Medial gastrocnemius and soleus muscle-tendon unit, fascicle, and tendon interaction during walking in children with cerebral palsy

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ABBREVIATIONS

EMG	Electromyography
MTU	Muscle-tendon unit
SPM	Statistical parametric mapping

AIM This study investigates the in vivo function of the medial gastrocnemius and soleus muscle-tendon units (MTU), fascicles, and tendons during walking in children with cerebral palsy (CP) and an equinus gait pattern.
METHOD Fourteen children with CP (9 males, 5 females; mean age 10y 6mo, standard deviation [SD] 2y 11mo; GMFCS level I=8, II=6), and 10 typically developing (6 males, 4 females; mean age 10y, SD 2y 1mo) undertook full body 3D gait analysis and simultaneous B-mode ultrasound images of the medial gastrocnemius and soleus fascicles during level walking. Fascicle lengths were analysed using a semi-automated tracking algorithm and MTUs using OpenSim. Statistical parametric mapping (two-sample *t*-test) was used to compare differences between groups ($p<0.05$).
RESULTS In the CP group medial gastrocnemius fascicles lengthened during mid-stance gait and remained longer into late-stance compared to the typically developing group ($p<0.001$). CP medial gastrocnemius fascicles shortened less during stance (1.16mm [SD 1.47mm]) compared to the typically developing group (4.48mm [SD 1.94mm], $p<0.001$). In the CP group the medial gastrocnemius and soleus MTU and tendon were longer during early- and mid-stance ($p<0.001$). Ankle power during push-off ($p=0.015$) and positive work ($p<0.002$) and net work ($p<0.001$) were significantly lower in the CP group.
INTERPRETATION Eccentric action of the CP medial gastrocnemius muscle fascicles during mid-stance walking is consistent with reduced volume and neuromuscular control of impaired muscle. Reduced ankle push-off power and positive work in the children with CP may be attributed to reduced active medial gastrocnemius fascicle shortening. These findings suggest a reliance on passive force generation for forward propulsion during equinus gait.

Children with cerebral palsy (CP) show significant deviations from the walking patterns of a typically developing child. Equinus deformity is the most prevalent lower limb abnormality in children with CP and is caused by structural and functional adaptations of the calf muscle-tendon unit (MTU). Equinus gait is characterized by increased plantar flexion at the ankle and presents as a 'toe-walking' gait pattern.[1] Children with CP with an equinus gait pattern typically have slower preferred walking speed,[2] a shorter stride length,[3] reduced range of motion of the joints of the lower limb,[4] generate less power at the ankle and knee joints,[5] and have less positive work at the ankle compared to typically developing children.[6] While quantitative gait analysis, musculoskeletal modelling,[7] and simulation[8] are useful tools to assess overall muscle function during gait,[9] it is difficult to understand how the primary (neural) and secondary (musculoskeletal) impairments in CP influence gait mechanics and energetics.

The upper motor neuron lesion that causes CP creates a cascade of musculoskeletal alterations which contribute to reduced joint range of motion, increased joint stiffness, and muscle weakness.[10] In addition to these joint-level changes, lower limb musculotendinous adaptations in individuals with CP will impact the function of the muscles during gait and impact on how forces are generated for body support and propulsion during walking. Recent studies of the calf muscle have demonstrated that the plantar flexor muscle fascicles of children with CP are stiffer than typically developing children, even in the absence of active or reflex muscle contributions.[11] These muscles have been shown to have a smaller physiological cross sectional area and increased antagonist co-contraction which reduces force generation by up to 50 per cent.[10] The calf muscles generate a substantial force during level walking[12] with the impairment of calf strength impacting on gait in children with CP.[13,14] In addition to these muscle adaptations, the Achilles tendon is longer in young adults with CP,[10,15] presumably to counteract smaller muscle belly lengths.

