.5: Peak muscle-tendon stretch velocities in A,C stance and B,D swing as a function of nondimensional walking speed for gastrocnemius (top row) and soleus (bottom row). TD: typically developing (n=11); SPAS-NC: spastic CP muscles with no contracture (GM: n=8; SO: n=14); SPAS-C: spastic CP muscles with contracture (GM: n=21; SO: n=15). Symbols and thin lines represent means and standard deviations of measured data, bold lines represent modeled data from the generalized estimating equation analysis (Table 5.2). Note that spastic muscles show faster peak velocity in stance and slower peak velocity in swing compared to TD.
In swing, spastic muscles were stretched approximately one third slower than muscles in TD subjects (Figure 5.5B,D; Table 5.2: B1), with similar peak velocities in SPAS-C and SPAS-NC muscles. With increasing walking speed, the peak stretch velocity in swing increased (Table 5.2: B2), equally in all groups (Table 5.2: B3).

5.4. Discussion

This study investigated GM and SO length and lengthening velocity during gait in spastic calf muscles of children with CP, as well as the effect of walking speed and the interacting effect of walking speed and spasticity on muscle-tendon length and lengthening velocity. Since spasticity has been defined as a velocity-dependent increase in muscle tone (Lance, 1980), increasing walking speed was hypothesized to enhance the effects of spasticity. It was found that differences in muscle-tendon lengths pattern between spastic muscles and muscles in TD subjects did indeed increase with increasing walking speed, irrespective of muscle contractures. Furthermore, spastic muscles were slower than normal in swing, and faster in stance.

The muscle-tendon length and velocity profiles found in this study were comparable to those reported in previous studies (Baddar et al., 2002; Bang et al., 2002; Orendurff et al., 2002; Wren et al., 2004), despite differences in modeling techniques used. Wren et al. (2004) found no difference in dynamic muscle-tendon length between contractured and non-contractured muscles, whereas in our study we found shorter peak length in contractured muscles (Figure 5.2 & 5.3). This may be due to the small sample size in the Wren et al. study (n=4 in the contractured group).

In the present study, muscle-tendon lengths showed a double peak pattern in spastic muscles, unlike non-spastic muscles, which became more pronounced at faster walking speed. As can be seen in Figure 5.2, the effects of spasticity and of increasing speed were similar. The muscle-tendon length pattern of contractured and non-contractured spastic muscles was remarkably similar, as was the change in pattern with increasing walking speed. This indicates that this pattern is likely caused by velocity-dependent spasticity effects and not by muscle contractures.

Interestingly, muscles in TD subjects also showed a shift towards a double peak pattern at fast walking speed (Figure 5.2 & 5.3). This finding may reflect a velocity-dependent effect in all SO and GM muscles, with stretch reflex activity counteracting elongation of the muscle. Stretch reflex activity is indeed thought to play an important role in calf muscle activation during normal walking, and has been found to have higher amplitudes at fast walking than at slow walking (Sinkjaer et al., 1996; 2000). This stretch reflex activity is thought to be enhanced in spastic muscles, which is in line with our finding that the double peak pattern is increased in spastic muscles compared to TD and at faster walking speeds.

Spastic GM and SO muscles were approximately one third slower in swing compared to TD muscles at comparable walking speed. This coincided with decreased dorsiflexion velocity of
the ankle and (for the GM) with reduced knee extension velocity in swing (Figure 5.1). The slow peak velocities in swing could be caused by evoked muscle activity, resulting in muscle contraction which, due to the small mass of the foot and lower leg, inhibited further elongation of the muscle. The slow stretch could also be a compensation strategy to prevent the occurrence of excessive muscle activity in swing. However, other factors such as increased passive stiffness of calf muscles could also play a role. Furthermore, weakness of the dorsiflexor muscles could add to the decreased stretch of the calf muscles in swing.

Contrary to our hypothesis, the difference in peak stretch velocity in swing between spastic and TD muscles did not increase with faster walking speed (Figure 5.5B,D), which may indicate that other factors than spasticity alone may be responsible for the slow stretch in swing. However, the latency of the stretch reflex (with shortest reflex loops of about 40 ms (Sinkjaer et al., 1996) may also allow a fast stretch in swing before excessive muscle activation occurs and limits further stretch. Further study of muscle activation patterns is necessary to investigate these effects.

In stance, spastic GM and SO were stretched approximately twice as fast as normal. This may be a result of the more equine position of the foot at foot contact, resulting from the slow stretch velocities in swing. In stance, the load of the body will stretch calf muscles, despite involuntary contractions as a result of spasticity. The muscle activation that is, presumably, evoked by this high early stretch velocity will result in muscle contraction to support the weight of the body in an equine foot position during stance. Nevertheless, this involuntary activation causes premature contraction of the calf muscles in stance, leading to shorter and slower than normal calf muscles in the second half of stance, and to the deviating muscle-tendon length pattern (Figure 5.2 & 5.4).

The fast peak stretch velocity of spastic muscles in early stance was higher than the peak stretch velocity in swing (Figure 5.5A,C). Thus, spastic muscles showed their overall fastest stretch velocity peak in early stance. Contrarily, muscles in TD showed their fastest stretch velocity peak in swing. When evaluating (peak) muscle tendon lengths and velocities it is thus of importance to distinguish between these different gait phases.

Our finding that spasticity effects are enhanced at faster walking speed has several clinical implications. First, preferred walking speed can be reduced in children with spastic calf muscles to limit the effects of spasticity. Since SO length and ankle angle are closely related, the effects of walking speed on muscle-tendon length will be reflected in foot and ankle kinematics. Thus, an equinus gait pattern will also become more severe at faster walking speeds. Walking slowly can therefore be a way to ease the severity of the equine position during gait. Second, our results emphasize the need to account for variations in walking speed when comparing gait data between subjects, or between pre- and post intervention gait analyses. For example, if a patient walks in a more severe equinus position after treatment, and walks faster, the increase in equinus may reflect the effect of walking speed rather than a deterioration in the gait pattern.

The speed-dependent effects found in this study need not solely be attributed to spasticity. For example, limited selective control could also play a role in CP, especially at faster
walking speeds, with more synergistic muscle activation patterns causing the plantar flexor muscles to be activated earlier in the gait cycle, in co-activation with quadriceps muscle. Further study of muscle activation patterns may elucidate some of these effects. Comprehensive dynamic modeling of human gait, including forward simulations, will be necessary to further investigate the effects of spasticity on gait.

5.5. Conclusions

- Spastic GM and SO muscles in CP subjects showed a muscle-tendon length pattern during gait with two peaks in stance, which differed from muscles in TD subjects at comparable walking speed. Although peak length was lower in contractured muscles, this deviating pattern was similar in spastic muscles with and without contractures, and became more pronounced as walking speed increased. The same pattern occurred in muscles of TD subjects at fast walking speed.

- In swing, spastic muscles were stretched approximately one third slower than muscles in TD subjects for the same walking speed. Peak stretch velocity increased with speed equally in all groups, which was not in line with the notion of spasticity.

- In stance, spastic GM and SO muscles were stretched approximately twice as fast as muscles in TD subjects, and this peak velocity occurred earlier in the gait cycle. This fast stretch presumably evoked excessive muscle activity, leading to shorter and slower muscles in the second half of stance, as reflected by the deviating muscle-tendon length pattern.

- The walking speed-dependent effect on muscle-tendon length pattern in stance indicates a velocity-dependent spasticity component. This impairs walking especially at faster speeds, which affects the gait pattern and may limit comfortable walking speed.
Chapter 6

Dynamic spasticity of plantar flexor muscles in cerebral palsy gait

Submitted
Marjolein M. van der Krogt
Caroline A.M. Doorenbosch
Jules G. Becher
Jaap Harlaar
Abstract

Introduction. The purpose of this study was to quantify dynamic spasticity, i.e. the coupling between muscle-tendon stretch velocity and muscle activity during gait, of the gastrocnemius and soleus muscles in children with spastic cerebral palsy.

Methods. 17 ambulatory children with cerebral palsy with spastic calf muscles, and 11 matched typically developing children. The children walked at three different speeds. 3D kinematic and electromyographic data were collected. Muscle-tendon velocities of gastrocnemius medialis and soleus were calculated using musculoskeletal modeling.

Results. In typically developing children, muscles were stretched fast in swing without subsequent muscle activity, while spastic muscles were stretched slower for the same walking speed, followed by an increase in muscle activity. The average ratio between peak activity and peak stretch velocity in swing was approximately four times higher in spastic muscles, and increased with walking speed. In stance, the stretch of muscles in typically developing children was followed by an increase in muscle activity. Spastic muscles were stretched fast in loading response, but since muscle activity was already built up in swing, no clear dynamic spasticity effect was present.

Interpretation. Spastic calf muscles showed an increased coupling between muscle-tendon stretch velocity and muscle activity especially during the swing phase of gait.
6.1. Introduction

Spastic paresis is the most common motor disorder in children with cerebral palsy (CP), accounting for 85% of all children with CP (McManus et al., 2006). In this group of children, spasticity is one of the main symptoms of disturbed muscle function. Although different definitions of spasticity exist throughout the literature, the most commonly used definition is that of Lance (1980), stating that spasticity is a velocity-dependent increase in muscle tone, resulting from hyperexcitability of the stretch reflex. Spasticity is thought to lead to gait deviations, and in the long term to muscle contractures and bone deformities. In clinical practice, spasticity is measured in physical examination using passive muscle tests such as the (Modified) Ashworth Scale or the (Modified) Tardieu Scale (Scholtes et al., 2006).

For patient care, it is of particular importance to understand the effect of spasticity not only during passive muscle testing, but also during functional tasks such as gait. Determining the effect of spasticity on gait is also essential for accurate treatment planning and evaluation. However, due to the interplay with other impairments in CP, such as muscle contractures, weakness, bony deformities, and diminished selective motor control, it is difficult to determine the precise effect of spasticity on gait. Therefore, little is known about the clinical significance of spasticity during functional tasks such as gait (Lin, 2004).

Insight into the effect of spasticity on gait can be gained by studying the relationship between spasticity measured during physical examination and gait parameters (e.g. Chapter 4; Tuzson et al., 2003; Damiano et al., 2006; Jonkers et al., 2006). These studies yielded ambiguous results, possibly due to the different measures of spasticity used, and the fact that gait parameters are often assessed at joint level rather than muscle level. Moreover, the expression of spasticity during dynamic tasks such as walking may differ from that during passive tests in physical examination (Neilson and Andrews, 1973; Knutsson and Martensson, 1980; Crenna, 1998).

Spasticity can also be assessed directly during gait, by studying the relationship between muscle stretch velocity and muscle activity. This concept was introduced by Crenna et al. (1992; 1998) and termed dynamic spasticity. Crenna (1998) found that in children with CP this coupling between muscle lengthening velocity and muscle activity is often increased compared to normal. The increased coupling could be present either in terms of a decreased ‘threshold’ (velocity at which muscle activity is evoked) or increased ‘gain’ (change in muscle activity relative to change in stretch). However, Crenna (1998) also discusses that differences exist in the expression of spasticity during gait, between gait phases and between muscles.

However, dynamic spasticity of the calf muscles has never been systematically studied in a group of children with CP. Therefore, the aim of this study was to explore the relationship between muscle-tendon stretch velocity and muscle activity of gastrocnemius and soleus muscles, in a group of children with CP with spastic calf muscles. It was hypothesized that in spastic muscles the coupling between stretch velocity and muscle activity is increased, and that spastic muscles will not stretch fast without concomitant excessive muscle activity.
6.2. Methods

Subjects
17 children with spastic CP and 11 typically developing (TD) children, matched in age, height, and weight, participated in this study. The children with CP were aged 8.9 ± 2.1 years (range 6-12); height 136 ± 13 cm; and weight 33 ± 10 kg (mean ± SD). The TD children were aged 8.2 ± 1.8 years (range 6-12); height 134 ± 12 cm; and weight 32 ± 13 kg. All children with CP were clinically diagnosed with spastic CP (13 bilateral, 4 unilateral), were able to walk independently without walking aids, were classified on the gross motor function classification scale (GMFCS) as level I-II (Palisano et al., 1997), had no prior orthopedic surgery, rhizotomy or baclofen treatment, and had no prior botulinum toxin treatment within the previous 16 weeks. All children showed spasticity in the calf muscles of their affected legs, as measured by a standard physical examination (Schottes et al., 2007a), except for one leg, which was excluded. All affected legs showed an equinus gait pattern or an early heel rise (higher than normal plantar flexion of the foot at mid stance) at comfortable and/or fast walking speed. All children and their parents provided informed consent. The study protocol was approved by the Medical Ethics Committee of the VU University Medical Center.

Design
The children underwent a standard clinical gait analysis, at three different walking speeds. They all walked at self-selected comfortable walking speed (CWS), followed by SLOW (70 ± 5% of CWS) and FAST (130 ± 5% of CWS) speed, in random order. Walking speed was varied in order to be able to control for differences in walking speed between patients and controls, and in order to modulate the velocity-dependent effect of spasticity. Walking speed was recorded online and controlled by giving instant feedback to the children. Six successful trials were collected for each speed condition, divided over two separate sessions. The two sessions were part of a larger study, and took place 17.6 ± 11.6 days apart, at the same time of day. There were no interventions in between the two sessions, and data from both sessions were included in the analysis. For logistic reasons, two children could be measured only once.

3D kinematic data were collected during the walking trials using a motion capture system (Optotrak, Northern Digital, Waterloo, Ontario) for the trunk, pelvis, upper and lower legs and feet. The movement of each segment was tracked using technical clusters of three markers, which were anatomically calibrated using virtual anatomical markers (Cappozzo et al., 2005).

Electromyographic (EMG) data were collected for the gastrocnemius and soleus muscles (Noraxon Telemyo). Surface electrodes were placed according to the SENIAM guidelines (Freriks et al., 1999). EMG data were collected at 1000 Hz and online high pass filtered at 20 Hz to remove artifacts.
### Analysis

3D kinematic data were analyzed with open source Matlab® software (www.BodyMech.nl). Initial contact (IC) and toe-off (TO) values were calculated from the forward foot velocity, and defined as the moments at which this velocity became lower (IC) or higher (TO) than 20% of its maximal value (Chapter 3). From each trial, one successful stride (IC to IC) was selected, for both the left and the right leg for the CP subjects; and for the right leg only for the TD subjects. For one patient, data on only one leg were available for technical reasons, resulting in a total of 28 affected legs in the CP group and 11 legs in the TD group.

Actual walking speed during the successful strides was calculated as the average forward velocity of the pelvis markers over the full stride, and nondimensionalized by $\sqrt{g \cdot \text{leg}}$ (Hof, 1996), with $\text{leg}$ the leg length, calculated as the summed length from trochanter major to lateral epicondyle to lateral malleolus.

Muscle-tendon lengths of gastrocnemius medialis (GM) and soleus (SO) were calculated with SIMM musculoskeletal modeling software (Delp et al., 1990; Delp and Loan, 1995). The SIMM standard generic model was used and scaled to the individual subject sizes, using 3D kinematic data from the anatomical landmarks. Varus-valgus motion of the model’s knee was allowed, and the maximal ankle plantarflexion range was increased to 75° to allow the ankle motion as observed in CP subjects (Wren et al., 2004).

Muscle-tendon lengths were low-pass filtered using an 8 Hz low-pass symmetric filter, and differentiated, in order to obtain muscle-tendon velocities. Muscle-tendon velocities were nondimensionalized by $\sqrt{g \cdot \text{ref}}$, with $\text{ref}$ the anatomical reference length with all joint angles set at zero, as calculated with SIMM. EMG signals were rectified and low-pass, symmetrically filtered at 5 Hz. EMG was normalized to the peak value during the stride at CWS for CP; and to the peak value at SLOW speed for TD, which turned out to be similar absolute speeds (see results). Muscle-tendon velocities and EMG data were time-normalized to 100% gait cycle.

