Spastic cerebral palsy (CP) is characterized by increased joint resistance, caused by a mix of increased tissue stiffness, as well as involuntary reflex and background muscle activity. These properties can be quantified using a neuromechanical model of the musculoskeletal complex and instrumented assessment. The construct validity of the neuromechanical parameters was examined (i.e. the internal model validity, effect of knee angle, speed and age, sensitivity to patients versus controls, spasticity severity and treatment), together with the repeatability. We included 38 children with CP and 35 controls. A motor driven footplate applied two slow (15 °/s) and two fast (100 °/s) rotations around the ankle joint, at two different knee angles. Ankle angle, torque and EMG of the gastrocnemius (GA), soleus (SO) and tibialis anterior (TA) muscle were used to optimize a nonlinear neuromuscular model. Outcome measures were tissue stiffness, reflex and background activity for GA, SO and TA. The internal model validity showed medium to high parameter confidence and good model fits. All parameter could discriminate between patients with CP and controls according to CP pathology. Other measures of external model validity (effect of test position, speed and age) showed behavior along the lines of current knowledge of physiology. GA/SO background activity was sensitive to spasticity severity, but reflex activity was not. Preliminary data indicated that reflex activity was reduced after spasticity treatment. The between-trial and -day repeatability was moderate to good. The large variance between patients in the ratio of stiffness and neural resistance indicates that the method could potentially contribute to patient-specific treatment selection.

Introduction

Spastic cerebral palsy (CP) is characterized by increased joint resistance to motion, which is caused by neural as well as tissue impairments. Among the neural impairments are increased reflex activity (i.e. a velocity dependent increase in muscle tone or spasticity), co-contraction, and non-stretch related contractions (i.e. background activation). The tissue impairments comprise altered viscoelastic properties of connective tissues, muscle or tendon. The discrimination between neural and tissue impairments largely guides the selection of treatments that aim to reduce joint resistance. In case of suspected neural origin, muscle activation can be reduced by botulinum toxin (BTX-A) or selective dorsal rhizotomy (SDR), while suspected tissue impairments can be treated by corrective casting or splinting. Thus, objective quantification of neuromechanical joint parameters could contribute to patient specific treatment in CP.

Current clinical assessment of joint resistance is based on manual testing of the resistance to slow and/or fast movements, such as the Ashworth and Tardieu-like tests. These clinical tests do not allow for objective quantification, because they are found to be subjective, of low resolution, and limited in discriminating neural and tissue components. Instrumented manual measurements have been shown to result in increased precision. Nevertheless, these manual tests still lack standardization of exerted force and movement velocity, while spasticity is known to be force- and velocity dependent.

Neuromuscular modelling combined with motorized assessment has been demonstrated to quantitatively distinguish between reflex activity and tissue viscoelasticity around the ankle in stroke patients and patients with CP. However, previous models only tested in the dorsiflexion direction and did not include the tissue properties of the major antagonist (tibialis anterior), even though its field of activity could overlap with those of a shortened triceps surae muscle. In addition, they modelled the lumped properties of the triceps surae and were thus unable to distinguish between the contribution of the soleus and gastrocnemius. These models did not take any non-stretch related background activity into account. Finally, the validity of the model has not yet been established in young children with CP, but only in adolescents, while the impairments of these children worsen during growth and are thus often assessed and treated before the end of their growth.

Therefore, the aim of the study was to present an extended motorized assessment protocol (to separate between soleus and gastrocnemius) combined with an extended neuromuscular ankle model (including antagonists and estimation of background activity); and to assess the construct validity and repeatability of derived neural and tissue parameters. For construct validity, i.e. the extent to which the results correspond with current knowledge of the neuromuscular ankle system in CP by a lack of a true gold standard, we examined the internal model validity (parameter confidence and model fit), and external model validity (effect of two different test positions, movement speed, and age; patients versus controls; spasticity severity, and treatment).
Methods

We included a convenience sample of 38 children with CP (10.5±2.9 yr; 16 GMFCS-I, 16 GMFCS-II, 6 GMFCS-III; 18 male) and 35 typically developing children (10.1±2.7 yr, 15 male). Patients were included if they were between 6 and 18 years old, had a clinical diagnosis of spastic uni- or bilateral CP and excluded if they were not able to extend their knee to 20° knee flexion, had additional medical problems interfering with joint neuromechanical characteristics, or severe cognitive deficits interfering with participation in the study. Informed consent was provided and the study was approved by the local medical ethics committee.