The historical view of ankle motion and lower limb muscle function during the mid-stance phase of walking in typically developing individuals describes progression of the tibia anteriorly as the ankle dorsiflexes with the motion controlled by an eccentric contraction of the plantar flexor muscles (interpreted to be MTU lengthening).[16] Then, once the ankle reaches maximum dorsiflexion during late-stance, concentric plantar flexor contraction results in plantar flexion motion, the main power generation for forward propulsion in normal gait.[16] Recent investigations have differentiated the interaction of the muscle contractile component and the tendon of the plantar flexors and have better defined the muscle and tendon interaction. During steady pace, flat walking, the fascicles of the medial gastrocnemius and soleus contract at levels that maintain a relatively constant length during mid-stance while the more compliant Achilles tendon acts like a spring to absorb and store energy while it lengthens. This stored energy is returned during push-off when the MTU shortens.[17–20] It has been demonstrated using modelling that the muscle fibre length and tendon stiffness are optimally tuned to maximize muscle efficiency during walking and running.[21]

The interaction between the contractile component and the tendon during equinus gait in children with CP may be different than in typically developing individuals because of both the altered neuromuscular control and the secondary muscle adaptations (e.g. weakness, contracture) that occur with CP. Two recent studies investigating the calf muscle during walking in children with spastic type CP have provided differing findings. In the first study, children with diplegic CP, walking supported on a treadmill with crouch gait pattern, had medial gastrocnemius muscle fascicles that maintained a relatively constant length during mid-stance.[22] In contrast, the second study found the medial gastrocnemius muscle belly lengthened during the same gait phase in a group of children with hemiplegic and diplegic CP with a more toe-walking gait pattern and during overground independent walking.[23] The contrasting calf muscle behaviour may be a function of crouch versus toe-walking gait pattern or variability of muscle measurement and walking methodologies. Further investigation of the role of muscle–tendon interaction during CP gait is therefore necessary to distinguish the relative length changes of muscle fascicles (contractile component) from the tendon (elastic component).[18]

This study aimed to directly investigate the function of the medial gastrocnemius and soleus MTUs, fascicles, and tendon during walking in children with CP compared to typically developing children. We hypothesized that tendinous tissue would play a large role in both absorbing and returning mechanical energy in children with CP, and would therefore undergo the vast majority of stretch and shortening of the MTU – as has previously been demonstrated in typically developing individuals.[21] Given that a large amount of energy is absorbed in early-stance by plantar flexors in equinus gait (which is usually absorbed by the dorsiflexors in typically developing gait),[6] we proposed that muscle fascicles may undergo stretch in early- and mid-stance. Finally, because of the lower power generation in late-stance in CP compared to typically developing individuals,[6] we hypothesized that muscle fascicles in CP would shorten less during late-stance and therefore contribute less to overall power production.

What this paper adds

- Medial gastrocnemius fascicles in children with cerebral palsy (CP) contract eccentrically during mid-stance phase.
- Soleus fascicles in CP and typical development function similarly during stance.
- The Achilles tendon contributes significantly to the stretch and shorten of the muscle-tendon unit in CP and typical development.
- Reduced ankle power and work in CP may be attributed to reduced medial gastrocnemius fascicle shortening.

METHOD

Participants

Fourteen children with CP (9 males, 5 females; mean age 10y 6mo, standard deviation [SD] 2y 11mo; 8 hemiplegia, 6 diplegia, GMFCS level I=8, GMFCS level II=6) and 10 typically developing children (6 males, 4 females; mean age 10y, SD 2y 1mo) participated in the study. The participants with CP were recruited from the Queensland Children's Motion Analysis Service, Children's Health Queensland. The children with CP were included in the study if they had equinus gait and were under review for lower limb orthopaedic surgical correction. Participants were excluded if they had received: (1) any previous lower limb orthopaedic surgery; (2) lower limb intramuscular Botulinum neurotoxin A (BoNT-A) injection treatment in the 6 months before testing; or had (3) a lower limb injury in the 6 months before testing. The typically developing participants were recruited from the local community to participate in this study, were of similar age, and were required to have had no lower limb injury in the 6 months before testing. All procedures were approved by the institutional ethics committee (HREC/12/QRCH/60) and participants and parents gave informed consent before the testing protocol.

Participants with CP may had received one or all of the following: (1) lower limb intramuscular BoNT-A injections for the treatment of lower limb spasticity (not within the 6 months before testing); (2) physiotherapy management including isolated muscle strengthening, functional muscle conditioning, muscle stretching, gait re-education, balance and stability training, and functional and play skills; (3) orthotic management (ankle–foot orthoses and in-shoe orthoses) in accordance with the impact of lower limb spasticity and/or contracture and following gait pattern review; (4) lower limb serial casting.