To evaluate dynamic spasticity, the coupling between muscle-tendon stretch velocity and muscle activity was compared between the affected CP limbs (CP; n=28) and the control limbs of the TD subjects (TD; n=11). First, dynamic spasticity was assessed qualitatively, by simultaneously plotting the time series of EMG and stretch velocity averaged over all CP and TD subjects, and by plotting EMG versus stretch velocity, for the stance and swing phase separately.

Second, dynamic spasticity was assessed quantitatively, for those gait phases where the muscles were stretched and muscle activity should normally be absent, which is the case in the swing phase (Perry, 1992). For this phase we calculated:

1. The peak stretch velocity;
2. The peak EMG following this stretch, i.e. the peak EMG as built up in swing that occurred in the time period from onset of stretch plus 40 ms (as an estimate for the
shortest possible stretch-reflex delay; based on Sinkjaer et al. (1996)) until the end of swing;

3. EMG-velocity ratio, i.e. the ratio between peak EMG and peak stretch velocity. This ratio was calculated as a measure for dynamic spasticity, representing increased EMG activity for a certain muscle-tendon stretch velocity;

4. The absolute time-delay in milliseconds between peak stretch velocity and peak EMG.

An analyses of variance (ANOVA) for repeated measures, with Bonferroni adjustment for multiple comparisons was used to test the effects of group (CP and TD) and speed condition (SLOW, CWS, FAST) on actual achieved nondimensional walking speed, peak stretch velocity in swing, peak EMG following stretch in swing, and EMG-velocity ratio. A student’s t-test was used to compare comfortable walking speed in CP with slow walking speed in TD; as well as fast walking speed in CP with comfortable walking speed in TD.

6.3. Results

Table 6.1 shows nondimensional walking speeds for the CP and TD groups. The CP subjects walked significantly slower than the TD subjects ($p<0.001$). Comfortable walking speed in CP was similar to slow speed in TD ($p=0.25$), and fast speed in CP was close to comfortable walking speed in TD ($p=0.85$). Thus, to eliminate the effect of absolute differences in walking speed, comparisons between the two groups were made at comparable walking speeds, i.e. CWS in CP and SLOW in TD; and FAST in CP and CWS in TD.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Condition</th>
<th>CP</th>
<th>TD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nondimensional walking speed</td>
<td>SLOW</td>
<td>0.26 ± 0.06</td>
<td>0.36 ± 0.03</td>
</tr>
<tr>
<td></td>
<td>CWS</td>
<td>0.39 ± 0.07</td>
<td>0.51 ± 0.04</td>
</tr>
<tr>
<td></td>
<td>FAST</td>
<td>0.51 ± 0.07</td>
<td>0.66 ± 0.05</td>
</tr>
<tr>
<td>Stride time (s)</td>
<td>SLOW</td>
<td>1.27 ± 0.21</td>
<td>1.11 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>CWS</td>
<td>0.97 ± 0.10</td>
<td>0.92 ± 0.08</td>
</tr>
<tr>
<td></td>
<td>FAST</td>
<td>0.84 ± 0.08</td>
<td>0.81 ± 0.08</td>
</tr>
</tbody>
</table>

TD: typically developing; CP: cerebral palsy; CWS: comfortable walking speed
Walking speed increased with speed condition (as imposed; $p<0.001$), and was lower in CP than in TD ($p<0.001$). Stride time decreased with speed condition ($p<0.001$) and was lower in CP than in TD for the same absolute walking speed ($p<0.001$)

Figure 6.1 shows both the muscle-tendon stretch velocity and the EMG activity for the gastrocnemius (mean ± SD), for all speed conditions, for CP and TD. Since the main effects occurred around initial contact, the horizontal scale in Figure 6.1 runs from 0 to 150% gait cycle. At matched walking speed (i.e. compare Figure 6.1B with D, or C with E), the GM velocity pattern in spastic muscles differed from the velocity pattern in TD subjects. In CP,
Dynamic spasticity

muscles showed a fast peak stretch velocity in early stance and a slower stretch in swing. In TD, GM showed the fastest peak in swing and a slower stretch in mid-stance.

When simultaneously examining the EMG and velocity patterns, the most notable effect shown in Figure 6.1 is that in TD subjects, GM stretched fast in swing without a subsequent increase in muscle activity. Contrarily, in CP a slower stretch of spastic muscles was seen in swing, followed after a short delay by an increase in muscle activity. In stance, a totally different pattern occurred. Here the stretch phase in TD subjects was followed almost instantly by an increase in muscle activity. In CP, the fast stretch in early stance coincided with a peak in EMG activity, but was not followed by a clear additional increase in EMG activity following stretch.

Figure 6.2 shows the same graphs for the soleus. A similar pattern was seen in SO as in GM: in TD a fast stretch occurred in swing without concomitant muscle activity, while in CP the stretch was much slower, and followed, with some delay, by an increase in muscle activity. In stance, the stretch in TD was followed by muscle activity, while in CP a fast stretch was seen in early stance, coinciding with but not followed by additional muscle activity.

Figure 6.3 shows loops of the muscle activity versus muscle-tendon stretch, as proposed by Crenna (1998). Graphs are shown for GM, for the matched CP CWS and TD SLOW speeds. The graphs are separated into stance and swing stretch phases. CP muscles in swing showed a decreased stretch velocity and an increased muscle activity compared to TD muscles in swing, which show high velocity without concomitant muscle activity. TD muscles showed a similar loop in stance, while in spastic muscles activity was already built up during swing, and no loop was present in stance.

Peak stretch velocity and peak EMG as built up in swing (to exclude possible effects of the fast stretch in stance), as well as their ratio are shown in Figure 6.4 for both GM and SO. At matched speed, spastic muscles were approximately one third slower in swing than muscles in TD. Also, spastic muscles showed about three times higher EMG activity in swing compared to muscles in TD. This resulted in a ratio between peak EMG and peak stretch velocity in swing that was approximately four times higher in CP than in TD.

To get an indication of the time-delay between stretch velocity and EMG, delays were calculated between peak stretch velocity and peak EMG as built up in swing, and Table 6.1 gives absolute stride times as a reference for Figure 6.1 & 6.2. The average delay between peak stretch velocity and peak EMG in swing over all trials was 155 ± 80 ms for GM, and 236 ± 64 ms for SO, and did not differ between groups (GM: p=0.82; SO: p=0.36).

With increasing walking speed, both peak stretch velocity and peak EMG in swing increased, for both groups (Figure 6.4A-D; p<0.001 for all). The ratio between the peak EMG and peak stretch also increased with walking speed (Figure 6.4E-F; p=0.015 for GM and p=0.025 for SO). The delay between peak stretch and peak EMG was constant with speed for GM (p=0.40), while it slightly decreased in SO from 265 ± 67 ms at slow speed to 209 ± 46 ms at fast speed (p<0.001).
6.4. Discussion

In this study we investigated dynamic spasticity, i.e. an increased coupling between muscle-tendon stretch velocity and muscle activity during gait, of spastic calf muscles in children with CP. It was found that spastic muscles showed an increased coupling between stretch and activity in swing. In TD subjects, muscles were stretched fast in swing without subsequent muscle activity, while in CP subjects muscles were stretched slower, followed by an increase in muscle activity. The ratio between peak EMG and peak stretch was four times higher in spastic CP muscles on average, and increased with walking speed.

The results as found in swing are in line with the concept of spasticity: a velocity-dependent increase in muscle activity. Both a decreased stretch velocity and an increased muscle activity were observed in spastic muscles. These two can be logically related: more dynamically spastic muscles show an increased activity already at low stretch velocity, which slows down the movement and results in low peak velocity. Although most of the literature on spasticity effects during gait in calf muscle focuses on the stance phase (e.g. Crenna, 1998; Lamontagne et al., 2001), our results show the clearest effect of dynamic spasticity already in swing.

Our findings are also in line with literature on stretch reflex activity during gait. Hyperexcitability of the stretch reflex is assumed to be one of the main underlying causes of spasticity (Lance, 1980). Several studies have investigated the strength and modulation of the stretch reflex during normal and CP gait. Sinkjaer et al. (1996) tested the stretch reflex during gait by mechanically perturbing the soleus length. They showed that in normal walking, the amplitude of the soleus stretch reflex is high during stance, and low during swing. This allows a fast stretch in swing without evoking muscle activity, as observed in TD subjects in the present study. During stance, the GM and SO activity following stretch in healthy subjects may for a large part be attributable to stretch reflex activity (Sinkjaer et al., 1996). Hodapp et al. (2007) tested the stretch reflex during gait in CP, by measuring H-reflexes. They showed that in spastic calf muscles in CP the stretch reflex is amplified during the entire stride compared to normal. Hence, contrary to control subjects, the stretch of spastic muscles in swing is likely to evoke a stretch reflex and subsequent muscle activity. This is in line with our results, as illustrated by the four times higher ratio between EMG and stretch velocity.

Furthermore, the ratio between peak EMG and peak velocity increased with walking speed (Figure 6.4E-F), indicating that the effect of dynamic spasticity in swing is enhanced at faster walking speed. Interestingly, the ratio of EMG and stretch also increased with faster walking speed in TD subjects, indicating that stretch activity in swing may play a role at faster speed in normal gait as well. This is in line with increased stretch reflex amplitudes at faster walking speed in healthy subjects as reported by Sinkjaer et al. (1996).
Figure 6.1: Gastrocnemius EMG and muscle-tendon stretch velocity versus the gait cycle, for spastic cerebral palsy (CP) and typically developing (TD) children. Because the main effects occurred around initial contact, the x-axis is plotted from 0 to 100% and again to 50% of the gait cycle. For better comparison, data at matched speeds are presented in the same rows. EMG is normalized to peak EMG during the stride at comfortable walking speed (CWS) for CP, and at SLOW speed for TD. Stretch velocity is nondimensionalized by $\sqrt{\text{G}}$. Gray areas indicate swing phase.
Figure 6.2: Soleus EMG and muscle-tendon stretch velocity versus the gait cycle, for spastic cerebral palsy (CP) and typically developing (TD) children. Because the main effects occurred around initial contact, the x-axis is plotted from 0 to 100 and again to 50% of the gait cycle. For better comparison, data at matched speeds are presented in the same rows. EMG is normalized to peak EMG during the stride at comfortable walking speed (CWS) for CP, and at SLOW speed for TD. Stretch velocity is nondimensionalized by dividing \( \sqrt{F \cdot \gamma} \). Gray areas indicate swing phase.
In stance, the fast stretch in CP muscles during loading response coincided with muscle activity, but contrary to our hypothesis, this stretch was not followed by an additional increase in muscle activity (Figure 6.1 & 6.2). This discrepancy may be due to the fact that the muscles were already active at the onset of the second stretch peak. Therefore, the muscle belly was contracting and, as a consequence, the muscle belly may not have been lengthening at similar rate as the muscle-tendon complex. Muscle force can be expected to be increasing in this phase to support body weight, resulting in lengthening of the tendon, possibly accounting for most of the muscle-tendon stretch velocity. In voluntary toe walking, the muscle belly has been shown to even be shortening in this phase of the gait cycle, using ultrasound measurements (Fry et al., 2006). Since reflex activity is evoked by stretch of the muscle spindles (i.e. stretch in the muscle belly) rather than the muscle-tendon complex, this may well explain why no further muscle activity is evoked following the fast stretch in early stance. In swing, where the muscle force is initially low, the muscle belly length follows muscle-tendon length more closely. In voluntary toe walking, peak CE stretch velocity in swing of GM muscles has been shown to occur slightly later than peak muscle-tendon stretch, but at a similar rate (Fry et al., 2006). To accurately estimate stretch reflex activity during stance, future study should quantify CE velocity in relation to muscle activity, for example using ultrasound or modeling studies.

Figure 6.3: Gastrocnemius EMG versus muscle-tendon stretch velocity for spastic cerebral palsy (CP) and typically developing (TD) children, at matched speed (CP-CWS and TD-SLOW). Note that the graphs contain the same data as Figure 1B & D, but presented differently. EMG is normalized to peak EMG during the stride; stretch velocity is nondimensionalized by dividing by $\sqrt{\frac{F_{\max}}{m}}$.  

---

C. TD STANCE  
D. TD SWING
Some delay occurred between peak stretch velocity and peak EMG activity. The delay between peaks as found in this study was 155 ± 80 ms for GM and 236 ± 64 ms for SO respectively. Stretch reflex latencies during walking vary between approximately 40 and 120 ms for short and long-latency reflexes (Sinkjaer et al., 1999). This difference can partly be attributed to the fact that we determined the delay from peak stretch velocity to peak EMG, which occurs considerably later than the onset of the EMG activity. Furthermore, as described above, the peak stretch of the muscle belly may occur somewhat later in time than the peak stretch of the muscle-tendon complex. Moreover, the delays were comparable to the delay between peak stretch and peak activity during stance in TD muscles, where stretch-

<table>
<thead>
<tr>
<th></th>
<th>Peak stretch velocity swing</th>
<th>Peak EMG swing</th>
<th>Peak EMG / peak velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>CP SLOW</td>
<td>0.15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>CP CWS</td>
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</tr>
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</tr>
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</tr>
<tr>
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<td>0.20</td>
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</tr>
<tr>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>TD FAST</td>
<td>0.30</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

** Figure 6.4: A-B Peak stretch velocities in swing, C-D peak EMG values in swing, and E-F the ratio of peak EMG and peak stretch velocity; for increasing matched walking speeds. The group effect was evaluated for the two matched speeds only, ** p<0.01, *** p<0.001. All outcomes increased significantly with walking speed (p<0.001 for peak stretch velocity and peak EMG; p<0.05 for peak EMG / peak stretch ratio). CWS: comfortable walking speed; CP: cerebral palsy; TD: typically developing.

Some delay occurred between peak stretch velocity and peak EMG activity. The delay between peaks as found in this study was 155 ± 80 ms for GM and 236 ± 64 ms for SO respectively. Stretch reflex latencies during walking vary between approximately 40 and 120 ms for short and long-latency reflexes (Sinkjaer et al., 1999). This difference can partly be attributed to the fact that we determined the delay from peak stretch velocity to peak EMG, which occurs considerably later than the onset of the EMG activity. Furthermore, as described above, the peak stretch of the muscle belly may occur somewhat later in time than the peak stretch of the muscle-tendon complex. Moreover, the delays were comparable to the delay between peak stretch and peak activity during stance in TD muscles, where stretch-
Dynamic spasticity

reflex activity is thought to play a role as well (Sinkjaer et al., 1996). Taken together, the delays were within reasonable limits to attribute the rise in EMG to reflex activity.

Although the present data are in line with the concept of spasticity and stretch reflex modulation during gait, it cannot be concluded with certainty that spasticity is the only cause of the low muscle stretch velocity and increased muscle activity in swing in spastic muscles. First, spastic muscles also showed higher baseline EMG in initial swing (Figure 6.1 & 6.2) which partly explains why spastic muscles were slower in swing. Passive stiffness is also higher in spastic muscles (e.g. Lieber et al., 2004), which can lead to slower muscles in swing. Second, if healthy subjects walk on their toes voluntarily, they also show increased muscle activity in terminal swing, to prepare for toe-landing (Davids et al., 1999; Romkes and Brunner, 2007). However, the ankle and knee kinematic data in these studies differed between voluntary and CP toe walking, and no data were available on muscle-tendon velocities during voluntary toe walking. Moreover, stretch reflex modulation may be altered during voluntary toe walking, so that reflexes can be used to initiate GM and SO activity in terminal swing to support the body during stance in equine position.

Our results are somewhat different from those reported by Crenna (1998), who was the first to study dynamic spasticity in CP. Although Crenna did not present data on a group of subjects, he did show EMG versus lengthening velocity curves during stance, for the soleus of one child with diplegia and one healthy control subject. For the diplegic child, he found a looped curve similar to the curves found in swing in our spastic muscles (Figure 6.3B). The difference may be due to the fact that only one example muscle was shown, which may have been less affected compared to our subjects. Nevertheless, the concept of dynamic spasticity as proposed by Crenna (1998) was similar to our results.