Procedures

All children were measured at the ankle dynamometer. In a random subset of 29 patients (10.3±3.3 yr), the spasticity score (SPAT) was collected and subdivided into a low (SPAT 0 and 1, n=16) and high (SPAT 2+, n=13) spasticity severity group. Four patients (12.3±1.5 yr, GMFCS I-III) were measured before and after treatment (two received BTX-A in the gastrocnemius muscles and two underwent SDR). To assess repeatability, 12 patients (9.8±3.1 yr; GMFCS I-II) were measured on two occasions, 9.0±7.3 days apart, without any treatment in between.

Instrumentation

Subjects were seated in an adjustable chair, with a fixed 120° hip angle and the knee adjusted to 20° and 70° flexion (Fig. 2-1). In patients, the foot of the most affected leg was fixed in an adjustable foot plate that allowed for talus repositioning, i.e. correction of ab/adduction and pro/supination of the forefoot with respect to the talus and calcaneus for an optimal fixation. In controls, the right foot was fixed in a rigid foot plate. The foot plates were motor driven and applied rotations around the ankle joint (MOOG, Nieuw Vennep, The Netherlands). The axes of the ankle (talo crural joint) and the motor were visually aligned by minimizing knee translation during rotation. EMG electrodes (Ø 15mm, 24mm inter-electrode distance) were placed on the tibialis anterior (TA), soleus (SO), gastrocnemius medialis and lateralis (together GA) muscles according to SENIAM guidelines. Angular displacement, foot reaction torque and muscle activity (Porti7, TMSi B.V., The Netherlands) were measured at 1024 Hz. EMG was high-pass filtered (bidirectional 3rd order Butterworth at 20 Hz), rectified and low-pass filtered (unidirectional 4th order Bessel filter at 20 Hz) to obtain the envelope. The angle and torque data were identically low-pass filtered and all data were resampled to 128 Hz.

Measurement protocol

The ankle angle offset was calibrated by measuring the position of the foot plate corresponding to 30° ankle plantar flexion, which was determined by goniometry. The passive ankle range of motion (ROM) was determined by imposing an age- and disorder-dependent maximal flexion and extension torque (Appendix 2A). Next, two repetitions of ramp-and-hold rotations were imposed at 15 and at 100 °/s, starting
Neural vs Tissue

at a random time instant to prevent anticipation and with at least 20s rest between measurements. Subjects were instructed to remain relaxed and not resist any motion during the measurements. The measurements were performed at both 20° and 70° knee flexion, conform clinical testing \(^{18}\). The outcomes at these angles are from here on referred to as SO and GA/SO, because the soleus is expected to be the dominant muscle at 70° knee flexion, since the gastrocnemii are too short to generate force \(^{21}\). TA properties were included for both angles, but examined at 20° knee flexion.

**Neuromuscular model**

Ankle angle and EMG signals were cropped from start to stop of the dorsal and plantar flexion movement and used as an input to the nonlinear neuromuscular model. For each trial, the model estimated the passive stiffness and neural torques around the ankle by optimizing the quadratic difference between the modelled and recorded total ankle torque (Appendix 2B). This extended model comprised TA passive stiffness parameters (enabled by the inclusion of movement towards plantar flexion), relaxation
between the bidirectional movements, and age scaling of physiological parameters, while it did not include viscosity and inertia (Appendix 2B). A total of 10 model parameters were optimized, including four passive tissue parameters (two exponential shape parameters and muscle slack lengths), five neural parameters related to the activation dynamics (four EMG gains and the neural filter frequency), as well as an offset torque to account for the relaxation between the dorsal and plantar flexion curves (Appendix 2C).