Protocol

Height (cm) was measured using a stadiometer and mass (kg) was measured using a calibrated digital scale. Leg length (cm), fibula length (cm), calf circumference (cm), and ankle range of motion (°) with the knee extended were measured in the most involved lower limb in the CP group (determined by the treating paediatric specialist) and the right limb of the typically developing children (Table I). Participants walked barefoot at a self-selected speed over a level walkway 10m in length with three force platforms. Muscle architecture, 3D full body motion, ground reaction forces, and electromyography (EMG) data were captured simultaneously during 10 consecutive trials. Temporal and spatial gait parameters and ultrasound data of three strides (one clean stride from three separate trials) were analysed and averaged per participant.

Joint kinematics, joint kinetics, and MTU estimations

Reflective markers were attached to the trunk, pelvis, and lower limbs according to the modified Plug-in Gait marker set (Vicon Motion Systems; Oxford, UK). Additional markers included clusters of three markers on each thigh and shank segment and a marker on the fifth metarsal head. Marker trajectories were recorded at 100Hz using an eight-camera, 3D motion capture system (Vicon Motion Systems; Oxford, UK) and ground reaction force (GRF) data were acquired at 1 kHz using a three 510mm×465mm piezoelectric force platforms (AMTI; Watertown, MA, USA) arranged in line with 95mm between the first two platforms and 145mm between the second and third force platform. Marker trajectory and GRF data were filtered at 6Hz and a threshold of 20N on vertical GRF was used to determine the step and stride times for each step. Surface EMG activity was recorded using bipolar surface electrodes (Duo-trode, Myotronics; Washington, USA) with an inter-electrode distance of 2cm. Data were collected telemetrically (Aurion ZeroWire; Milan, Italy) from the soleus, medial gastrocnemius, lateral gastrocnemius, and tibialis anterior muscles at 1kHz. Raw EMG signals were high-pass filtered (Butterworth, zero-lag, 4th order, 30Hz) to remove movement artifact, full wave rectified, and low passed filtered (Butterworth, zero-lag, 4th order, 6Hz) and normalized to the maximum value of the EMG envelopes for that muscle across gait trials.[24]

Three-dimensional gait kinematics and kinetics, and muscle-tendon length estimates were computed in OpenSim[25] using a modified Gait2392 model that included three rotational degree-of-freedom at the knee and ankle to better represent the CP cohort.[26] In brief, this method involved creation of a custom OpenSim model that matched the degrees of freedom of the Plug-in Gait model. The biomechanical model was developed for an adult male and has been used previously in studies examining plantar flexor muscle lengths in children with CP.[7,27] Before model scaling, the custom model was assigned the static angles from the kinematic solution gained from the Plug-in Gait model in the static trial. The model was then scaled to the anthropometry of each participant using estimated joint centre locations to calculate the segment lengths and scaling ratios. Additional surface markers were then registered to match the position of the experimental markers. Analysis of walking data was then conducted using the Inverse Kinematics tool in OpenSim, while MTU length change of the medial gastrocnemius and soleus were exported using the OpenSim application program interface. The gait cycle was divided into stance and swing phases based on the individual participants' toe-off timing.

Table I: Physical characteristics of the participants

	CP (n=14)	Typical development (n=10)	p
Age	10y 6mo (SD 2y 11mo)	10y (SD 2y 6mo)	0.929
Height, cm	140 (20)	141 (20)	0.923
Weight, kg	41.5 (17.8)	37.7 (14.5)	0.809
Leg length, cm	74.3 (11.6)	72.6 (12.6)	0.641
Fibula length, cm	32.1 (4.2)	31.5 (5.5)	0.746
Calf circumference, cm	27.2 (4.3)	28.1 (3.8)	0.590
Ankle MDF, °	–7.1 (10.3)	27.7 (3.3)	<0.001

Data are mean (standard deviation) CP, cerebral palsy; MDF, maximum ankle dorsiflexion (positive values are dorsiflexion; negative values are plantar flexion).