The ratio of EMG and muscle-tendon stretch velocity during swing may be a good and simple measure for dynamic spasticity of the calf muscles during gait. First, it is in line with the concept of spasticity, defined as a velocity-dependent increase in muscle tone, since it incorporates both stretch velocity and muscle activity. Second, it distinguishes clearly between spastic and non-spastic muscles (approximately four times higher in CP, on average). Third, since muscle force is low and few dynamic effects (e.g. due to body weight support) are present in swing, it clearly shows the increased coupling between muscle stretch and activity, with few possible confounding factors.

To what extent the observed dynamic spasticity correlates with spasticity as measured in passive spasticity tests during physical examination still remains to be determined. In the present study, spasticity was measured clinically. In order to correlate dynamic spasticity during gait with passively measured spasticity, future study should quantify spasticity in a more objective, quantitative, and sensitive manner using instrumented tests.

The effect on the gait pattern of the evoked muscle activity in terminal swing is that it will limit further elongation of the calf muscles in terminal swing, thereby limiting knee extension and ankle dorsiflexion at initial contact, preventing accurate foot positioning. Because of electromechanical delay, most of the resulting muscle force will occur in early stance, thereby supporting the ankle extension in the equine position after landing. The high
Chapter 6

muscle activity in loading response and consequent high muscle force will then prevent further stretching of the muscle, supporting body weight in the equine position, consequently leading to heel rise or toe walking in early and mid stance. Due to the relatively higher increase in EMG activity with faster walking speed, this effect becomes more pronounced as walking speed increases, leading to more severe toe-walking pattern at faster speed. Walking with limited knee extension and limited ankle dorsiflexion in terminal swing and a toe walking gait pattern could thus, at least partly, be attributed to dynamic spasticity effects in the spastic calf muscles, which are already initiated in swing.

In general, we can conclude that the most prominent effect of dynamic spasticity in calf muscles occurred during the swing phase of gait: spastic calf muscles showed decreased stretch velocity combined with subsequent increased muscle activity, compared to non-spastic muscles, and this effect increased with walking speed.

Acknowledgements

The authors wish to thank Tanneke Vogelaar, Kim van Hutten, Esther Suurland, Carry Doeven, and Alexander Reeuwijk for their assistance in collection of the data presented in Chapter 4, 5, and 6 of this thesis.
Chapter 7

How crouch gait can lead to stiff-knee gait
A dynamic walking approach

Manuscript in preparation
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Daan J.J. Bregman
Steven H. Collins
Caroline A.M. Doorenbosch
Jaap Harlaar
Martijn Wisse

I am never content until I have constructed
a mechanical model of the subject I am studying.
If I succeed in making one, I understand.
Otherwise, I do not.

Lord Kelvin
Abstract

Introduction. Lack of adequate knee flexion during the swing phase of gait (stiff-knee gait) is a common gait deviation in children with cerebral palsy. It is often accompanied by a flexed-knee (crouch) gait pattern in stance. The aim of this study was to study the effect of a crouched posture, as well as the effects of push-off strength and hip torque, on knee flexion in swing.

Methods. We developed a simple dynamic walking model of human gait, with a passive knee in swing. The model was powered by an instantaneous push-off impulse under the trailing leg. It produced stable limit cycle gait patterns for a range of stance leg knee flexion (crouch) angles. The effect of crouch angle on knee flexion in swing was evaluated, as well as the influence of push-off impulse size and the addition of a spring-like hip torque on knee flexion in swing.

Results. In upright posture, the model showed sufficient knee flexion and clearance in swing. When increasing the crouch angle of the model, the knee flexed much less in swing, resulting in a ‘stiff-knee’ gait pattern and reduced clearance. The decreased knee flexion in swing could be explained by the passive dynamics of the model’s swing leg due to differences in position of the leg at swing initiation. Increases in push-off impulse size and hip torque led to more knee flexion in swing, but the effect of crouch angle on swing leg knee flexion and clearance remained.

Interpretation. These findings demonstrate that decreased knee flexion in swing can occur purely as a result of crouch, without any differences in actuation. This suggests that a stiff-knee gait pattern may result from uncontrolled dynamics of the system, rather than from altered muscle function or pathoneurological control alone.
7.1. Introduction

Adequate progression of the leg into swing is an essential aspect of human gait. In normal gait, the hip and knee are quickly flexed during pre-swing and initial swing, leading to forward progression of the swing leg and sufficient toe clearance. In many patient populations, such as cerebral palsy (CP) or stroke, knee flexion and knee flexion velocity in (pre)swing can be limited, leading to a stiff-knee gait pattern (Figure 7.1; Sutherland and Davids, 1993). Stiff-knee gait has been reported to be present in 80% of ambulatory children with CP (Wren et al., 2005) and can lead to reduced clearance, frequent tripping, reduced step length, and reduced speed, thereby limiting functional performance.

Several causes of stiff-knee gait have been proposed in the literature. The cause most often mentioned is excessive activity in quadriceps muscles, especially in rectus femoris, during swing (Piazza and Delp, 1996; Riley and Kerrigan, 1998) or during pre-swing (Anderson et al., 2004; Goldberg et al., 2004; 2006; Reinbolt et al., 2008). Reduced or ineffective push-off, for example due to gastrocnemius weakness (Kerrigan et al., 1991) or due to toe-walking (Kerrigan et al., 2001), have also been mentioned as possible causes of stiff-knee gait. Furthermore, reduced hip flexion torque during (pre)swing has been shown to reduce knee flexion in swing in simulations of gait (Piazza and Delp, 1996; Kerrigan et al., 1998; Riley and Kerrigan, 1999).

Although stiff-knee gait can coincide with variable alignment of the knee in stance, it often occurs in combination with excessive knee flexion in stance (crouch gait), as shown in Figure 7.1 (Sutherland and Davids, 1993). In crouch gait, the positioning of the leg during stance is affected, and as a result knee angles at the onset of push-off can differ vastly between subjects (Figure 7.2). To support such a posture during stance, the knee extension moment is
increased, which may limit knee flexion velocity at toe-off and thereby peak knee flexion in swing (Goldberg et al., 2006).

A crouched leg positioning during push-off may also, by itself, influence the progression of the leg into swing, for example by influencing the swing leg dynamics, the effectiveness of push-off, or the distribution of energy between the trunk and the swing leg. However, there is still a limited understanding of the biomechanical factors that lead to adequate knee flexion in swing, and little is known about possible effects of leg positioning on knee flexion in swing.

Many of the studies on stiff-knee gait used forward dynamic simulation and induced acceleration techniques using complex musculoskeletal models to study the role of local muscle functioning during (pre)swing on swing leg knee flexion. These analyses have been performed using full body simulations (e.g. Riley and Kerrigan, 1999; Goldberg et al., 2004), or on the swing leg only, prescribing the pelvis motion in time (Piazza and Delp, 1996). These approaches have yielded valuable insight into the role of individual muscle function on stiff-knee gait. However, the complexity of the models used may also hamper a more conceptual understanding of the causes of stiff-knee gait.

A different approach to gain insight into the mechanisms of human walking is that of (passive) dynamic walking. This approach uses relatively simple, conceptual models that can produce stable limit cycle gait. The concept of passive dynamic walking was introduced by McGeer (1990; 1993), and the simplest model of human walking was studied by and Garcia et al. (1998). Variations on this model have been applied to predict the preferred speed-step length transition (Kuo, 2001) and to study step-to-step transition costs (Kuo, 2002; Donelan et
al., 2002). Passive dynamic walking-based models have also been successfully applied in robotic research, resulting in stable and highly efficient walking machines (Collins et al., 2005).

Although simple models clearly do not cover all characteristics of human walking, they can help improve our understanding of the underlying principles of gait, and may give insight into the basic concepts of push-off and swing leg characteristics in normal and stiff-knee gait. Furthermore, because dynamic walking models can produce stable limit cycle gait, the effect of changes in parameters can be studied on the entire gait cycle for consecutive steps. The dynamic walking approach also takes optimal advantage of the dynamics of the system itself, and only limited control is necessary. To our knowledge, the underlying causes of stiff-knee gait have never been studied from a dynamic walking perspective.

The purpose of this study was to develop a dynamic walking model that can perform stable limit cycle gait, and to use this model to study the effect of a crouched posture on knee flexion in swing. We also evaluated the effect of push-off strength and of adding a hip torque to the model on swing leg behavior, since these are two of the main factors thought to influence knee flexion in swing.

7.2. Methods

Model description

We developed a forward dynamic simulation of human gait, based on the simplest walking model (Garcia et al., 1998), with several adaptations to make the model more anthropomorphic. The model and simulation methods used are outlined below and described in detail in Appendix A. The model was constrained to planar motion and is shown in Figure 7.3. It consisted of rigid segments connected with frictionless hinge joints, i.e. a point mass upper body, two upper legs, two lower legs, and two feet. The leg segments had anthropomorphic length, mass and inertia, and the point mass upper body had anthropomorphic mass (Van Soest et al., 1993). The model parameters can be found in Table 7.1.

The ankles of both legs were locked at 0°, so that shank and foot formed one rigid body. The knee of the stance leg was locked at a prescribed angle (the crouch angle), so that the stance leg acted as a single inverted pendulum. The knee of the swing leg was passive during swing, so that it could passively flex and then extend until it reached its prescribed crouch angle, at which point knee strike occurred and the knee was locked to prevent further extension. As such, this knee extension lock did not influence the knee flexion movement in early swing, which was our main outcome of interest, but only limited further extension in terminal swing, to make sure that the knee angle at foot contact was equal to the prescribed crouch angle.
The feet were modeled as curved feet and rolled over the arc bottom until the toe was reached. At this point, the toe became the new contact point, around which the foot rotated. The foot parameters were based on experimental roll-over shapes in humans (Hansen et al., 2004).

The walking motion is depicted in Figure 7.4, and consisted of a single support phase and an instantaneous double support phase. The model was powered by a pre-emptive, instantaneous push-off impulse, applied under the trailing (rearmost) foot just before contralateral heel strike, and directed towards the hip. Immediately after push-off, heel strike of the leading leg with the floor was modeled as an instantaneous, perfectly inelastic...
collision. The knee joint remained locked during push-off and collision, and were unlocked at the beginning of swing. During swing, scuffing of the swing leg through the ground was allowed, but evaluated as an outcome parameter, as described below.

Equations of motion were derived following Wisse et al. (2001) and are described in detail in Appendix A.1-6. A limit cycle analysis was performed using a first-order gradient search method to find periodic gait, of which the stability was assessed using Floquet analysis (McGeer, 1990; Appendix A.7). All simulations were performed in MatLab®.

Model studies

We performed a set of studies to evaluate the behavior of the model, and to study the effect of crouch angle, increasing push-off strength and hip torque. An overview of the studies performed and the main outcome measures is given in Table 7.2.

First we studied the general behavior of the model when walking with straight legs in stance (‘upright model’). This was done at a push-off impulse size of 40 Ns. This value was chosen to approximate human speed and step length, and the effect of impulse size was evaluated as described below. The resulting limit cycle was evaluated in terms of thigh, shank, and knee angles.

The effect of crouch angle was studied by performing a parameter study, in which the crouch angle was gradually increased, and a new limit cycle solution was searched for each crouch angle. The search was stopped when no further solutions existed, or when the model became unstable. The general behavior of the model in crouch in terms of thigh, shank, and knee angle, was evaluated at a crouch angle of 22.5°.

The main outcome measure of the parameter study was the increase in knee flexion in swing (ΔKFS) as a measure for a ‘stiff knee’, calculated as the peak knee flexion reached in swing minus the crouch angle. We also evaluated the amount of clearance, calculated as the lowest position reached by any point of the foot during the mid swing phase. Since scuffing was

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**Table 7.2: Overview of studies performed**

<table>
<thead>
<tr>
<th>Study</th>
<th>Crouch angle [°]</th>
<th>Push-off impulse size (Ns)</th>
<th>Hip spring stiffness (Nm/rad)</th>
<th>Main outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>General behavior</td>
<td>0</td>
<td>40</td>
<td>0</td>
<td>thigh, shank and knee angles</td>
</tr>
<tr>
<td>General behavior in crouch</td>
<td>22.5</td>
<td>40</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Crouch angle</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Push-off impulse size</td>
<td>0</td>
<td>min (16)-max (100)</td>
<td>0</td>
<td>ΔKFS, clearance, speed, step frequency, step length, energy²</td>
</tr>
<tr>
<td>Hip spring stiffness</td>
<td>0</td>
<td>40</td>
<td>0-max (4.9)</td>
<td>ΔKFS, clearance</td>
</tr>
<tr>
<td>Push-off impulse size x crouch angle</td>
<td>0-max</td>
<td>16-100</td>
<td>0</td>
<td>ΔKFS, clearance</td>
</tr>
<tr>
<td>Hip spring stiffness x crouch angle</td>
<td>0-max</td>
<td>40</td>
<td>0-max</td>
<td></td>
</tr>
</tbody>
</table>

¹ ΔKFS: increase in knee flexion in swing
² Energy parameters: total energy added; energy added to swing leg; energy added to trunk plus stance leg; total energy lost; energy lost at knee strike; energy lost at heel strike
allowed, the clearance could be negative, being a sign of inadequate swing leg behavior. Furthermore, we evaluated the spatiotemporal parameters speed, step frequency, and step length. Finally, since counterintuitively a constant push-off impulse does not necessarily lead to constant energy input, we evaluated several energy parameters. On the one hand we evaluated the total amount of energy added during impulse, and the distribution of this energy to the swing leg and the rest of the body (trunk + stance leg). On the other hand, we evaluated the total amount of energy lost, and the distribution between energy lost at heel strike and energy lost at knee strike. These energy values were calculated as the changes in total (i.e. sum of potential and kinetic) energy of the segments.

Next, we studied the effect of push-off impulse size. This was done first for the upright model, by gradually decreasing and increasing the push-off impulse size using the gradient search method, in order to find the full range of impulse sizes that yielded stable limit cycle solutions. The outcomes of this parameter study were again ΔKFS, clearance, speed, step frequency, step length, and energy parameters. Second, both push-off impulse size and crouch angle were varied simultaneously, to find solutions for all possible combinations of push-off impulse size and crouch angle values. The outcome of this 2-dimensional parameter search was evaluated in terms of the main outcome measures ΔKFS and clearance.

Finally, a hip torque was applied to the model by adding a torsional spring between the stance and swing leg, acting oppositely on both legs, with a torque linearly dependent on the angle between the two legs. This spring thus pulled the swing leg forward in initial swing, and slowed down the swing leg in terminal swing. Such a hip spring is commonly used as a simplified model representing the combined effect of muscles around the pelvis (Kuo, 2001; Dean and Kuo, 2008). A hip spring also allows to achieve more human-like step frequencies, by increasing step frequency and reducing step length for a given speed (Kuo, 2003). The effect of hip spring stiffness was evaluated again first for the upright model and subsequently for all possible combinations of hip spring stiffness and crouch angles.
7.3. Results

General behavior of the model

The gait pattern of the upright model is depicted in Figure 7.4A, and Figure 7.5A,B shows the corresponding thigh, shank, and knee angles as a function of the gait cycle (stance + swing). With the parameters set as described in Table 7.2 (top row) the model walked at a speed of 0.85 m/s, a step frequency of 1.03 steps/s and a step length of 0.83 m. Peak knee flexion in swing was 38° and occurred relatively early in swing, at about one third of the swing phase (Figure 7.5B). Knee strike occurred at approximately 50% of swing phase. The swing foot just cleared the ground in mid-swing, by 2.2 mm.

Energy was added by the push-off impulse only (10.2 J) and lost during the knee strike (2.5 J) and heel strike (7.7 J) collisions. Approximately 25% of the energy added was distributed to the swing leg and 75% to the rest of the body.