The passive stiffness and neural torques estimated by the optimized model were used to determine the outcome parameters (Fig. 2-2). Tissue ‘stiffness’ was expressed as the limitation in ankle rotation due to passive resistance during the slow trials, being the flexion angle corresponding to 3 Nm of stiffness torque (dorsiflexion for TS and plantar flexion for TA), subtracted from the highest measured angles in all subjects (60° for TS and 80° for TA) for better comparison. Reflex and background activity were calculated from the fast trials. Background activity torque was the minimum muscle activation measured over the entire trial related to force via the Hill type muscle model (Appendix 2B). The reflex activity torque was the total neural torque subtracted by the background torque. Reflex and background activity were both parameterized by the root-mean-square (rms) of their corresponding torque. All modelling and analyses were performed in Matlab (The Mathworks Inc., Natick MA).
To examine the internal model validity, we determined the standard error of the mean (SEM) of the parameter estimation, with low values representing high parameter confidence. The goodness of the model fit was represented by the variance accounted for (VAF). VAF values lower than 90% were discarded from the analysis (a total of three trials) for possible poor fixation or misalignment of the motor and ankle. For external model validity, the effects of knee angle, movement velocity and age were examined using sign-rank tests and Spearman’s correlation coefficients. Median differences between patients and controls and between low and high spasticity groups was examined using rank-sum tests; differences in variance between patients and controls using Levene’s tests. The (preliminary) effect of spasticity treatment was evaluated using sign-rank tests. To determine the reliability for the patients, data were checked for any systematic difference using the sign rank tests. The between-trial and -day repeatability were examined using intra class correlation coefficients (ICC, 2-way mixed model), the standard error of measurement (SEM) and the smallest detectable difference (SDD). Statistics were performed using IBM SPSS statistics 20 and Matlab.

### Results

#### Internal model validity

SEM values were less than 0.1% for 6 out of 10 parameters, but were high for the slack length parameters of both TS and TA (125% and 40%), the neural filter frequency \( f_0 \) (19%) and the relaxation offset (104%) (Appendix 2C). Median VAF value was 98.7 (IQR = 1.3).

#### External model validity

Stiffness changed with knee angle, with a 9.8% increase in GA/SO compared to SO stiffness and a 4.5% decrease for TA at the same angles (both \( p < 0.001 \)). Reflex activity was 79.9% increased in GA/SO compared to SO (\( p < 0.001 \)). TA reflex activity and

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Muscle</th>
<th>TD</th>
<th>CP</th>
<th>CP vs. TD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stiffness (°)</td>
<td>SO</td>
<td>41.20 [15.26]</td>
<td>53.50 [16.90]</td>
<td>12.30 &lt;0.001</td>
</tr>
<tr>
<td></td>
<td>GA/SO</td>
<td>48.90 [16.85]</td>
<td>52.98 [11.80]</td>
<td>4.08 0.008</td>
</tr>
<tr>
<td></td>
<td>TA</td>
<td>33.85 [8.61]</td>
<td>28.05 [18.05]</td>
<td>-5.80 0.008</td>
</tr>
<tr>
<td>Reflex (Nm)</td>
<td>SO</td>
<td>0.15 [0.26]</td>
<td>1.51 [2.50]</td>
<td>1.36 &lt;0.001</td>
</tr>
<tr>
<td></td>
<td>GA/SO</td>
<td>0.26 [0.41]</td>
<td>1.61 [3.11]</td>
<td>1.35 &lt;0.001</td>
</tr>
<tr>
<td></td>
<td>TA</td>
<td>0.11 [0.46]</td>
<td>0.50 [1.11]</td>
<td>0.39 0.030</td>
</tr>
<tr>
<td>Background (Nm)</td>
<td>SO</td>
<td>0.28 [0.25]</td>
<td>0.65 [0.79]</td>
<td>0.38 &lt;0.001</td>
</tr>
<tr>
<td></td>
<td>GA/SO</td>
<td>0.35 [0.33]</td>
<td>0.76 [0.72]</td>
<td>0.41 &lt;0.001</td>
</tr>
<tr>
<td></td>
<td>TA</td>
<td>0.05 [0.05]</td>
<td>0.10 [0.37]</td>
<td>0.05 0.004</td>
</tr>
</tbody>
</table>