Ultrasound

A personal computer-based ultrasound system (LogicScan 128; Telemed, Vilnius, Lithuania) with a 96-element low-profile linear probe (B-mode; 5MHz; 60mm field of view) was used to simultaneously image the medial gastrocnemius and soleus muscle fascicles at a sampling frequency of 80Hz. The probe was positioned over the medio-lateral centre of the medial gastrocnemius and aligned with the fascicle planes of the medial gastrocnemius and soleus[20,28] to minimize errors attributable to probe orientation[29] and secured over the skin surface with a compressive bandage to minimize probe movement relative to the skin. Rotation of the probe was also minimized because of its flat shape, which leads to very little inertia around the sagittal plane axis (approximately 0.8° rotation in the mediolateral direction).[30] A digital output signal from the ultrasound system was used to synchronize data collection. The length and pennation angle of the medial gastrocnemius and soleus muscle fascicles were determined throughout the stride using a semi-automated fascicle tracking algorithm.[31,32]

Data analysis and statistics

Parameters of interest included sagittal plane ankle and knee kinematics (°), ankle moments (Nm.kg^{-1}) and ankle power (W.kg^{-1}), and MTU length. Net ankle work was calculated as the integration of power and time using the trapezoidal method across the gait cycle. Negative and positive work across the gait cycle was calculated during the periods of positive and negative power respectively. All work values were normalized to body weight. The length of the tendon was determined by $L_{TENDON} = L_{MTU} - L_{FASCICLE} * \cos(\alpha)$,[18] where α is the pennation angle of the fascicle relative to the deep aponeurosis. MTU, fascicle, and tendon lengths for the medial gastrocnemius and soleus were normalized to the medial gastrocnemius and soleus MTU, fascicle, and tendon lengths at initial foot contact. The length changes of the medial gastrocnemius and soleus MTU, fascicle, and tendon from initial foot contact to toe-off were calculated.

Participant characteristics, ankle work, and medial gastrocnemius and soleus MTU, fascicle, and tendon length changes were compiled and compared between the CP and typically developing groups using unpaired *t*-tests (SPSS 24, IBM; Armonk, NY) (α=0.05). Statistical parametric mapping (SPM) was used to compare time series data. SPM is a statistical method able to perform hypothesis testing on one-dimensional (e.g. time series) kinematic and kinetic data in a continuous manner and takes into account the dependency between different time instances of the gait cycle and reduces post hoc regional focus bias and inter-component covariance bias in time series data.[33] SPM has been used previously to evaluate the effect of lower limb intramuscular injections of BoNT-A treatment on 3D gait kinematic parameters in children with spastic CP.[34] A 1D two-tailed paired *t*-test was performed on the time-normalized gait cycles of sagittal plane ankle and knee kinematics, ankle moments and ankle power, and medial gastrocnemius and soleus MTU, fascicle, and tendon lengths taking into consideration the dependency of all points of each gait cycle (α=0.05) to calculate critical threshold (t*).[35] All SPM analyses were implemented in Matlab (R2015b, The MathWorks, Massachusetts, USA) using open-source SPM1D code (www.spm1d.org).

RESULTS

There was no difference in demographic and anthropometric data between the groups (Table I). Children in the CP group had significantly reduced maximum ankle dorsiflexion range (t(22)=10.27, p<0.001).

The mean sagittal knee and ankle angle (Fig. 1a,b) for the CP group demonstrates an equinus pattern with ankle plantarflexion persisting throughout the gait cycle compared to the typically developing group with significant differences during stance at 0 to 6 per cent, 25 to 50 per cent, and during swing 67 to 100 per cent of the gait cycle (t*=2.870, p<0.010). Normalized ankle moment in the sagittal plane (Fig. 1c) in the CP group showed an absence of the internal dorsiflexion moment in the initial loading response and was significantly lower than the typically developing group during early-stance, 0 to 9 per cent of the gait cycle (t=3.588, p<0.001). Ankle power in the sagittal plane (Fig. 1d) showed significant negative power during early-stance, 0 to 9 per cent of the gait cycle (p<0.001), and significantly less push-off power in the CP group (t*=3.724, p<0.015). Normalized EMG linear envelope for medial gastrocnemius, lateral gastrocnemius, soleus, and tibialis anterior are presented in Figure 1e to h. Normalized medial gastrocnemius EMG was greater during 4 to 9 per cent of the gait cycle (t*=3.211, p=0.038) and normalized tibialis anterior EMG was significantly less around initial foot contact, 0 to 2 per cent and 98 to 100 per cent of the gait cycle (t*=3.190, p<0.033), in the CP group compared to the typically developing group.