Increasing crouch angle

The crouch angle imposed on the stance knee could be increased from 0 to 28°. With higher crouch angles, the model tended to fall forward, because the knee flexion shifted the effective center of mass of the legs forward. As a result, the swing foot did not rise above the ground, and no cyclic gait pattern could be achieved. As an example, the crouch gait pattern at a crouch angle of 22.5° is depicted in Figure 7.4B. The corresponding segment and joint angles are shown in Figure 7.5C,D.
Figure 7.6 shows the effect of increasing crouch angle on the set of outcome measures. ΔKFS decreased from 28° to 0° with increasing crouch angle (Figure 7.6A), resulting in a ‘stiff-knee’ gait pattern. This is illustrated in Figure 7.7A, showing the knee angle as a function of the gait cycle for a number of increasing crouch angles. At higher crouch angles, no further knee flexion was achieved in swing at all and the knee remained fixed during the entire stride.

Figure 7.6: The effect of crouch angle on A ΔKFS, the increase in knee flexion in swing, and foot clearance; B speed, step frequency, and step length; and C energy distribution. Data are at a push-off impulse size of 40 Ns and no hip spring.

Figure 7.7: Knee angles as a function of the gait cycle for A increasing levels of crouch gait, B increasing push-off impulse size, and C increasing levels of hip spring stiffness.
Figure 7.7A also shows that not only ΔKFS, but also the absolute peak knee flexion decreased with crouch angle. Furthermore, the timing of both peak knee flexion and knee strike occurred earlier in the gait cycle. The reduced knee flexion in swing resulted in diminished clearance, which became negative at higher crouch angles (Figure 7.6A).

Speed, step frequency, and step length changed slightly with crouched posture (Figure 7.6B). Speed first decreased slightly from 0.85 to 0.83 m/s, and then started to increase again at higher crouch angles to 0.90 m/s. Step frequency increased from 1.03 to 1.16 steps/s. Step length slightly decreased with knee flexion, from 0.83 to 0.77 m.

The total energy added during push-off remained nearly constant with increasing crouch angle (Figure 7.6C). However, the amount of energy distributed to the swing leg decreased by approximately 25% from 2.7 to 2.0 J. The energy lost at knee strike also decreased with crouch angle, going to zero at higher crouch angles.

**Increasing push-off impulse size**

The upright model could be stably powered by a range of push-off impulses from 16 to 100 Ns. For smaller push-off impulses, the propulsion was insufficient to achieve a cyclic gait. The stance leg moved too slowly and the swing leg swung back before it could catch the fall of the stance leg. For larger push-off impulses, the model could reach cyclic solutions but became unstable.

ΔKFS and clearance increased with push-off impulse size, as did speed and step length (Figure 7.8A,B). ΔKFS ranged from 25° at low push-off impulse size, to 41° at a push-off impulse of 70. At higher push-off impulses ΔKFS leveled off and started to decrease slightly. The peak knee flexion and knee strike occurred somewhat later in the gait cycle with increasing push-off impulse size (Figure 7.7B). Speed increased up to 1.36 m/s, at a very large step length of up to 1.28 m (Figure 7.8B). Step frequency increased only slightly with push-off impulse size. The total energy added during push-off increased with impulse size (Figure 7.8C). The distribution between swing leg and trunk plus stance leg remained...
relatively constant, at approximately 25 versus 75%, while the energy was increasingly lost at heel strike.

Figure 7.9 shows ΔKFS and clearance as a function of both push-off impulse size and crouch angle. As can be seen in Figure 7.9A, ΔKFS decreased with increasing crouch angle for all push-off impulse sizes. For low push-off impulse values and high crouch angles, ΔKFS was zero, indicating that no further knee flexion in swing occurred. Clearance also decreased with increasing crouch angle for almost all push-off impulse sizes (Figure 7.9B). For low push-off impulse size and high crouch angles the clearance was close to zero.

Adding a hip spring

A hip spring could be added to the upright model with a peak spring stiffness of up to 4.9 Nm/rad. At higher hip stiffness, the swing leg moved too quickly to catch the fall of the stance leg, thus resulting in forward falling of the model.

The hip spring pulled the upper leg of the trailing leg forward in initial swing, resulting in increased ΔKFS and improved clearance with increasing hip spring (Figure 7.10A). Peak knee flexion occurred somewhat later in time with increasing hip spring, as did knee strike (Figure 7.10C). Speed and step frequency increased with hip spring stiffness (up to 13% and 18% respectively), while step length slightly decreased (Figure 7.10B). More energy was lost at knee strike and less at heel strike (Figure 7.10C). The distribution of energy to swing leg and body did not change with hip spring stiffness.
Figure 7.11 shows ΔKFS and clearance as a function of both hip spring stiffness and crouch angle. ΔKFS decreased with increasing crouch angle for all hip spring stiffness values. Clearance also generally decreased with increasing crouch angle, and was close to zero for low hip spring stiffness values and high crouch angles.

7.4. Discussion

The purpose of this study was to develop a dynamic walking model that could perform stable, limit cycle gait, and to use this model to study the effect of a crouched posture, as well as the effects of push-off strength and hip torque, on knee flexion in swing. Our results showed that by increasing the crouch angle of a simple walking model, both the change in knee flexion in swing (ΔKFS) and peak knee flexion in swing decreased strongly, resulting in
a stiff-knee gait pattern, and reduced clearance. Increasing push-off strength and adding a
hip flexion torque by means of a hip spring both led to more knee flexion in swing, but the
effect of crouch angle on ΔKFS and clearance remained largely the same.

The general behavior of the presented model was similar to that of comparable models
performing limit cycle gait with a passive knee in swing (McGeer, 1993; Dean and Kuo,
2008). The model in this study, in line with previously presented models, showed stable gait
patterns with several qualitative characteristics similar to human gait, although with limited
speed and relatively long step length (especially at faster speeds) compared to normal
human gait. The early peak knee flexion and knee strike in swing compared to normal
human gait are also in line with previous findings. The addition of a hip spring increased
step frequency for a constant speed and delayed the timing of peak knee flexion and knee
strike, resulting in more humanlike values. Considering the simplicity of the model and of
the powering and control applied, the speeds and gait patterns were not far from human
(pathological) gait values, and the main characteristics of human gait were consistent with
the model.

The effect of crouch angle on ΔKFS and clearance

The main finding of this study, the decrease in ΔKFS and occurrence of ‘stiff-knee gait’ with
increasing crouch angle, can largely be explained by the passive dynamics of the model’s
swing leg due to differences in position of the leg at swing initiation. In the crouch model,
the thigh of the trailing leg has a more vertical orientation at the onset of swing than in the
upright model, while the shank has a more horizontal orientation (Figure 7.4, compare
leftmost graphs). When considering the swing leg as a double pendulum, it can be seen that
the knee of the trailing leg will tend to flex more at the onset of swing in the upright model
than in the crouch model due to gravitational effects: the gravitational force acting on the
center of mass of the thigh has a larger moment arm relative to the hip in the upright model
than in the crouch model, and will tend to flex the hip more, simultaneously pulling the knee
into flexion. Similarly, the gravitational force acting on the center of mass of the shank has a
smaller moment arm relative to the knee in the upright model compared to the crouch
model, and therefore gravity is counteracting the knee flexion less in the upright model.

To test the importance of this gravitational effect on the swing leg relative to other potential
influencing factors, such as movement of the hip or differences in energy distribution, we
eliminated the effect of gravity in a separate simulation experiment. This was done using the
initial conditions of the model at the onset of swing, and simulating forward in time while
setting the gravitational force on the swing leg to zero. Without gravity on the swing leg, the
leg did not swing forward anymore, and no gait pattern could be generated. However, the
knee of the upright model initially flexed slightly, while the knee of the crouch model flexed
considerably. Thus, without gravity on the swing leg an opposite effect occurred, that was
outweighed by the effect of gravity in the normal simulation. Moreover, the opposite
simulation experiment, i.e. passively swinging the leg in a gravitational field but without hip
motion and with zero initial velocity, yielded a similar decrease of ΔKFS with crouch angle
as in the walking model, although with lower absolute peak knee flexion in all conditions. The effects of these factors are not linearly separable over the entire swing motion, but the experiments help lend insight into their roles, showing that gravitational effects were the main cause of the reduced ΔKFS in the crouch model. More generally, the natural dynamics, including gravity and the dynamic interactions of the stance and swing legs, in the absence of any neuromuscular control, predict the observed behavior.

Our results further showed that ΔKFS decreased with crouch angle, even when push-off impulse size was increased or when a hip torque was added. This shows that this effect is robust and remains a contributing factor, even when more complexity is added to the model. Moreover, the imposed crouch angle had a larger influence on ΔKFS than push-off impulse size or hip spring stiffness, and the effect of crouch angle could not be neutralized by any of these factors in our model. This further emphasizes the relevance of crouch angle on swing leg knee flexion.

Apart from decreasing ΔKFS, the clearance generally worsened with crouch angle as well. ΔKFS and crouch angle were not linearly related, as shown by the difference between Figure 7.9A and B, and between 7.11A and B. This is due to the fact that the clearance is influenced both by the degree of knee flexion in swing and by the timing of knee flexion. In the crouched posture, peak knee flexion occurred early in swing (Figure 7.7), and the knee was extending again at mid-swing, which resulted in foot scuffing. Thus, not only knee flexion itself, but also the timing of knee flexion is important for adequate foot clearance in mid swing.

Speed, step length, and step frequency were slightly affected by increasing crouch angle (Figure 7.6B). Since these parameters may by themselves affect ΔKFS, an additional simulation was performed in which the crouch angle was increased while keeping speed, step length, and step frequency constant. This resulted in similar outcomes, in which the effect of crouch angle on ΔKFS was even slightly enhanced. Thus, the change in spatiotemporal parameters with crouch angle did not have a large effect on the results.

The effect of push-off impulse and hip spring on ΔKFS and clearance

ΔKFS and clearance both generally improved when increasing push-off impulse size and increasing hip spring stiffness. These findings are in line with previous studies showing that hip flexion moments in (pre)swing and push-off strength are factors that help progressing the swing leg into flexion (Kerrigan et al., 1991; Piazza and Delp, 1996; Goldberg et al., 2004).

In our simulations, the push-off was directed towards the hip, in line with previous comparable model studies (e.g., Kuo, 2001; Donelan et al., 2002; Dean and Kuo, 2008). This push-off direction had a small effect on the results. In the crouch model the hip was somewhat lower and therefore the push-off impulse pointed slightly more forward compared to the upright model. This more forward direction of the push-off impulse tended to extend the knee in swing, as evidenced by an extra simulation in which only the push-off impulse direction was varied. This could therefore explain a small part of the difference.
between the upright and the crouch model. However, the difference in push-off direction with crouch angle was small, and when we repeated the simulations with constant absolute push-off direction, increasing the crouch angle still led to a similar decrease of ΔKFS and clearance.

However, the fact that a more forward direction of the push-off impulse leads to less knee flexion in swing has some interesting implications. In the present study we modeled the push-off as an external impulse with prescribed direction, but in human gait the push-off obviously is the result of joint torques, generated by muscles. The interplay between the applied joint torques, in combination with the positioning of the leg, will ultimately determine the effective ground reaction force size and direction, and may thereby affect the knee flexion in swing. For example, an ankle torque generated in a crouch/equinus position, may point the ground reaction force more forward than an ankle torque in upright position, which may lead to less knee flexion in swing. Further study replacing the external push-off under the trailing foot by (impulsive) ankle, knee, and/or hip torques, may give more insight into this effect.

Study limitations
One important factor in stiff-knee gait that was not incorporated in our model is the knee flexion velocity at toe-off. In our model, the knee was locked during stance and during the instantaneous push-off and heel strike collision, resulting in zero knee flexion velocity at toe-off. This may be one of the reasons for the overall slow speed, and the low and early peak knee flexion in swing, compared to human walking. Also, since no finite-time double phase was included in our model, the influence of factors during this phase on knee flexion in swing could not be studied, such as the transfer of body weight from the trailing leg to the leading leg, or the effect of joint torques during this phase. However, by excluding these more complex factors, our model was able to show that when walking in a crouched posture, the dynamic effects of gravity make it harder to achieve this rapid knee flexion in early swing, than when walking in an upright posture.

Another factor that may be added to the model to improve its walking performance, and that may give insight into swing leg knee flexion, is the addition of springs or actuators to the swing leg. Dean and Kuo (2008) showed that the addition of bi-articular springs around the hip and knee to a comparable model with knees contributes to the speed range, stability, and economy of the modeled gait. However, even with bi-articular springs, peak knee flexion in swing remained limited, indicating that other factors are likely to contribute to knee flexion in swing than those modeled so far in dynamic walking models.

Implications
In the present study a relatively simple model was used to gain a better understanding of underlying causes of a complex problem in pathological gait, i.e. a stiff-knee gait pattern. The existing literature on stiff-knee gait mainly emphasizes the role of local muscle functioning
during pre-swing and swing as causes for the limited knee flexion in swing, showing that muscles such as rectus femoris and hip flexors can substantially affect knee flexion in swing (e.g. Piazza and Delp, 1996; Goldberg et al., 2004; Reinbolt et al., 2008). However, the present study showed that stiff-knee gait can also arise purely from differences in posture, and without any differences in actuation. This indicates that part of a stiff-knee gait pattern may result from uncontrolled dynamics of the system, rather than from deviations in muscle functioning or neurological control alone. In other words, the knee in ‘stiff-knee gait’ need not necessarily be ‘stiff’, but the limited knee flexion movement may be caused elsewhere. Specifically, patients walking in crouch may experience problems with knee flexion in swing due to the dynamics arising from the crouched posture. Based on the current results, treatment of these patients at the (stiff-)knee level, e.g. with rectus femoris transfer or botulinum toxin treatment, may not achieve the desired effects if the stiff-knee has a dynamic cause. Treatment of these patients may better be directed at improving their upright posture, which may by itself improve their knee motion in swing and reduce the stiff-knee gait.

7.5. **Conclusions**

- When increasing the crouched posture in a simple dynamic walking model, and keeping all other parameters constant, peak knee flexion angle in swing decreased, resulting in a ‘stiff-knee’ gait pattern.
- Increasing push-off strength or adding a spring-like hip torque led to more knee flexion in swing, but the effect of crouch angle remained.
- The decreased knee flexion in swing can be explained by the passive dynamics of the swing leg due to differences in position of the leg at swing initiation.
- Our findings demonstrate that decreased knee flexion in swing can occur without any differences in actuation. This indicates that part of a stiff-knee gait pattern may be explained by uncontrolled dynamics of the system, rather than from altered muscle function or neurological control alone.
Appendix A. Model equations

A.1. Equations of motion

The method used to derive the equations of motion is derived from Wisse et al. (2001) and based on the concept of virtual work. This method is also called the ‘TMT-method’, and the resulting equations are equal to those obtained with Lagrange’s method.

According to Newton, the sum of the forces must be equal to the mass times the accelerations:

\[ \Sigma f - Mx = 0 \]  

[1]

In combination with ‘virtual velocity’, this yields the virtual power equation:

\[ \delta x [\Sigma f - Mx] = 0 \]  

[2]

which says that the sum of the work done by all internal forces must be zero. This is true because all internal forces have opposite but equal reaction forces, delivering opposite and equal work, cancelling each other out for each instant in time.

First, a vector of global coordinates is defined, three for each segment:

\[ x = [x_1, y_1, \ldots, x_N, y_N]^T \]  

[3]

with \( x_i \) the x-coordinate of the center of mass of segment \( i \); \( y_i \) the y-coordinate of the center of mass of segment \( i \); \( p_i \) the orientation (angle) of segment \( i \) relative to global; and \( N \) the number of segments.

Next, a vector of generalized coordinates is defined, one for each degree of freedom:

\[ q = [p_1, \ldots, p_N, x_h, y_h]^T \]  

[4]

with \( p_i \) the angle of segment \( i \) relative to global, \( N \) the number of segments, and \( x_h \) and \( y_h \) the position of the hip joint.