Table 2-1a. TD and CP outcome values with Mdn median, IQR interquartile range.
background activity of all muscles did not change with knee angle. Speed affected the neural components: with a 32.1% and 63.3% increase in SO and GA/SO reflex activity during the fast trial, and a 82.0% decrease in TA background activity (all \( p<0.002 \)). Stiffness of all muscles, TA reflex activity as well as SO and GA/SO background activity did not change with movement speed. Age was related to increasing stiffness of all muscles in the control population (SO: \( r=0.65, p<0.001 \); GA/SO: \( r=0.76, p<0.001 \); TA: \( r=0.40; p=0.02 \)) as was GA/SO background activity ( \( r=0.36, p=0.04 \)). In patients, age was less related to SO stiffness (SO: \( r=0.38, p=0.02 \)), but slightly more to TA stiffness and background activity ( \( r=0.64, p<0.001; r=0.36, p=0.03 \)).

In patients, stiffness was increased by 30% and 8% in SO and GA/SO compared to controls, while it was 17% decreased in TA (all \( p<0.01; \) Table 2-1). Reflex activity was 10.4, 6.3 and 4.6 times increased in SO, GA/SO and TA and background activity 2.4, 2.2 and 2.1 times (all \( p<0.03; \) Fig. 2-3A). Variances of TA stiffness as well as reflex and background activity of all muscles were larger in patients compared to controls (all \( p<0.007; \) Table 2-1). Ratios between stiffness and neural components differed considerably between patients and between SO and GA/SO (Fig. 2-3B). GA/SO background activity was 2.0 times increased in the high versus the low graded spasticity group ( \( p=0.01; \) Fig. 2-4A), with a trend of increased SO reflex and background activity (3.5 times, \( p=0.11; \) 1.5 times, \( p=0.08 \)). There was a trend of reduced SO and GA/SO reflex activity after treatment (70.0 %, \( p=0.13; \) 60.5 %, \( p=0.13 \); Fig. 2-4B).

### Repeatability

No significant differences were found between the two repetitions or days, except a 28% decrease in SO reflex activity between the trials on the first day ( \( p=0.02 \)). Between-trial repeatability was moderate to good (ICC: 0.76-0.99) except for TA reflex activity (ICC: 0.22; Table 2-1). SDD values were 2.6-3.4° for stiffness and 0.4-2.6 Nm for neural activity. Between-day repeatability was somewhat lower (ICC: 0.64-0.95), and even worse for TA and SO reflex activity (ICC: 0.33 and 0.44 respectively). SDD values were 7.1-18.8 ° for stiffness and 0.5-2.2 Nm for neural activity.

---

**Table 2-1b. Repeatability of the outcome values**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Muscle</th>
<th>Inter-trial</th>
<th>Between-day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>ICC</td>
<td>SEM</td>
</tr>
<tr>
<td>Stiffness (°)</td>
<td>SO</td>
<td>0.98</td>
<td>1.12</td>
</tr>
<tr>
<td></td>
<td>GA/SO</td>
<td>0.99</td>
<td>1.22</td>
</tr>
<tr>
<td></td>
<td>TA</td>
<td>0.98</td>
<td>0.95</td>
</tr>
<tr>
<td>Reflex (Nm)</td>
<td>SO</td>
<td>0.83</td>
<td>0.49</td>
</tr>
<tr>
<td></td>
<td>GA/SO</td>
<td>0.76</td>
<td>0.52</td>
</tr>
<tr>
<td></td>
<td>TA</td>
<td>0.22</td>
<td>0.59</td>
</tr>
<tr>
<td>Background (Nm)</td>
<td>SO</td>
<td>0.77</td>
<td>0.39</td>
</tr>
<tr>
<td></td>
<td>GA/SO</td>
<td>0.83</td>
<td>0.22</td>
</tr>
<tr>
<td></td>
<td>TA</td>
<td>0.78</td>
<td>0.13</td>
</tr>
</tbody>
</table>

with ICC intra class correlation coefficient, SEM standard error of measurement and SDD smallest detectable difference.
Fig 2-3. A) stiffness, reflex and background activity compared between patients with CP and controls. B) the contribution of tissue stiffness and neural activity between patients and among muscles, with the median values of the controls indicated in grey. Note that the neural activity is a combination of reflex and background activity and that the units on the y-axis differ.
Discussion