Positive work (t(22)=3.621, p=0.002) and net work (t(22)=5.567, p<0.001) were significantly less in the CP versus the typically developing group (Fig. 2).

There was no difference in medial gastrocnemius and soleus MTU, fascicle, and tendon lengths at initial foot contact between the groups (Table II). Normalized medial gastrocnemius MTU length was significantly longer during 1 to 25 per cent and 53 to 67 per cent of the gait cycle (t*=4.081, p<0.001) (Fig. 3a). Normalized medial gastrocnemius fascicle length lengthened during the mid-stance period and remained significantly longer during 51 to 78 per cent of the gait cycle (t*=4.081, p<0.001) (Fig. 3b). Normalized medial gastrocnemius tendon length was significantly longer during 1 to 19 per cent of the gait cycle (t*=4.081, p<0.001) (Fig. 3c). Normalized soleus MTU length was significantly longer during 1 to 21 per cent of the gait cycle (t*=4.081, p<0.001) (Fig. 3d). There was no difference in normalized soleus fascicle length across the gait cycle between the groups (Fig. 3e). Normalized soleus tendon length was significantly longer during 1 to 19 per cent of the gait cycle (t*=4.081, p<0.001) (Fig. 3f). Medial gastrocnemius fascicle shortening from initial foot contact to toe-off was significantly less in the CP group (1.16mm [SD 1.47mm] vs 4.48mm [SD 1.94mm] in the typically developing group; t(22)=4.78, p<0.001).

DISCUSSION

This study demonstrates the behaviour of the medial gastrocnemius and soleus muscle fascicles, MTU, and tendon during gait in children with CP with equinus gait compared to typically developing peers. In children with CP the medial gastrocnemius muscle fascicles lengthened during mid-stance and, in contrast, the typically developing medial gastrocnemius fascicles maintain a relatively constant length. In the CP group the plantar flexed ankle position at initial foot contact results in a large plantar flexor moment and concomitant increase in medial gastrocnemius, LG, and soleus muscle EMG. The large amount of negative work (energy absorption) being performed by these muscles during early-stance is in contrast to energy being absorbed by the dorsiflexors in typically developing heel-toe walking.[6] While some of this energy is returned from the tendon during push-off, the overall net shortening of the fascicles is not sufficient to generate additional positive work and hence results in significantly less ankle net work across the gait cycle in the participants with CP.

Lengthening of the medial gastrocnemius muscle fascicles during mid-stance during human lower limb gait has not been reported. Even in activities with high ground reaction forces the medial gastrocnemius fascicles in