We then express \( x \) as a function of the generalized coordinates by means of a kinematic transfer function \( F \):

\[ x = F(q) \]  

[5]

Next, we define \( T \) the partial derivatives matrix of \( x \) to \( q \), so:

\[ T = \text{Jacobian} (x,q) \]  

[6]

Equation [6] is used in order to calculate the derivatives of \( x \) as a function of \( q \), to input in our virtual power equation:

\[ \frac{\partial x}{\partial q} \frac{\partial q}{\partial t} = Tq \]  

[7]

and, using the product rule:
We define:

\[ T_i = \frac{\partial T}{\partial q_i} \]  

Combining equation [8] and [9] gives:

\[ \dot{x} = T_i \dot{q}_i + T \]  

Now we go back to the virtual power equation [2], and fill in [7] and [10]

\[ \delta (T_q) [ \Sigma f - MT_qq + T_q] = 0 \]

which has only generalized coordinates \( q \). Equation [11] must be true for all virtual velocities, so for all \( \dot{q} \). Rearranging gives:

\[ T^T M T \dot{q} = T^T \Sigma f - T^T M T \dot{q}_q \]  

Equation [12] can then be simplified by defining \( \tilde{M} \), the reduced Mass matrix (hence the 'TMT method'):

\[ \tilde{M} = T^T M T \]  

and \( \tilde{f} \) the reduced force vector which becomes, when adding \( Q \) as the generalized forces that are expressed directly in the coordinates of \( q \) (see A.6.)

\[ \tilde{f} = T^T \Sigma f - T^T M T \dot{q}_q + Q \]  

\( T^T M T \dot{q}_q \) represents the Coriolis forces, apparent forces resulting from accelerations of the system.

Adding [13] and [14] to [12] yields the simplified equation:

\[ \tilde{M} \ddot{q} = \tilde{f} \]

A.2. Constraint equations

Now that we have the basic equations of motion, describing the system when no constraints are present, we still need to add constraint equations that describe the contact with the ground, as well as the locking of joints.

It is assumed that if the foot is in contact with the ground, it is fully fixed to its attachment point, so no sliding is allowed. Each foot rolls over the arc until it reaches the toe, which is modeled as a hinge constraint.

The rolling arc foot constraint is formulated as follows:
with $x_{arc}$ the lowest point of the arc foot, which is in contact with the ground, $R$ the foot radius, $p_f$ the foot angle, $p_{f1}$ the foot angle at first foot contact, and $x_{c1}$ the horizontal position of the bottom of the foot at first foot contact.

The toe constraint is modeled as:

$$d_{tow} = x_{tow} - \begin{bmatrix} x_{c2} \\ 0 \end{bmatrix} = 0$$

with $x_{tow}$ the position of the toe and $x_{c2}$ the horizontal toe position at first toe contact.

Similar constraint equations are formulated to lock the ankle and knee joints:

$$d_{j0} = p_d - p_p - p_c = 0$$

with $p_d$ the angle of the distal segment, $p_p$ the angle of the proximal segment, and $p_c$ the constraint angle of the joint.

It is evident that $d$ changes for different phases of the gait cycle: only those constraints are modeled that describe the foot contacts and joint locks that are present in each gait phase. The derivatives of $d$ can then be calculated as:

$$\dot{d} = \frac{\partial d}{\partial q} \dot{q} + \frac{\partial d}{\partial \dot{q}} \ddot{q} + \frac{\partial d}{\partial \ddot{q}} \dddot{q}$$

and, similarly as above for $x$:

$$\dot{d} = D_1 \dot{q} + D_2 \ddot{q} + \frac{\partial D_1}{\partial \ddot{q}} \dddot{q}$$

or:

$$D_1 \dot{q} = -\dot{D}_1 \ddot{q}$$

Adding the constraint forces $f_c$ to the general equation of motion and combining with the constraint equation gives:

$$\begin{bmatrix} M & D^T \\ D & 0 \end{bmatrix} \begin{bmatrix} \ddot{q} \\ f_c \end{bmatrix} = \begin{bmatrix} \bar{f} \\ -D_1 \dot{q} \end{bmatrix}$$

The equations of motion are solved forward in time by numerical integration using Matlab® ODE23 function.

### A.3. Event detection

Figure 7.4 shows the gait phases of normal gait. Arbitrarily, the beginning of each stride is defined as toe-off of foot 2, thus the beginning of single support on leg 1. In the single stance phase, the model searches for the following events:
• Event 1: toe strike: bottom of the arc foot passes the toe. At this point the arc foot constraint is replaced by the toe constraint

• Event 2: knee strike: knee angle crosses the prescribed stance leg knee angle. At this point the knee is locked by the knee constraint

• Event 3: heel strike: the swing leg arc foot hits the floor. At this point an instantaneous push-off impulse is applied under the trailing leg (A.4.), followed by an instantaneous collision of the leading foot (A.5.).

• Event 4: foot lift: the force under the stance foot crosses zero and becomes negative. At this point the model tends to lift off and the simulation is stopped.

A.4. Impulsive push-off

At event 3, an instantaneous push-off impulse is applied under the rear foot. During this infinitely small time period, positions of the system are assumed to remain constant and only velocities change. It can be said that over a short interval of time, from \( t^- \) (prior to impact) to \( t^+ \) (after impact), the equations of motion must be true:

\[
\lim_{t \to t^-} \int Mq dt + \lim_{t \to t^+} \int D^T f dt = \lim_{t \to t^-} \int T dt
\]  

The second term of [23] includes the constraint forces of the joints to be locked during impulse and the push-off impulse. The foot constraints are not included, as the leading leg has not yet touched the ground, and the trailing leg is allowed to come off the ground after the push-off impulse.

We can define the push-off impulse \( \rho_p \) as:

\[
\rho_p = \lim_{t \to t^-} \int f_c dt
\]  

and the resulting impulses in the constraints:

\[
\rho_c = \lim_{t \to t^-} \int f_c dt
\]  

The second term in [23] can then be split into the known impulses applied under the trailing foot: \( D^T \rho_p \), and the unknown resulting impulses in the joint constraints: \( D^T \rho_c \).

with \( D_p \) describing the foot contact of the trailing leg where the push-off impulse is applied (based on \( a_{arc} \) or \( a_{end} \)) and \( D_c \) the constraints to lock the joints.

The first term of [23], the change of momentum, is equal to:

\[
\lim_{t \to t^-} \int Mq dt = \tilde{M} \ddot{q} - \tilde{M} \ddot{q}^-
\]  

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The right hand site term in [23] goes to zero, since all forces other than the impulses are not infinitely high. Rewriting [23] then gives:

\[ \bar{M} \ddot{q} + \bar{D}^T \dot{q} = \bar{M} \ddot{q} - \bar{D}^T \dot{q} \]  

[27]

Combining [27] with the constraint equation yields the push-off impulse equations:

\[ \begin{bmatrix} \bar{M} & \bar{D}^T \\ \bar{D} & 0 \end{bmatrix} \begin{bmatrix} \ddot{q} \\ \dot{\rho} \end{bmatrix} = \begin{bmatrix} \bar{M} \ddot{q} - \bar{D}^T \dot{q} \\ 0 \end{bmatrix} \]  

[28]

### A.5. Impact equations

Impact is modeled as a fully inelastic, instantaneous collision, after which the leading foot is fixed to the ground. The impact equation is comparable to the push-off equation [28], \( \bar{D}^T \) and \( \dot{\rho} \) now include the (unknown) constraints and impulses of the joint constraints, as well as of the leading foot, since this foot is fixed to the ground after impact. Equation [27] then becomes:

\[ \bar{M} \ddot{q} - \bar{D}^T \dot{q} = \bar{M} \ddot{q} - \bar{D}^T \dot{q} \]  

[29]

Finally \( e \) can be defined as the restitution coefficient, the relative velocity after impact divided by the relative velocity before impact, with \( e = 1 \) if fully elastic and \( e = 0 \) if fully inelastic. For the general case of \( 0 \leq e \leq 1 \),

\[ e = \frac{\dot{q}}{\dot{q}} = \frac{\ddot{q}}{\ddot{q}} \]  

[30]

Combining [29] and [30] yields the impact equations:

\[ \begin{bmatrix} \bar{M} & \bar{D}^T \\ \bar{D} & 0 \end{bmatrix} \begin{bmatrix} \ddot{q} \\ \dot{\rho} \end{bmatrix} = \begin{bmatrix} \bar{M} \ddot{q} \end{bmatrix} \]  

[31]

with \(-e\dot{D}\ddot{q} = 0\) for a fully inelastic impact.

### A.6. Hip spring

The hip spring is modeled as a joint moment \( Q_j \), depending on inter-leg joint angle \( p_j \):

\[ Q_j = -k(p_j - p_{\text{neutral}}) \]  

[32]

with \( k \) the stiffness constant, and \( p_{\text{neutral}} \) the neutral joint angle, which is set to zero. \( Q_j \) is then expressed in the general coordinates \( \mathbf{q} \) by a kinematic transfer function.

### A.7. Cyclic motion and stability assessment

The stability of the model is assessed by comparing the state at the beginning and the end of one step. For this, a step function is defined as:
\[ v_{n+1} = S(v_n) \text{ with } v = (q, q) \]  
which is cyclic if:
\[ S(v_0) = v_c \]

This cyclic limit cycle is searched for using a first-order gradient search method. The stability of this cyclic initial state \( v_c \), i.e. the ability of the model to go back to its cyclic motion if a small perturbation occurs, can then be determined by calculating the Jacobian \( J \) as the partial derivative of \( S \) to \( v \). The state \( v_c + \Delta v \) after a perturbation \( \Delta v \) can be quantified as:
\[ v_c + \Delta v = S(v_c + \Delta v) = S(v_c) + J \Delta v \]  
with \( J = \frac{\partial S}{\partial v} \)

Thus:
\[ \Delta v = J \Delta v \]

For stability, \( \Delta v < \Delta v \) for all small perturbations \( \Delta v \). Therefore, the cycle is stable if all eigenvalues of \( J \) are < 1.
Chapter 8

General discussion
8.1. Introduction

The many impairments that can occur in cerebral palsy (CP), combined with the complexity of human gait in general, make it difficult to determine the underlying causes of gait deviations in CP. Yet, for optimal treatment planning and evaluation, unraveling which factors are responsible for a specific gait pattern is essential.

The aim of this thesis was to gain insight into the underlying causes of gait deviations in children with spastic CP; with a specific focus on the role of spasticity during gait; the interplay between the effects of spasticity, muscle contractures, and walking speed; and the role of dynamics.

Musculoskeletal modeling was used to study the gait pattern at the level of the impairment, i.e. by investigating muscle-tendon lengths and velocities during gait. In order to separate the role of impairments during gait, this technique was combined with the use of healthy subjects as a model (Chapter 3), the variation of walking speed to modulate spasticity (Chapter 4-6), and the measurement of muscle activity during gait (Chapter 6). Finally, in Chapter 7, a forward dynamic modeling approach was applied to study possible dynamic causes of gait deviations.

This general discussion starts with some considerations regarding the methods used in this thesis. Next, the main findings of the thesis and their contribution towards understanding the underlying causes of gait problems in CP will be discussed. The clinical implications of this research will be discussed, as well as recommended directions for further research. Finally, the main conclusions of this thesis will be summarized.

8.2. Methodological considerations

Musculoskeletal modeling

An important aspect of the research presented in this thesis was the use of musculoskeletal modeling for the calculation of muscle-tendon lengths during gait. In Chapter 3 we applied the SIMM musculoskeletal model (Delp et al., 1990; Delp and Loan, 1995) for calculation of semitendinosus, biceps femoris and psoas length in healthy adults, and in Chapter 4 to 6, we used the same model for calculation of hamstrings, gastrocnemius, and soleus length in typically developing (TD) children and in children with CP.

Some debate exists in the literature on the accuracy of a generic model, based on healthy adult male data, for the calculation of muscle-tendon lengths in individual subjects. In Chapter 2 of this thesis it was shown that individual differences can exist in calculated muscle-tendon lengths, even for healthy adults. Scheys et al. (2008a) also found that differences exist between the scaled generic SIMM model and individualized models based on MRI data, in moment arms and muscle-tendon length. On the other hand, the generic models yielded accurate values for changes in muscle-tendon length during gait for all main
muscles (Scheys et al., 2008a), which indicates that muscle-tendon velocities are expected to be accurately estimated. Moreover, no systematic errors were found for the SIMM model in Chapter 2, which indicates that the model is expected to yield good results at a group level.

Some additional error may be introduced when a generic model is used for the calculation of muscle-tendon length in children with CP. Some of our subjects presented with mild bony deformities (severe cases were excluded), such as increased femoral anteversion and tibial torsion, which were not taken into account in our modeling. However, it has been shown that the generic model can provide accurate estimates of medial hamstrings and psoas lengths in persons with neuromuscular disorders when scaled accurately to the individual subject sizes (Arnold et al., 2001), as was done in the present thesis. Furthermore, calculated hamstrings length has been found to be relatively insensitive to femoral rotational deformities (Schutte et al., 1997).

Contrarily, Scheys et al. (2008b) found that hamstrings hip extension moment arms were slightly overestimated in the case of femoral anteversion. This indicates that the effect of hip flexion on hamstrings length is slightly overestimated. Since the hips are generally overly flexed in CP, hamstrings length would be slightly overestimated by the model in CP, leading to a smaller difference between CP and TD children than present in reality. In general, Scheys et al. found that differences in moment arm lengths in CP, and consequent increased or reduced muscle-tendon length estimates, were consistent with major gait characteristics presented in CP patients. In other words, differences between CP and TD subjects tend to be underestimated rather than overestimated by the model. Therefore, differences between groups as found in the musculoskeletal modeling studies of this thesis can be considered true differences and not a result of modeling error.

**Statistical analysis**

In Chapter 4 and 5 we used a generalized estimating equation (GEE) analysis to study the effect of spasticity level (Chapter 4), muscle contractures (Chapter 5), walking speed, and their interactions on muscle-tendon length and velocity. This GEE analysis allows to study the individual effect of various independent parameters on the outcome measures. Several other parameters could theoretically also be included in this analysis (e.g. muscle stiffness, selectivity, muscle strength, etc), so that their relative contribution on the outcome measures could be estimated. However, in the present studies the sample size was too small to include more factors in one analysis. This makes it difficult to definitely conclude on which factors are responsible for specific gait deviations, since other factors may interact with the independent variable under study. Yet, even when more factors could have been included, correlations do not necessarily imply a causal relationship. In the absence of such a large dataset of gait data, we focused on one specific group distribution in each chapter, in combination with the effect of walking speed.
8.3. Gait deviations explained?

From impairments to muscle behavior during gait

In Chapter 1, spasticity was defined following Lance (1980) as a velocity-dependent increase in muscle tone. It was stated that little is known about the clinical relevance of spasticity during gait. This thesis attempted to measure the effect of spasticity on gait. Referring back to Figure 1.2, we purposefully studied gait kinematics at the muscle-tendon level rather than at joint level, since this more closely relates to the impairment of spasticity.

In Chapters 4 to 6, it was hypothesized that spasticity during gait would lead to:

• Decreased muscle-tendon stretch velocity;
• Increased coupling between muscle-tendon stretch velocity and muscle activity;
• Amplification of these effects with increasing walking speed.

Chapter 4 and 5 showed that spastic muscles in children with CP are indeed stretched at slower velocity than non-spastic muscles, even when controlled for walking speed. Spastic hamstrings muscles reach increasingly slow stretch velocity as their spasticity score in clinical testing increases (Chapter 4). Spastic soleus and gastrocnemius muscles are stretched markedly slower than control muscles in swing (Chapter 5).

Contractured muscles, i.e. muscles showing a decreased range of motion in physical examination, are shorter during gait than non-contractured muscles, but not necessarily slower. Gastrocnemius and soleus muscles with contractures (as measured with the ankle range of motion) were considerably shorter during gait than muscles without contractures. Nevertheless, they were almost equally slow; indicating that the slow stretch velocity cannot be attributed to contractures. Additional analyses showed that hamstrings contractures (as measured with the popliteal angle during physical examination) were also strongly related to hamstrings peak length during gait, possibly explaining an additional part of hamstrings shortness during gait. Yet, these contractures could not significantly explain muscle-tendon peak stretch velocity, indicating that hamstrings contractures mostly affect peak length during gait. Thus, in general, spasticity appears to be most strongly related to slow muscle stretch velocity during gait, while contractures are most strongly related to short muscle length during gait.