The aim of this study was to present a motorized assessment combined with an extended neuromuscular ankle model and to assess the construct validity and reliability of the derived neural and tissue parameters in children with CP.

Internal model validity

The SEM values were low for most parameters, indicating that these parameters were all required to describe the measured joint torque. The high VAF values demonstrated that the measured torque could be well explained by the model, in line with previous versions of the model. However, SEM values were high for the neural filter frequency, relaxation offset and slack length parameters. The filter frequency could not be well determined in trials with limited neural activity, but the effect was small given the limited range over which it could vary. The offset parameter was a crude approach to capture relaxation, which worked well in most cases, however, it could be further optimized by including a time-dependent relaxation model. Capturing richer data sets, including active tasks, could further increase these parameters’ confidence.

External model validity

The effects of knee angle, movement speed and age were according to current anatomical knowledge. Reflex activity and stiffness were higher for GA/SO than SO, illustrating the knee angle dependency of the force generation of the gastrocnemii. TA reflex activity was insensitive to knee angle, as expected from a mono articular muscle, however the stiffness was slightly increased with a flexed knee, which was possibly due to extra muscular connectivities. As expected, the high velocity trials did provoke more reflex activity compared to the slow movement in both SO and GA, whereas the stiffness of all muscles was insensitive to movement speed. Background activity was insensitive to knee angle and movement speed for SO and GA/SO, however, it was found to be higher in TA at slow speed, possibly as a tendency to involuntarily assist dorsiflexion movement. In control children, the role of tissue stiffness increased with age, although this was not as evident for children with CP.

The method was able to discriminate between controls and patients with CP. The differences illustrate the characteristic pathology of CP: increased SO and GA stiffness, related to changes in the muscle fibre bundle properties and/or a lower sarcomere number, but decreased TA stiffness, which could be related to a lengthened TA tendon due to equinus deformity of the foot. In addition, we found increased GA, SO and TA reflex activity, corresponding to spasticity, and background activity, which is suggested to results from elevated muscle activation due to neural changes at the spinal motor neuron level. The combined values for reflex and background activity are in the same range as the reflex activity previously reported for a group of adolescents with CP. Next to an overall increase, the variances were also larger in patients compared to controls, which reflect the large heterogeneity of patients with CP. There was a large variability between patients and
among muscles in the contribution of stiffness and neural components to ankle joint resistance, which could not be explained by age differences, but might reflect different clinical phenotypes.

There was some indication of sensitivity to spasticity severity and treatment. Increased GA/SO background activity was found in the group with higher spasticity scores, but only a trend of increased GA/SO and SO reflex activity. Although the Ashworth spasticity score was previously found to correlate with modelled tissue stiffness and reflex activity, most instrumented tests show poor correlations with the clinical spasticity scores. Thus, the weak relation found for SO and GA/SO reflex activity might result from the inability of the subjective clinical scale to properly assess spasticity, rather than a lack of concurrent validity of the presented method. Preliminary results indicate that the method might be sensitive to spasticity reducing treatment, with reductions in SO and GA/SO reflex activity larger than the smallest detectable change in three out of the four patients. However, these findings should be interpreted with care, because of the small sample size, the lumping of different treatments, target locations and dosage, and further study is warranted.