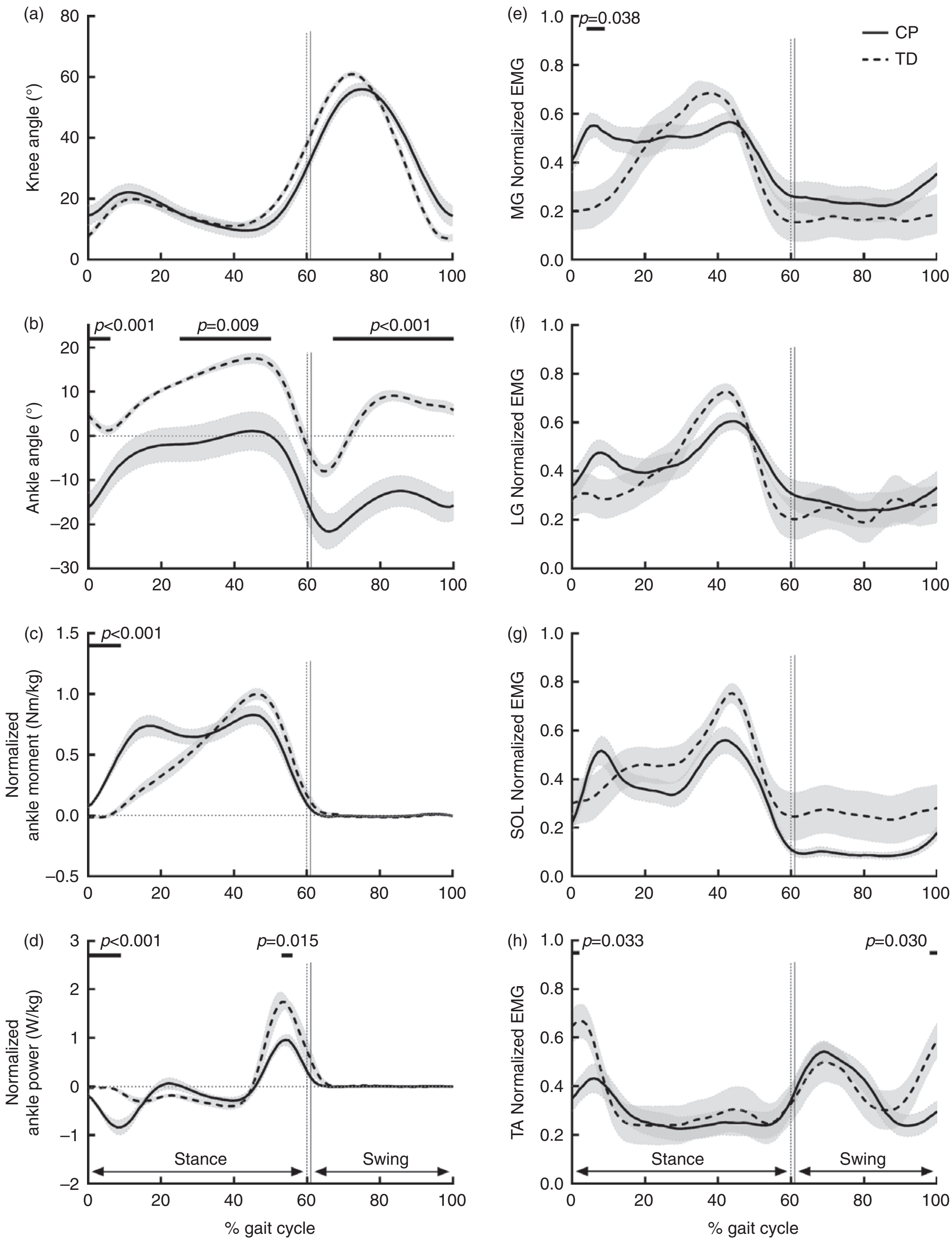

Figure 1: (a) knee angle; (b) ankle angle; (c) ankle moment; (d) ankle power; normalized electromyography (EMG) linear envelope for (e) medial gastrocnemius (MG), (f) lateral gastrocnemius (LG), (g) soleus (SOL), and (h) tibialis anterior (TA) during the walking gait cycle (0–100% foot contact to foot contact, vertical line toe-off) for the CP group (solid) and TD group (dashed), mean (standard error of measurement). Lines and *p*-values above plots show significant difference between groups during the proportion of the gait cycle. CP, cerebral palsy; TD, typically developing.

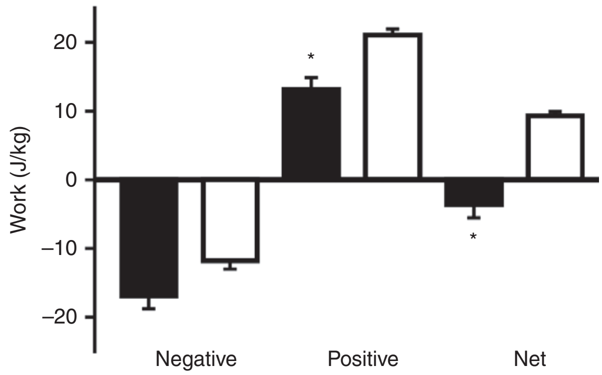

Figure 2: Negative, positive, and net ankle work during the walking gait cycle. Children with cerebral palsy (black) versus typically developed children (white), mean (standard error of measurement). *Significant difference between groups (p<0.01).