Spastic muscles also showed an increased coupling between muscle-tendon stretch and muscle activity. The stretch of spastic calf muscles always coincided with muscle activity, while in typically developing children muscles could also stretch fast without concomitant muscle activity (Chapter 6). Our data also revealed that spastic hamstrings muscles showed increased muscle activity during the stretch phase compared to non-spastic muscles (Van der Krogt et al., 2007). These findings are similar to Crenna et al (1999), and further indicate that spasticity does play a role during gait.
In order to modulate the effect of spasticity during gait, the children walked at a range of walking speeds. Faster walking speed led to faster stretching of hamstrings, gastrocnemius, and soleus muscles. Contrary to our hypothesis, peak stretch velocity in spastic muscles increased with walking speed just as much as in control muscles. Only the hamstrings muscles showed a trend towards relatively slower spastic muscles at faster speed. However, the muscle activity that followed this (faster) muscle-tendon stretch did increase with walking speed (Chapter 6). Moreover, muscle activity increased relatively more than stretch velocity itself with increasing walking speed, as indicated by the increase in activity/stretch ratio, which is in line with the notion of spasticity. Thus, the combined measure of stretch and activity, i.e. the dynamic spasticity, did worsen with increasing walking speed. This combination of stretch velocity and EMG activity therefore gives the best picture of spasticity effects during gait.

The change in muscle-tendon length pattern of the calf muscles with walking speed could be due to these increasing dynamic spasticity effects: the high muscle activity in terminal swing and loading response (Chapter 6) can explain the shortening of the muscles in mid-stance (Chapter 5). The hamstrings muscles did not show such a change in muscle-tendon length pattern with walking speed, but only showed an increase in range of motion with speed.

Contrary to the first hypothesis, gastrocnemius and soleus were stretched faster than normal during stance (Chapter 5). This effect could not be explained by spasticity effects per se, but rather by the muscles’ activation history in the preceding terminal swing phase, and to dynamic effects due to toe-landing. The effect was not contradictory to spasticity, i.e. muscles were active during and following the fast stretch, so no fast stretch of muscles occurred without muscle activity. Rather, this result illustrates that during a complex tasks such as gait, dynamic factors need to be taken into account as well.

All results taken together, it can be concluded that spasticity effects can be recognized during CP gait at the muscle-tendon level. In contradiction to recent studies that attributed a major role to muscle strength and selectivity during gait (Desloovere et al., 2006; Ross and Engsberg, 2007), our results indicate that spasticity does play a role in CP gait. The following paragraph will discuss how these effects translate to joint and segment kinematics.

**From muscle behavior during gait to joint kinematics**

Muscle behavior during gait is obviously related to joint and segment kinematics (refer to Figure 1.2). Muscle-tendon lengths were calculated from the 3D kinematic data of the segments, and each effect seen at the muscle-tendon level must thus be reflected in joint or segment kinematics, and vice versa.

However, the link between the two is not always straightforward, especially when bi-articular muscles are involved. Crouch gait has often been attributed to hamstrings spasticity or contracture. Although spastic or contractured hamstrings muscles in CP are indeed shorter and slower than normal during gait (Chapter 4), Chapter 3 showed that this does not necessarily lead to a crouched posture. The healthy subjects in Chapter 3, who walked in
crouch voluntarily, walked with limited knee extension in terminal swing. Yet, due to the simultaneously increased hip flexion, hamstrings muscles reached similar peak lengths during gait as normal. Moreover, neither hamstrings nor psoas muscles were stretched slower than normal.

The short length and slow stretching of hamstrings muscles that are seen in children with CP must thus result from a different cause than a crouched posture alone. Specifically, the short and slow hamstrings during gait oftentimes coincided not only with increased knee flexion, but also with posterior tilt of the pelvis in terminal swing, which was not seen in the healthy subjects walking in crouch. Short peak length was also achieved by taking shorter steps. These results illustrate that in the case of bi-articular muscles such as the hamstrings it is important to assess both joints that are crossed by the muscle. Furthermore, it should be noted that the peak length and peak stretch velocity of the hamstrings both occurred in swing. Limitations due to hamstrings spasticity or contractures should thus also be analyzed in swing. Altered kinematics in terminal swing or around initial contact (e.g. posterior pelvic tilt, limited knee extension) could be attributed to hamstrings tightness or spasticity, but altered kinematics later in stance could not. More generally, in order to attribute a certain gait deviation to spasticity or contracture of a specific muscle, it is important to analyze the gait phase in which the muscle is longest or stretched fastest; and to evaluate the muscle over its entire range of motion.

The dynamic spasticity effects in the calf muscles are also reflected in joint and segment kinematics. The slow gastrocnemius and soleus stretch velocity in swing coincided with decreased ankle dorsiflexion and knee extension velocities in terminal swing, leading to excessive knee flexion and ankle plantar flexion at initial contact. The relatively large peak in muscle activity in terminal swing and early stance at faster walking speed could not prevent a fast ankle dorsiflexion in loading response, but caused early heel rise and excessive planar flexion of the ankle in the second half of stance, especially at faster walking speed. Indeed, with increasing walking speed, we saw a clear increase in the severity of equinus gait in mid-stance (Van der Krogt et al., 2008). Patients with contractured calf muscles walked with shorter soleus lengths, and thus with more ankle plantar flexion throughout stance. Nevertheless, the increase in this toe-walking gait pattern with walking speed was similar in children with and without contractures, and could thus be attributed mainly to spasticity effects and not to contractures.

The role of gait dynamics

Human walking is a complex task, in which the movements of joints and segments are coordinated by neuromuscular control, but also follow the dynamics of the musculoskeletal system. Similarly, muscle-tendon behavior is influenced by the stimulation to the muscle, but is also affected by other external forces and moments on the segments, such as those due to inertia and gravity. An example of such an effect was already discussed above for the spastic calf muscles that were stretched fast despite spasticity, due to the large forces put on the muscle during the loading of the body in stance.
Another illustration of the role of gait dynamics was given in Chapter 7, in which the dynamic effect of a crouch gait pattern on knee flexion in swing was studied, using a relatively simple forward dynamic model. This study showed that knee flexion in swing can be limited due to the crouched posture per se. Muscle activity can influence these dynamics (e.g. hip flexion torque again increased knee flexion in swing), but the behavior of these muscles is in turn influenced by the dynamics of the system. For example, in this case of stiff-knee gait, rectus femoris may shorten and lengthen over a limited length range and at limited velocity during gait due to the dynamics of crouch, rather than due to spasticity effects, increased tone, or intrinsic muscle properties.

These examples illustrate that such dynamic effects are important to consider as possible causes of gait deviations, next to the attribution of gait deviations to abnormal muscle functioning.

**From gait deviations to gait limitations**

The focus of this thesis was mainly on the level of structures and functions during gait. However, from a patient’s perspective, it is especially important whether these structural effects limit their walking capacity in terms of (comfortable) walking speed, energy cost, or their risk of falling. As discussed in Chapter 1 and illustrated in Figure 1.1, most patient-oriented goals lay at the ICF domains of activity and participation. Based on the present results, no claims can be made at the level of participation. Yet, the findings of this thesis do translate to walking capacity in the activity domain.

First, the interplay of walking speed with gait parameters yielded some interesting findings. Walking speed has previously been shown to affect kinematic, kinetic, and electromyographic gait data in typically developing children in a complex manner (Schwartz et al., 2008). Our findings showed that walking speed also affects muscle and joint kinematics in children with CP. Analyses on joint kinematics revealed that especially equinus gait and knee (hyper)extension in stance increased with walking speed (Van der Krogt et al., 2008). As a result, the gait classifications as defined by Becher (2002) changed with walking speed in 19 out of 33 evaluated limbs in CP. These effects of walking speed can partly be attributed to spasticity, which influence increased with walking speed. Most likely, the changes in muscle and joint kinematics can also be attributed to the dynamic effects of walking speed per se.

These results show that walking speed and gait deviations are closely related.

Since the gait pattern thus generally worsened with increasing walking speed, walking more slowly can be a good strategy to prevent this from happening. For example, slow walking speed prevents fast muscle stretching (Chapter 3-5), and therefore limits consequent (inadequate) muscle activity to occur (Chapter 6). Slow walking may ease the severity of a toe-walking gait pattern or of knee hyperextension in stance. These findings may (partly) explain why it can be more optimal for a child to walk at slower walking speed, given the constraints imposed by spasticity, and thus why comfortable walking speed is limited in children with CP.
Furthermore, gait deviations have previously been shown to be related to energy expenditure during gait, and interruption of a normal gait pattern results in increased energy expenditure (Waters and Mulroy, 1999). For example, children with spastic hemiplegia walking with a dynamic equinus deformity on their affected side, showed a 1.3 times greater energy expenditure than typically developing children (Van den Hecke et al., 2007). This increased energy cost could be attributed to increased mechanical work performed by the muscles due to the toe-walking gait pattern. Similarly, a crouched gait pattern leads to a large increase in energy cost (Waters and Mulroy, 1999). It can thus be expected that the gait deviations as studied in this thesis also translate to changes in energy expenditure. Although no one-to-one relationship exist, insight into the causes of gait deviations may therefore also give a possible indication for the causes of increased energy cost.

Finally, little is known about the relationship between gait deviations and stability, and stability during gait is difficult to quantify. However, it can be expected that an increased equinus or toe-walking gait pattern will reduce stability in stance, and consequently increase the risk of falling.

In conclusion, the findings of this research at the level of body structures and function may also provide a starting point to improve walking capacity in terms of speed, energy cost, and stability.

**Gait deviations – a step further**

The present thesis, with the exception of Chapter 7, focused on specific structural components of the body, i.e. on individual muscle contributions to the gait pattern. Yet, it is quite likely that the overall gait pattern emerges as an optimal solution given the set of changes in the central nervous and effector system (Latash and Anson, 1996). Also, the (largely unknown) set of priorities of the system to optimize for, i.e. what is ‘optimal’ to a patient, will also most likely be altered in CP. As such, the changes in motor patterns could be considered to be adaptive to the altered dynamic properties of the neuromusculoskeletal system, rather than a sign of inability (Latash and Anson, 1996).

The typical gait patterns seen in CP, such as toe-walking, crouch, or knee-hyperextension, may thus be optimal for the patient, given the total of altered properties occurring in CP, such as spasticity, increased muscle / joint stiffness, limited selective control, stability, or weakness. This is illustrated below, as an example, for the typical crouch / toe-walking gait pattern (Type 4 of Becher et al. (2002)). This gait pattern may be the ‘optimal’ solution given either (or more) of the following impairments:

*Spasticity:* In a crouch / toe-walking gait pattern, the calf muscles are not stretched fast in swing, since foot lift and knee extension are limited (Chapter 5). This limits the amount of muscle activity that is evoked in swing (Chapter 6). Moreover, the muscle activity that is evoked in terminal swing can be used in stance to support body weight, and hence is effective within this gait pattern. Similar effects could be the case for the hamstrings or rectus femoris. The hamstrings may not be stretched fast in a crouch / toe-walking gait pattern.
because of posterior tilt and reduced knee extension in terminal swing, while the rectus femoris muscles may not be stretched fast in (pre)swing because the knee is already flexed in stance and less increase in knee flexion is necessary in swing. Thus, spasticity of these muscles may have less effect in a crouch / toe-walking gait pattern.

**Selective motor control:** In a crouch / toe-walking gait pattern, all extensor muscle groups (glutei, quadriceps, plantar flexors) can be activated simultaneously during stance, while most flexor muscles can be active simultaneously in swing. This is in line with the primitive control strategy of flexion-extension synergies that are thought to persist after early childhood in CP (Perry, 1992; Lin, 2004; Fowler and Goldberg, 2009). Thus, a crouch-equinus gait pattern may be easier to control when selectivity is poor.

**Stiff muscles/joints:** When muscles and joints are intrinsically stiffer than normal, the stiffness of the leg as a whole will also increase for a certain joint configuration. Overall leg stiffness has been shown to be an important factor in running and hopping, where the leg acts in a highly spring-like manner (McMahon and Cheng, 1990; Farley and Gonzalez, 1996). Recent studies show that also in walking spring-like behavior and overall stiffness of the leg are important (Geyer et al., 2006; Iida et al., 2008). Simple spring-mass models were shown to yield very realistic walking patterns, with realistic M-shaped vertical ground reaction curves, double support time, and high stability. Overall leg stiffness can be reduced by a more flexed positioning of the joints, which has been shown to be an effective adaptation strategy in hopping and running on different terrain (Farley et al., 1998). The crouch / toe-walking position may thus be an effective manner to reduce overall leg stiffness when joint stiffness is high, and allow an effective gait pattern – even with intrinsically stiff joints.

**Stability:** The flexed gait pattern, especially the flexion of the knee during stance, may even be more optimal than an upright posture in terms of stability. This can be imagined intuitively: if someone threatens to push you over, you will try to prevent this by bending your knees and hips. Several studies have shown that stability in standing position is increased when the knees are slightly flexed, possibly due to the lowering of the body’s center of mass (Pereira et al., 2008). Although speculative, a similar mechanism may increase overall stability when walking with flexed knees. The knee flexion may also give an additional degree of freedom to reject disturbances. Wagner and Blickhan (1999) showed that the stability of a two-segment leg model with Hill-type muscle models depended on the tuning between the joint geometry, the force-length and force-velocity relation, and intrinsic properties of the muscles. These may be altered in such a way that a flexed gait pattern can be more easily stabilized than an upright gait pattern in CP.

This illustration shows that in order to fully understand the origin of deviating gait patterns, a complete understanding of underlying factors as well as of the priorities to optimize for is necessary. The effects of spasticity, contractures, walking speed, and gait dynamics as described in this thesis, all represent a small part of this big ‘puzzle’.
8.4. Clinical implications

The research presented in this thesis was carried out using a translational approach, at the interface of fundamental and clinical research. It was aimed to shed light on the relationship between impairments, with a primary focus on spasticity, and gait deviations. By doing so, this thesis did not focus specifically on the (more fundamental) underlying processes of impairments, nor did it aim to develop directly clinically applicable tools or treatment methods. Yet, this research was inspired by questions arising from clinical practice, and we believe that the present thesis has yielded several insights that may be of importance to the clinic.

First, it was shown that dynamic spasticity can be measured during gait as the coupling between muscle-tendon stretch velocity and muscle activity. The ratio of gastrocnemius peak EMG activity and peak stretch velocity during swing as presented in Chapter 6 was a first specific example of how this measure could be used. The possible role of spasticity on the gait of a particular patient could thus be evaluated in this same manner, by studying the coupling between muscle-tendon stretch velocity and muscle activity. This may turn out to be an important measure to analyze in order to determine whether or not spasticity sincerely affects the gait and thus whether or not treatment should be focused on spasticity reduction. The addition of muscle-tendon lengths and velocity data during gait to a gait analysis report, preferably presented together with muscle activity graphs, would be a useful tool for the evaluation of possible dynamic spasticity effects for the individual patient.

Musculoskeletal modeling can also be useful in clinical decision-making and treatment evaluation in the case of muscle-lengthening surgery. The studies presented in this thesis add to the existing literature on short muscle length and slow muscle stretch velocity during gait, showing that affected muscles can, but need not necessarily, reach shorter length or slower lengthening velocity than normal during gait (Delp et al., 1996; Thompson et al., 1998; Arnold et al., 2006a). As shown by Arnold et al. (Arnold et al., 2006a), surgically lengthening of hamstrings muscles that are not short or slow during gait gives a higher risk of worsened anterior pelvic tilt after treatment, while lengthening of muscles that are short or slow has a higher chance of being successful. Furthermore, the present thesis emphasized that walking speed is an important aspect to consider especially when determining whether or not muscles are stretched at an abnormally slow rate. Therefore, muscle-tendon length and velocity, in combination with walking speed, are important factors to consider when planning orthopedic surgery. Yet, some caution should be taken when interpreting individual muscle-tendon length data, due to possible inter-individual differences in modeling results.