Fig 2-4a. The relation between low disease grade (SPAT 0 and 1) and high grade (SPAT 2+), with control values for comparison.
Repeatability
There was no consistent learning effect between repetitions or day. The between-trial repeatability was moderate to good, and in the same order of magnitude as other instrumented assessments in patients with CP\textsuperscript{15,26}. The repeatability decreased between days for stiffness, which seemed in part due to variability in ankle angle calibration, but remained comparable for most neural parameters. This indicates that neither reflex nor background activity were largely affected by a change in ability to relax between days. The actual change that could be measured within one day (SDD-values) was smaller than the median difference between patients and controls for stiffness and SO reflex activity, but this does not hold for the between day SDD’s. This suggests that the method needs further standardization, although large between day variability has also been suggested to be a true phenomenon of spasticity and thus typical for patients with CP\textsuperscript{13}.

Limitations
Several simplifications were made in the model. Muscle length change was assumed to be linearly related to rotation of the foot. Especially in children with CP, however, foot deformations are frequently present that can affect this relation\textsuperscript{19}. We used a foot
plate that allowed for patient-specific fixation of the foot in patients, but the extent to which it really prevented foot deformations should be further examined. The rigid foot plate was used in the controls for practical reasons, since no systematic effects were found between the adjustable and rigid foot plate in healthy adolescents. In addition, the properties of the GA could not be determined separately, because both GA and SO are active with an extended knee. The subject specific scaling of some muscle properties could be improved, for it was based on linear scaling to the measured fibula length, and differed between patients and controls to account for muscle shortening in the optimal muscle length. Finally, the compliance of the Achilles tendon was not taken into account.

The outcome parameters were chosen to match the clinical measurements as much as possible. Originally we expressed stiffness as the change in moment around the maximal common dorsiflexion angle reached by all subjects, but we would have excluded the severe patients with limited range of motion, while the controls and less severe patients were still within their slack length. Therefore, stiffness was pragmatically defined as the limitation in ankle angle due to a certain stiffness torque. Future studies should focus on collecting rich data that allows for a distinction between mechanical muscle stiffness and muscle length shortening (Appendix 2B) and that is applicable in a heterogeneous population. This study included non-stretch related background muscle activity, but this may have included some noise signal as well. Although the background activity can be discriminated from noise based on its effect through the force-length relationship, some noise could still have been erroneously identified as background activity. Previous studies subtracted the rest EMG before applying the Hill type muscle model, thereby certainly discarding noise but also deleting meaningful signal. Further refinement of the model should thus focus on better separation of noise from background activity, for instance based on the force-length relationship of the latter. Finally, the reflex activity measure cannot differentiate involuntary reflex activation from intended muscle activation. This could be affecting individual measurements, especially in younger children who are less able to follow the instructions to relax, and should be taken into account in future studies.

Conclusion

A motorized measurement protocol combined with an extended neuromuscular model was presented. The internal model validity was satisfactory, with medium to high parameter confidence and good model fits. The approach allowed us to discriminate patients with CP from controls accordingly to CP pathology. Other measures of external model validity (effect of test position, speed and age) showed behavior along the lines of current knowledge of physiology. Sensitivity to spasticity severity and treatment showed expected behavior on some but not all measures, although a larger group should verify these findings. All parameters showed moderate to good repeatability. The large variance in ratio between stiffness and neural resistance between patients indicates that the method could potentially contribute to patient-specific treatment selection, pending follow up studies.
Acknowledgements

The authors would like to thank Mariska Adriaansen, Lizanne van den Akker, Anke van Erp, Ivo van Eibergen Santhagens, and Frans Simmes for their assistance with the measurements. This research was financially supported by the Dutch Technology Foundation STW (grant 10733).

References


### Appendix 2A. Maximum imposed ROM torques

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Plantar flexion (Nm)</th>
<th>Dorsiflexion (Nm)</th>
<th>Plantar flexion (Nm)</th>
<th>Dorsiflexion (Nm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-7</td>
<td>5</td>
<td>7</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>8-9</td>
<td>6</td>
<td>8</td>
<td>5</td>
<td>7.5</td>
</tr>
<tr>
<td>10-11</td>
<td>7.5</td>
<td>10</td>
<td>6</td>
<td>7.5</td>
</tr>
<tr>
<td>12-18</td>
<td>10</td>
<td>13</td>
<td>7.5</td>
<td>10</td>
</tr>
</tbody>
</table>

Maximum torques that were imposed during a slow trial to determine the passive ankle ROM.

