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.