Walking speed was shown to have a large effect on muscle-tendon length and velocity during gait, as well as on joint kinematics and gait classification. This is important to consider when analyzing gait data, especially when comparing gait data between subjects or between different gait analyses of the same subject. Deviations in muscle-tendon length patterns of gastrocnemius and soleus, and the degree of equinus gait and knee (hyper)extension in
stance were shown to increase with increasing walking speed. This was (at least partly) due to the enhanced velocity-dependent effects of spasticity at faster walking speed. Comfortable walking speed may therefore be limited to prevent further gait deviations to occur, in order to prevent increased loading on muscles, joints, and bones, or to prevent excessive balance problems or increased energy cost. Increasing walking speed per se may thus not always be a good treatment goal, when not intervening in the underlying causes. At the same time, reducing spasticity by specific interventions may automatically improve walking speed, by reducing the limitations caused by spasticity on muscle-tendon stretch velocity.

Dynamic causes of gait deviations, as illustrated in Chapter 7 of this thesis, should also be kept in mind, as an alternative explanation for gait deviations next to disturbed muscle functioning. Specifically, in cases of stiff-knee gait, possible alternative causes next to quadriceps over-activity should be considered, such as a crouched gait pattern, reduced push-off, or reduced hip flexion moment in (pre)swing.

8.5. Recommendations for further research

Muscle dynamics

In the present thesis, muscle-tendon lengths and velocities were studied as a first estimate for muscle behavior during gait. However, the underlying muscle dynamics are obviously more complex than that. Further study of the behavior of spastic muscles during gait is recommended. Specifically, the stretch reflex is evoked by stretch of the muscle belly rather than by stretch of the muscle-tendon complex. Therefore, study on muscle belly versus muscle-tendon length and velocities, and on the coupling between muscle belly stretch velocity and muscle activity during gait may give further insight into enhanced reflexes during gait in CP. Such a study of muscle belly length during gait could be performed in two ways. First, muscle belly and muscle tendon length could be measured directly, for example using ultrasound. This has been done previously in healthy subjects walking (Fry et al., 2006; Lichtwark et al., 2007) or stair climbing (Spanjaard et al., 2007). Although several methodological difficulties still exist with this approach (Benard et al., 2009), these studies showed that a large difference can occur between muscle-tendon and muscle belly length changes. Second, muscle belly length can be estimated using inverse dynamic computer simulations. In these simulations muscle force and muscle tendon and belly lengths can be estimated from the net joint torques, using assumptions for force sharing between synergistic muscles and a muscle model to calculate muscle dynamics. This approach has for example been used by Neptune et al. (2007) to study the neuromuscular demands of toe-walking. Challenging to these simulations is to make accurate assumptions of changes in muscle and tendon properties that may occur in CP, such as increased stiffness or altered force-length relationships. Yet, the approach also allows studying the possible effects of these altered muscle properties on modeled outcomes, for example on muscle fiber length changes or on force generating capacities of muscles during different walking patterns.
Clinical studies

Further study is recommended on quantitative measures of spasticity in passive tests, such as the instrumented SPAT (Van den Noort et al., 2008), or using system identification techniques (e.g. Van der Helm et al., 2002; De Vlugt et al., 2006). Such studies will increase our understanding of underlying principles of spasticity, and also allow a better distinction between different impairments, e.g. spasticity and intrinsic muscle properties. Furthermore, their outcomes could be compared to dynamic spasticity measures during gait, to increase our understanding of how impairment measures at rest relate to impairment measures during dynamic tasks such as walking.

The concept of dynamic spasticity also requires further investigation in terms of treatment evaluation. It can now be used as an outcome measure to study whether treatment aiming at reducing spasticity, e.g. selective dorsal rhizotomy or baclofen treatment, is effective during gait. This can also elucidate whether the treatment works as expected, i.e. whether it reduces the coupling between muscle-tendon stretch velocity and muscle activity during gait.

Moreover, further study is recommended on a large set of patient data. This would make it possible to perform multivariate analyses in which more of the possible underlying causes of short muscle length and slow stretch velocity during gait could be included (e.g. spasticity, contractures, intrinsic muscle stiffness, muscle strength, selectivity), as well as further subgroup analyses. Such a study could also give further insight into the relationship between such short or slow muscles during gait and (typical) gait deviations; as well as into possible compensation strategies to walk with short or slow muscles, especially at fast speed.

Forward dynamic simulation

Forward dynamic modeling studies are recommended to further elucidate on cause and effect relationships between impairments and gait deviations. In this thesis, insight into the (relative) role of spasticity, contractures, and walking speed was gained using inverse kinematic musculoskeletal modeling. Although valuable insight was gained in this manner, it remains difficult to unquestionably conclude on cause and effect relationships. The next step is to use computer modeling for forward analysis, which can more directly establish this relationship.

A first step with forward dynamic modeling was made in Chapter 7 of this thesis using a passive dynamic walking-based model. Although such models do not cover all characteristics of human walking, they give insight into specific aspects of gait, starting from the simplest model of human gait, by gradually increasing the complexity of model. Further conceptual understanding of pathological gait could be gained using such relatively simple models. For example, possible advantages of toe and/or crouch walking could be studied, in terms of control (it may be easier to control a limb when all joints are in flexion, especially when selective control is limited), or in terms of optimizing total leg stiffness (flexed gait may be a way to reduce overall leg stiffness when joint stiffness is high, resulting in more ‘optimal’ gait).
In addition, forward dynamic simulation using more complex musculoskeletal models is a promising approach to study the effect of specific impairments or of specific therapeutic interventions on the gait of (individual) patients. Recent developments in musculoskeletal models, simulation techniques, and computational capacity now allow forward simulation within the context of clinical practice. For example, OpenSim (Delp et al., 2007) is an open-source platform for forward dynamic simulation and analysis of the neuromusculoskeletal system, operating on data that are acquired during instrumented gait analysis in the gait laboratory. This kind of modeling can be applied to simulate the effect of (surgical) intervention on the gait pattern. The simulated outcomes can then be compared to actual treatment outcomes, in order to test the accuracy of model simulations and their potential to assist in treatment planning.

For successful implementation of these models for individual patients in the clinical setting, improvement of musculoskeletal models is recommended. This will allow better prediction of muscle-tendon lengths and forces in individual subjects. Specifically, a model based on pediatric data, and more knowledge on the effects of individual bony variations or deformities on muscle moment arm calculations is expected to greatly improve the accuracy of calculations. In addition, in order to build more accurate muscle models for both inverse and forward dynamic patient gait simulations, further investigation of altered muscle properties in CP is necessary.

8.6. General conclusions

- **Spasticity** can be recognized during gait in children with cerebral palsy:
  - Spastic muscles are stretched slower during gait than non-spastic muscles and reach shorter peak lengths, even when controlling for walking speed.
  - An increased coupling exists between stretch velocity and muscle activity in spastic muscles during gait, which increases with walking speed.

- To investigate the effect of spasticity during gait of (individual) patients, muscle-tendon stretch velocity and muscle activity, as well as the coupling between the two should be assessed.

- **Muscle contractures** mainly affect the (peak) length at which a muscle operates during gait, but not the velocity with which it is stretched.

- **Walking speed** affects muscle-tendon length and velocity, as well as joint kinematics and gait classifications. The worsening of gait deviations with walking speed can (partly) be attributed to the velocity-dependent effect of spasticity, and may limit comfortable walking speed. Therefore, walking speed is important to consider when analyzing gait data.
Dynamic effects of a crouched pattern lead to reduced knee flexion in swing. Dynamic effects are important to consider as possible causes of gait deviations, next to abnormal local muscle functioning.

Musculoskeletal modeling gives insight into the role of impairments during gait, especially when impairments lay at the muscle or muscle-tendon level. The use of musculoskeletal modeling is recommended to assist in clinical decision making and treatment evaluation, for example in the case of spasticity treatment and muscle-lengthening surgery.

Further study using forward dynamic modeling techniques is recommended, to gain a better understanding of cause and effect relationships between impairments and gait deviations that are difficult to prove with experimental studies alone.
References


References

References


References


Summary

Gait deviations in children with cerebral palsy: a modeling approach

The gait pattern of children with cerebral palsy (CP) can be affected by many different underlying impairments, such as spasticity, contractures, weakness, and limited selective motor control. In combination with compensation strategies, that allow an optimal walking pattern despite these impairments, and considering the complex dynamics of human gait, it becomes difficult to determine the underlying causes of a specific gait deviation. Yet, a good understanding of these causes is essential in order to determine the best treatment for a patient. The aim of this thesis was to gain insight into the underlying causes of gait deviations; with a specific focus on the role of spasticity during gait, the interplay of spasticity with walking speed and muscle contractures, and possible dynamic causes of gait deviations.

Musculoskeletal modeling was used to study the gait pattern at the level of the impairment, i.e. to investigate muscle-tendon lengths and velocities during gait. In order to unravel the specific effects of spasticity, contractures, walking speed, and dynamics on the gait disorder, this technique was combined with the use of healthy subjects as a model (Chapter 3), modulation of spasticity effects by varying walking speed (Chapter 4-6), measurement of muscle activity during gait (Chapter 6), and forward dynamic simulation (Chapter 7).

Chapter 2 comprises a validation study, in which three models used to estimate hamstrings length were evaluated, i.e. M1: SIMM (Delp et al., 1990), M2: the Twente Lower Extremity Model (Klein Horsman et al., 2007), and M3: the model by Hawkins and Hull (1990). As a measure of accuracy, it was determined whether the estimated peak semitendinosus, semimembranosus, and biceps femoris long head lengths, as measured in eight healthy subjects, were constant over a range of hip and knee angles. It was found that the estimated peak hamstrings length depended on the model that was used, even with length normalized to length in anatomical position. M3 estimated shorter peak lengths than M1 and M2, showing that more advanced models (M1 and M2) are more similar. Peak hamstrings length showed a systematic dependence on hip angle for the biceps femoris in M2, and for the semitendinosus in M3, indicating that either the length was not correctly estimated, or that the specific muscle did not limit the movement. Considerable differences were found between subjects. It was concluded that modeling results for individual subjects should be interpreted with caution. Yet, no systematic deviations were found for M1 (the SIMM model)
indicating that this model is appropriate to be used for group comparisons. This model was subsequently used in Chapter 3-6.

**Chapter 3** explored how muscle-tendon length and lengthening velocity of hamstring and psoas muscles change as a result of crouch gait and as a result of walking speed. Eight healthy female subjects walked on a treadmill both normally and in crouch. In the crouch condition, subjects walked at three different walking speeds. Walking in crouch resulted in shorter psoas length compared to normal, but not in shorter hamstrings length. Moreover, crouch gait did not result in slower muscle-tendon lengthening velocities compared to normal gait. Contrarily, decreasing walking speed did reduce muscle-tendon lengths and lengthening velocities. These results do not support the role of hamstrings shortness or spasticity in causing crouch gait. Patients with short or spastic muscles may thus be more likely to respond by walking slower than by walking in crouch. The results also indicated that differences in walking speed should be avoided as a confounding factor when comparing patient groups with controls.

**Chapters 4, 5, and 6** describe the results of a set of experiments in which 17 children with CP and 11 matched typically developing children participated. The children walked in the gait laboratory at comfortable, slow, and fast walking speed, while 3D kinematic and electromyographic data were collected. All children with CP underwent a standard physical examination, in which spasticity and muscle contractures were tested. Muscle-tendon lengths and velocities during gait were calculated using the SIMM musculoskeletal model.

**Chapter 4** describes the effect of walking speed and spasticity on hamstrings length and velocity during gait. For any given walking speed, spastic hamstrings muscles acted at considerably shorter length and slower lengthening velocity during gait than normal, while non-spastic hamstrings in CP did not. Furthermore, peak hamstrings length increased slightly with walking speed, while peak lengthening velocity increased strongly with walking speed. The results indicate that the presence of spasticity is associated with reduced hamstrings length and lengthening velocity during gait, even when controlling for walking speed. Comparing the results of Chapter 3 and 4, it was discussed that the short length and slow velocity of hamstrings muscles in the children with CP likely resulted from a different cause than a crouched posture alone, such as increased posterior pelvic tilt or short step length.

In **Chapter 5**, gastrocnemius and soleus length and lengthening velocity during gait were investigated in spastic muscles with and without static contractures compared to non-spastic muscles. This study again included the effect of walking speed, and the interacting effect of walking speed and spasticity on muscle-tendon length and lengthening velocity. Spastic calf muscles showed a deviating muscle-tendon length pattern with two peaks in stance, which was found to be irrespective of muscle contracture. This deviating pattern became more pronounced as walking speed increased. In swing, spastic calf muscles were stretched approximately one third slower than normal, while in stance, spastic calf muscles were stretched twice as fast as normal, with peak velocity occurring earlier in the gait cycle. The increasingly deviating muscle-tendon length pattern at faster walking speed indicates a
velocity-dependent spasticity effect. This impairs walking especially at faster speeds, and may therefore limit comfortable walking speed.

Based on the concept of spasticity, stretch of spastic muscles during gait can be expected to lead to excessive muscle activity compared to control muscles. In Chapter 6 this dynamic spasticity, i.e. the coupling between muscle-tendon stretch velocity and muscle activity during gait, was evaluated for the gastrocnemius and soleus muscles. In typically developing children, muscles were stretched fast in swing without subsequent muscle activity, while in spastic muscles the slower stretch in swing (as reported in Chapter 5) was followed by an increase in muscle activity. The average ratio between peak activity and peak stretch velocity in swing was approximately four times higher in spastic muscles, and increased with walking speed. In stance, the stretch of muscles in typically developing children was followed by an increase in muscle activity. Spastic muscles were stretched fast in loading response, but since muscle activity was already built up in swing, no clear dynamic spasticity effect was present. It was concluded that spastic calf muscles showed an increased coupling between muscle-tendon stretch velocity and muscle activity especially during the swing phase of gait, which increased with walking speed.

Finally, in Chapter 7 forward dynamic simulation was used to study the effect of a crouched posture, as well as the effects of push-off strength and hip torque, on knee flexion in swing, as possible causes for stiff-knee gait. We developed a simple dynamic walking model of human gait, with a passive knee in swing. The model was powered by an instantaneous push-off impulse under the trailing leg. It produced stable limit cycle gait patterns for a range of stance leg knee flexion (crouch) angles. The effect of crouch angle on knee flexion in swing was evaluated, as well as the influence of push-off impulse size and the addition of a spring-like hip torque on knee flexion in swing. In upright posture, the model showed sufficient knee flexion and clearance in swing. When increasing the crouch angle of the model, the knee flexed much less in swing, resulting in a ‘stiff-knee’ gait pattern and reduced clearance. The decreased knee flexion in swing could be explained by the passive dynamics of the model’s swing leg due to differences in position of the leg at swing initiation. Increases in push-off impulse size and hip torque led to more knee flexion in swing, but the effect of crouch angle on swing leg knee flexion and clearance remained. These findings demonstrate that decreased knee flexion in swing can occur purely as a result of crouch, without any differences in actuation. This suggests that a stiff-knee gait pattern may result from uncontrolled dynamics of the system, rather than from altered muscle function or pathoneurological control alone.