### Appendix 2B. The neuromuscular model

The neuromuscular model was an extended version of the model by De Vlugt et al. In short, the model estimates the torque around the ankle due to passive muscle stiffness and neural activation by optimizing the quadratic difference between the modelled and recorded ankle torque. The model optimized a total of 10 parameters: four EMG gains, two exponential shape parameters and muscle slack lengths, the neural filter frequency and relaxation offset compensation. Compared to De Vlugt et al this version adds the passive tissue stiffness of the TA next to the stiffness of the triceps surae, it adds an offset torque to account for relaxation effects between...
the dorsal and plantar flexion movement, age scaling of physiological parameters, and does not include viscosity and inertia, both having only a small contribution to the measured torque. Because a bi-directional movement was applied with a hold period in between, the offset parameter was needed to account for the relaxation during the hold period, which causes a decrease in the torque measured at the start of the plantar flexion movement compared to the end of the dorsiflexion movement. Tendons were assumed infinitely stiff, as in the previous model 14.

The modelled ankle torque was described by:

$$T_{mod}(t) = (F_{tiss,tri}(x) + F_{neural,tri}(t)) \times r_{achil}(\theta) - (F_{tiss,ta}(x) + F_{neural,ta}(t)) \times r_{ta}(\theta) + T_{grav}(\theta) + T_{rel}$$  \hspace{1cm} (2.1)

with t time in seconds, x muscle length in m, \(\theta\) ankle angle in rad, \(T_{mod}\) total modelled ankle torque in Nm, \(F_{tiss}\) muscle force resulting from tissue stiffness in N, \(F_{neural}\) force resulting from muscle activation in N, subscript tri referring to the triceps surae (TS) muscle (gastrocnemius medialis, lateralis and soleus muscle) and subscript ta to the tibialis anterior (TA) muscle, \(r_{achil}\) and \(r_{ta}\) the moment arm of the Achilles tendon and tibialis anterior tendon in m, \(T_{grav}\) total torque due to gravity in Nm and \(T_{rel}\) a constant offset to compensate for tissue relaxation between dorsal and plantar flexion movement, which was limited to the average difference between these movements, in Nm.

The forces related to tissue stiffness (\(F_{tiss}\)) exponentially increased with ankle angle, describing the length-tension properties of ligamentous and muscular elastic tissues 14:

$$F_{stiff}(t) = e^{k(x(t) - x_0)}$$  \hspace{1cm} (2.2)

with k the shape factor for the exponential curve in 1/m, and \(x_0\) passive slack length of the muscle.

The forces resulting from muscle activation (\(F_{neural}\)) were estimated from the EMG signals as described by De Vlugt et al. 14. EMG gain parameters were optimized for the three TS and TA muscles, together with \(f_0\), the cut-off frequency of the neural activation filter. The optimal muscle lengths of TS and TA and the shape parameter for the force-length relationship are among the parameters of the Hill-type muscle model, which describe the relation between muscle force versus muscle length and velocity. Their values were chosen differently than in the previous model 14 and were set to 0.060 and 0.098 m and to 0.5, respectively, based on OpenSim (version 3.2, GAIT2392 model).

The total gravitation torque (\(T_{grav}\)) constituted of the gravitational contribution of the footplate and the foot itself. The gravitation torque of the footplate was measured, while the gravitation of the foot (\(T_{grav,foot}\)) was estimated as:

$$T_{grav,foot}(\theta) = m_{foot} g l_{com} \cos(\theta_{plate-hor} - \theta_{com-plate})$$  \hspace{1cm} (2.3)
with $m_{\text{foot}}$ as mass of the foot, $g$ as the gravitational acceleration ($9.81 \text{ m/s}^2$), $l_{\text{com}}$ as the distance of the center of mass from to the center of rotation, $\theta_{\text{plate-hor}}$ as the angle between the footplate and the horizontal in ° and $\theta_{\text{com-plate}}$ as the angle (set at 20 °) between the footplate and $l_{\text{com}}$. To take age and length differences into account, $m_{\text{foot}}$ was defined as 1.45% of body weight, and $l_{\text{com}}$ as the proportion of the subject’s distance (defined as 7.6% of the body height) compared to the adult distance (taken as 0.063 m, OpenSim).