All in all, the work presented in this thesis shows that spasticity can be recognized during gait in children with CP, when evaluating the gait pattern at the level of the impairment using musculoskeletal modeling. Walking speed affects muscle-tendon length and velocity, as well as joint kinematics. This can (partly) be attributed to the velocity-dependent effect of spasticity, and may limit comfortable walking speed. It is recommended that, in order to investigate the effect of spasticity on the gait of (individual) patients, muscle-tendon stretch velocity and muscle activity, as well as the coupling between the two should be evaluated.
Furthermore, the effects of walking speed and possible dynamic effects are important to consider when analyzing gait data, next to the effects of local muscle functioning. Further study using forward dynamic modeling techniques is recommended, especially to gain a better understanding of cause and effect relationships between impairments and gait deviations that are difficult to prove with experimental studies alone.
Samenvatting

Loopafwijkingen bij kinderen met cerebrale parese:
een modelmatige aanpak

Cerebrale parese (CP) is een houdings- of bewegingsstoornis als gevolg van een hersenbeschadiging, ontstaan voor het eerste levensjaar. CP komt voor bij ongeveer 2 op de 1000 kinderen en is daarmee de belangrijkste bewegingsstoornis bij kinderen in Europa. Het looppatroon van kinderen met CP is vaak afwijkend. Dit kan veroorzaakt worden door veel verschillende onderliggende stoornissen, zoals spasticiteit (een verhoogde activiteit van spieren op het moment dat deze gerekend worden), spiercontracturen (verkortingen), spierzwakte en beperkte selectieve controle van spieren. Door dit grote aantal factoren is het vaak moeilijk de precieze oorzaak van de loopafwijking te bepalen. Bovendien kunnen compensatiestrategieën optreden, die noodzakelijk zijn om optimaal te kunnen lopen ondanks deze stoornissen. Daarnaast is het menselijk lopen zelf een complexe dynamische taak.

Voor het bepalen van de beste behandeling voor de patiënt is het echter essentieel om de onderliggende oorzaken van een loopafwijking goed te begrijpen. Het doel van dit proefschrift is dan ook om meer inzicht in deze oorzaken. Het onderzoek in dit proefschrift richt zich in het bijzonder op de rol van spasticiteit tijdens het lopen. Daarnaast wordt ook gekeken naar de wisselwerking tussen spasticiteit, loopsnelheid en spiercontracturen; en naar hoe verschillende loopafwijkingen elkaar kunnen beïnvloeden als gevolg van de dynamica van de loopbeweging.

Deze onderliggende oorzaken zijn onderzocht met behulp van spierskeletmodellen. Dit zijn computersimulaties waarmee de lengte van een spierpeescomplex (spierpeeslengte) en spierpees verlengings- en verkortingsnelheid tijdens het lopen berekend kunnen worden. Gekoppeld aan experimentele meetgegevens van patiënten kan hiermee het lopen op spierniveau worden bestudeerd, het niveau waarop veel van de stoornissen optreden. Om de effecten van spasticiteit, spiercontracturen en loopsnelheid verder te ontrafelen, is tevens gebruik gemaakt van gezonde proefpersonen die het afwijkende looppatroon van patiënten nábouwen (hoofdstuk 3); is de loopsnelheid geverifieerd (hoofdstuk 3-6); en is spieractiviteit gemeten tijdens het lopen (hoofdstuk 6). Tevens is gebruik gemaakt van voorwaarts dynamische simulaties. Dit zijn simulaties waarbij een model van het lopen in de computer aangestuurd wordt en het effect van een verandering in aansturing of modelparameet op het looppatroon voorspeld kan worden (hoofdstuk 7).

Hoofdstuk 2 beschrijft een validatiestudie, waarin drie spierskeletmodellen voor het schatten van hamstringslengte geëvalueerd werden: M1: SIMM (Delp et al., 1990), M2: het ‘Twente...
Lower Extremity Model’ (Klein Horsman et al., 2007) en M3: het model van Hawkins and Hull (1990). Als maat voor nauwkeurigheid werden de maximale lengtes van de drie koppen van de hamstrings (de m. semitendinosus, de m. semimembranosus en de m. biceps femoris caput longum; alle heupstrekkers en kniebuigers) berekend voor een groep proefpersonen en is gekeken in hoeverre deze lengte constant was over een range van heup- en kniehoeken. De geschatte hamstringslengte bleek systematisch afhankelijk te zijn van het gebruikte model. M3 schatte kortere lengtes dan M1 en M2, terwijl de geavanceerder modellen (M1 en M2) meer gelijke resultaten lieten zien. Maximale hamstringslengte hing systematisch af van de heuphoek voor de m. biceps femoris in M2 en voor de m. semitendinosus in M3. Dit betekent dat de lengte ofwel niet correct werd geschat, ofwel dat de specifieke spier de beweging niet beperkte. Er werden aanzienlijke verschillen gevonden tussen proefpersonen. Geconcludeerd kon worden dat individuele modeluitkomsten met een zekere voorzichtigheid geïnterpreteerd moeten worden. Voor M1 werden geen systematische verschillen gevonden, wat erop duidt dat dit model het meest bruikbaar is voor vergelijkingen op groepsniveau. Dit model is gebruikt in hoofdstuk 3 tot en met 6.

**Hoofdstuk 3** beschrijft de verandering van spierpeeslengte en spierpeessnelheid van de hamstrings en de m. psoas (heupbuiger) als gevolg van een gehurkt looppatroon (of ‘crouch’-gang) en als gevolg van loopsnelheid. Hiervoor liepen acht gezonde proefpersonen op een loopband, zowel normaal als in crouchgang. Bovendien liepen zij in de crouchhouding op drie verschillende loopsnelheden. De crouchhouding leidde tot kortere psoaslengte vergeleken met normaal lopen, maar niet tot kortere hamstringslengte. Bovendien resulteerde de crouchhouding in langzamere rek van het spierpeescomplex van psoas of hamstrings vergeleken met normaal lopen. Een lagere loopsnelheid resulteerde echter wel in kortere spierpeeslengte en langzamere rek van deze spieren. Deze resultaten zijn geen ondersteuning voor de hypothese dat verkorte of spastische hamstrings een rol spelen bij crouchgang. Op basis van deze resultaten is het waarschijnlijker dat patiënten met korte of spastische hamstrings langzamer gaan lopen in plaats van dat zij in crouchgang gaan lopen. De sterke variatie van uitkomsten met loopsnelheid laat ook zien dat het belangrijk is rekening te houden met verschillen in loopsnelheid, bijvoorbeeld wanneer patiënten worden vergeleken met gezonde controleproefpersonen.

De **hoofdstukken 4, 5 en 6** beschrijven de resultaten van een serie experimenten waaraan 17 kinderen met CP en 11 vergelijkbare gezonde controlekinderen deelnamen. Deze kinderen liepen in het looplabatorium op comfortabele (zelfgekozen) loopsnelheid, extra lage en extra hoge loopsnelheid. Ondertussen werden 3D kinematica (bewegingsregistratie) en electromyografische data (spieractiviteit) verzameld. Alle kinderen met CP ondergingen ook een standaard lichamelijk onderzoek, waarin spasticiteit en spiercontracturen werden getest.

**Hoofdstuk 4** beschrijft de effecten van loopsnelheid en spasticiteit op hamstringslengte en -snelheid tijdens het lopen. Voor elke willekeurige loopsnelheid hadden de spastische hamstrings (gekeken is naar de m. semitendinosus) een aanzienlijk kortere lengte en een lagere maximale reknelheid tijdens het lopen dan bij gezonde controlekinderen, terwijl dit bij niet-spastische hamstrings bij kinderen met CP niet het geval was. De maximale
hamstringslengte nam licht toe met loopsnelheid, terwijl vooral de maximale hamstringsreksnelheid sterk toenam met loopsnelheid. Deze resultaten laten zien dat de aanwezigheid van spasticiteit samengaat met afgenomen hamstringslengte en reksnelheid tijdens lopen, zelfs als rekening wordt gehouden met loopsnelheid. Als de resultaten van hoofdstuk 3 en 4 vergeleken worden, dan blijkt dat deze korte en langzame hamstrings bij kinderen met CP waarschijnlijk het gevolg zijn van andere factoren dan een crouchhouding alleen, zoals toegenomen achteroverkanteling van het bekken of een kleine stappgrootte.

In hoofdstuk 5 werden de lengte en reksnelheid van de lange en korte kuitspieren (m. gastrocnemius en m. soleus) tijdens lopen onderzocht voor spastische spieren met en zonder contracturen en vergeleken met niet-spastische controlespieren. Deze studie bekeek wederom het effect van loopsnelheid en het interactie-effect van spasticiteit en loopsnelheid op spierpeeslengte en -snelheid. Spastische kuitspieren lieten een afwijkend patroon zien in de spierpeeslengte, met twee pieken in de standfase in plaats van één bij niet-spastische spieren. Dit afwijkende patroon was hetzelfde in spieren met en zonder contractuur en werd sterker naarmate de loopsnelheid toenam. Tijdens de zwaai van het been werden de spastische spieren ongeveer een derde langzamer gerekt dan normaal. In de standfase werden ze juist twee keer zo snel gerekt als normaal, waarbij de piek eerder in de gangcyclus optrad. De toenemende afwijking in het patroon van de spierpeeslengtecurve duidt op een snelheidsafhankelijk effect van spasticiteit. Dit zou het lopen vooral op hogere loopsnelheid kunnen bemoeilijken en daardoor de comfortabele loopsnelheid beperken.

Op basis van de definitie van spasticiteit, een (rek)snelheidsafhankelijke verhoogde activiteit van spieren, kan verwacht worden dat de rek van spastische spieren tijdens het lopen leidt tot excessieve spieractiviteit en ook interactie-effect van spasticiteit en loopsnelheid op spierpeeslengte en -snelheid. Spastische kuitspieren lieten een afwijkend patroon zien in de spierpeeslengte, met twee pieken in de standfase in plaats van één bij niet-spastische spieren. Dit afwijkende patroon was hetzelfde in spieren met en zonder contractuur en werd sterker naarmate de loopsnelheid toenam. Tijdens de zwaai van het been werden de spastische spieren ongeveer een derde langzamerペット

In hoofdstuk 6 wordt de lengte en reksnelheid van de lange en korte kuitspieren (m. gastrocnemius en m. soleus) tijdens lopen onderzocht voor spastische spieren met en zonder contracturen en vergeleken met niet-spastische controlespieren. Deze studie bekeek wederom het effect van loopsnelheid en het interactie-effect van spasticiteit en loopsnelheid op spierpeeslengte en -snelheid. Spastische kuitspieren lieten een afwijkend patroon zien in de spierpeeslengte, met twee pieken in de standfase in plaats van één bij niet-spastische spieren. Dit afwijkende patroon was hetzelfde in spieren met en zonder contractuur en werd sterker naarmate de loopsnelheid toenam. Tijdens de zwaai van het been werden de spastische spieren ongeveer een derde langzamerペット

In hoofdstuk 6 is deze ‘dynamische spasticiteit’, dus de koppeling tussen spier-pees reksnelheid en spieractiviteit, onderzocht voor de kuitspieren. Bij gezonde kinderen werden deze spieren snel gerekt tijdens de zwaaifase van het lopen, zonder dat dat gevolgd werd door een toename in spieractiviteit. In spastische spieren daarentegen werd de langzamere rek in de zwaaifase (zie ook hoofdstuk 5) wel gevolgd door een toename in spieractiviteit. De gemiddelde verhouding tussen maximale spieractiviteit en maximale reksnelheid in de zwaaifase was ongeveer vier keer zo hoog in spastische spieren en nam toe met loopsnelheid. In de standfase werd ook bij de gezonde kinderen de rek van de kuitspieren gevolgd door een toename in spieractiviteit. Bij de kinderen met CP werden de spastische spieren snel gerekt in stand, maar doordat de spieractiviteit al was opgebouwd in de zwaaifase was er geen duidelijk effect van dynamische spasticiteit zichtbaar. Spastische kuitspieren lieten dus een toegenomen koppeling zien tussen reksnelheid en activiteit, voornamelijk tijdens de zwaaifase van het lopen, die toenam met loopsnelheid.

Ten slotte werden in hoofdstuk 7 voorwaarts dynamische simulatietechnieken gebruikt om het effect van een crouchhouding, van afzetkracht en van een heupmoment op de mate van knieflexie tijdens de zwaaifase te bestuderen. Dit zijn namelijk mogelijke oorzaken voor ‘stiff-knee gait’, een looppatroon bij CP met verminderde knieflexie in de zwaaifase. Er werd een relatief eenvoudig dynamisch-lopend computermodel van het menselijk lopen
ontwikkeld, met een passieve kniebeweging in de zwaai fase, waarmee deze factoren op een conceptuele manier onderzocht konden worden. Het model werd aangedreven door een instantane afzetimpuls onder de achterste voet, en liet een stabiel cyclisch looppatroon zien voor een reeks van opgelegde kniehoeken voor het standbeen (de 'crouchhoeken'). Het effect van crouchhoek op de knieflexie in de zwaai werd geëvalueerd, net als de invloed van afzetkracht (gemodelleerd als een instantane impuls) en de toevoeging van een heupmoment (gemodelleerd als een veer). Als het model met gestrekte knie liep, dan liet het voldoende knieflexie in de zwaai fase zien zonder met het zwaaibeen de grond te raken (dus met voldoende 'clearance' van het zwaaibeen). Als de crouchhoek van het standbeen toenam, nam de knieflexie in de zwaai fase sterk af, resulterend in een 'stiff-knee' gangpatroon en afgenomen clearance. Deze afgenomen knieflexie in de zwaai fase kon verklaard worden vanuit de passieve dynamica van het zwaaibeen, als gevolg van de veranderde positie van het been bij het begin van de zwaai fase. Een toename van de afzetimpuls en een toevoeging van een heupmoment leidden beide tot meer knieflexie in de zwaai, waarbij het effect van crouchhoek op de mate van knieflexie en clearance in zwaai bleef bestaan. Deze bevindingen laten zien dat afgenomen knieflexie in de zwaai kan ontstaan puur als gevolg van een crouchhouding, zonder verandering in aansturing. Dit duidt erop dat een 'stiff-knee' gangpatroon het resultaat kan zijn van de dynamica van het systeem en niet het gevolg hoeft te zijn van een veranderde spierfunctie of van verstoorde spiercontrole alleen.

Samenvattend laat dit proefschrift zien dat de effecten van spasticiteit gemeten kunnen worden tijdens het lopen, als het looppatroon op het niveau van de stoornis, dus op spierniveau, geëvalueerd wordt met behulp van spierskeletmodellering. Voetafwaaihouding beïnvloedt de spierpeeslengte en -snelheid. Dit kan (deels) worden toegeschreven aan snelheidsafhankelijke effecten van spasticiteit en zou de comfortabele loopsnelheid kunnen beperken. Voor het bepalen van de effecten van spasticiteit tijdens het lopen bij (individuele) patiënten is het belangrijk om de spierskeletstatus, de spieractivatie en de koppeling tussen deze beide te bekijken. Verder moet bij het bestuderen van gangbeelddata rekening worden gehouden met de loopsnelheid en mogelijke dynamische effecten, naast de specifieke spierfuncties. Verdere studie met behulp van voorwaarts-dynamische modellering wordt aangeraden, in het bijzonder om meer inzicht te krijgen in oorzaak-gevolg relaties tussen onderliggende stoornissen en loopafwijkingen bij kinderen met CP.
About the author

Marjolein van der Krogt was born on August 16, 1979 in Leiderdorp, the Netherlands. She grew up in Zoeterwoude and attended the Bonaventuracollege in Leiden, where she graduated cum laude for her gymnasium in 1997. Afterwards, she studied Human Movement Sciences at the VU University in Amsterdam. During her master studies she performed an internship at the Locomotion Lab of the University of Colorado in Boulder, supported by a Gerrit Jan van Ingen Schenau Promising Young Scientist Award. She graduated cum laude in 2004. In the same year, she started her PhD in Rehabilitation Medicine at the VU University Medical Center, resulting in the present thesis. In May 2008 she (co-)organized the Dynamic Walking scientific conference in Delft, the Netherlands. After her PhD she will start a post-doc project at Gillette Children’s Specialty Healthcare in Minneapolis, in collaboration with Stanford University, funded by a Ter Meulen Fund stipendium. Apart from her research work, she enjoys bicycling, speed-skating, hiking, playing the saxophone, and spending time with family and friends.

Publications in international journals

Conference proceedings


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Dankwoord

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Dankwoord

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