The muscle moment arms and muscle lengths were derived from the recorded ankle angle, according to the relations in OpenSim:

$$r_{\text{achil}}(\theta) = (0.0468 - 0.0103 \theta - 0.0204 \theta^2 + 0.0033 \theta^3) \times s$$ 2.4

$$r_{\text{tib}}(\theta) = (0.0424 + 0.0110 \theta - 0.0195 \theta^2 - 0.0131 \theta^3) \times s$$ 2.5

$$x_{\text{tri}}(\theta) = (0.0515 + 0.0409 \theta + 0.0001 \theta^2 - 0.0085 \theta^3) \times s$$ 2.6

$$x_{\text{tib}}(\theta) = (0.0743 - 0.0412 \theta - 0.0019 \theta^2 + 0.0063 \theta^3) \times s$$ 2.7

The scale factor $s$ was introduced to scale the moment arm and muscle length from adult size to an appropriate size for young children. It was based on the proportion of the subject’s fibula length versus adult fibula length (0.35 m, OpenSim). The optimal muscle lengths used in the Hill-type muscle model were also scaled by this factor.

### Appendix 2C. Estimated model parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Optimization</th>
<th>Estimated value</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>$k_{\text{TS}}$</td>
<td>1/m</td>
<td>Stiffness coefficient TS</td>
<td>Initial: 250, Min: 50, Max: 700; Median: 167.457, IQR: 75.370</td>
</tr>
<tr>
<td>$k_{\text{TA}}$</td>
<td>1/m</td>
<td>Stiffness coefficient TA</td>
<td>Initial: 250, Min: 50, Max: 700; Median: 148.710, IQR: 75.580</td>
</tr>
<tr>
<td>$x_{\text{0,TS}}$</td>
<td>m</td>
<td>Muscle length shift TS</td>
<td>Initial: 0.05, Min: 0.011, Max: 0.06; Median: 0.025, IQR: 0.013</td>
</tr>
<tr>
<td>$x_{\text{0,TA}}$</td>
<td>m</td>
<td>Muscle length shift TA</td>
<td>Initial: 0.07, Min: 0.030, Max: 0.10; Median: 0.068, IQR: 0.028</td>
</tr>
<tr>
<td>$e_{\text{1,TA}}$</td>
<td>N/V EMG gain TA</td>
<td>Initial: $10^6$, Min: $10^6$, Max: $10^8$; Median: 1.923-$10^6$, IQR: 7.633-$10^6$</td>
<td>&lt; 0.1</td>
</tr>
<tr>
<td>$e_{\text{2,SO}}$</td>
<td>N/V EMG gains TS</td>
<td>Initial: $10^6$, Min: $10^6$, Max: $10^8$; Median: 1.000-$10^6$, IQR: 3.064-$10^6$</td>
<td>&lt; 0.1</td>
</tr>
<tr>
<td>$e_{\text{3,GM}}$</td>
<td>Hz</td>
<td>Activation filter freq.</td>
<td>Initial: 1.0, Min: 0.5, Max: 2.0; Median: 1.570, IQR: 1.369</td>
</tr>
<tr>
<td>$D_{\text{rel}}$</td>
<td>Nm</td>
<td>Relaxation offset</td>
<td>Initial: 0.1, Min: **, Max: **; Median: 0.974, IQR: 0.846</td>
</tr>
</tbody>
</table>

with TA as tibialis anterior and TS as triceps surae. Values indicated with () are scaled for fibula length and ** are taken as the negative and positive of the difference between the means of dorsal and plantar flexion movement (see Appendix B).