Table II: Medial gastrocnemius and soleus muscle-tendon unit, fascicle, and tendon lengths at initial foot contact in children with CP compared to typically developing children

	CP (n=14)	Typical development (n=10)	p
Medial gastrocnemius MTU length, mm	377.8 (65.8)	396.4 (58.1)	0.481
Medial gastrocnemius fascicle length, mm	43.7 (12.0)	50.2 (7.5)	0.141
Medial gastrocnemius tendon length, mm	334.7 (67.1)	346.8 (59.1)	0.650
Soleus MTU length, mm	243.7 (42.5)	259.2 (36.4)	0.362
Soleus fascicle length, mm	33.1 (10.7)	36.6 (7.2)	0.374
Soleus tendon length, mm	212.8 (43.9)	224.9 (34.6)	0.483

Data are mean (standard deviation). CP, cerebral palsy; MTU, muscle-tendon unit.

typically developing young adults have been shown to shorten – for example from initial foot contact to late mid-stance running[17,36] or throughout the contact phase (braking and propulsion) during repeated hopping.[37] Our findings were in support of Kalsi et al.[23] who reported the medial gastrocnemius muscle belly (estimation of the entire medial gastrocnemius contractile component) lengthening during mid-stance gait in children with CP and confirm the impact of equinus gait pattern on calf muscle function during overground walking. Interestingly, this study found that simulated toe walking did not result in muscle belly lengthening in typically developing adults. And therefore lengthening of fascicles may not be due to the high energy absorption requirements in early-stance when toe walking. Fascicle lengthening is indicative of an eccentric action of the muscle, the external force on the muscle being greater than the force that the contracting muscle is generating and resulting in energy being absorbed by the muscle rather than the tendon. Repeated eccentric contractions from toe walking could potentially result in muscle damage;[3] however, it remains to be seen whether this stimulus is sufficient in children with CP. These muscle actions and resultant tissue responses may not, however, be experienced in children with CP walking with crouch gait as medial gastrocnemius fascicle behaviour more closely follows the isometric contractions reported in typically developing children.[22]

It is unclear why the medial gastrocnemius muscle fascicles lengthen in the children with CP during early- and mid-stance. We propose two possibilities. The first possibility is that reduced muscle strength due to the smaller muscle size,[38] selective neural activation and antagonistic co-activation,[39] or shortened overall muscle lengths resulted in insufficient force to maintain the fascicle length and stretch the tendon during the energy absorption phase. In similarly aged children with CP, plantar flexor volume has been shown to be between 22 to 28 per cent less than typical developed peers,[40,41] which will reduce active ankle plantar flexion torque.[10] Our findings suggest that medial gastrocnemius muscle eccentric contraction during mid-stance gait occurs in conjunction with a relatively constant level of medial gastrocnemius muscle activation across the same period, but it is unclear whether the active force generated here is sufficient to provide the required tendon stretch or if passive forces may instead be recruited through stretch of the fascicles.

The second potential mechanism driving the stretch of the medial gastrocnemius fascicles is that the series elastic tendinous structures (which include the external tendon, aponeurosis, and internal connective tissue framework) are too stiff to be stretched by the forces generated by the muscle contractile tissue. There is ample evidence that the inherent muscle stiffness is much greater in CP than in typically developing muscle.[11,42] The increase in stiffness might reduce the capacity for the series elastic structures to absorb energy, and hence this energy is absorbed within the muscle instead. The changes in medial gastrocnemius fascicle length in the participants with CP across the stride shows a resemblance to a lower magnitude MTU pattern and may suggest that the active and passive components of the medial gastrocnemius MTU act somewhat uniformly because of the inability of the participants with CP to produce force in their lower limb muscles.

The lengthening of the medial gastrocnemius MTU relative to the fascicle length change is large during mid-stance in both groups and can be attributed to the compliant Achilles tendon which functions to store elastic energy to be returned for the subsequent propulsive phase of gait.[21] Although the medial gastrocnemius MTU stretched to greater lengths in the children with CP, the tendon reached similar lengths to the typically developing children. The difference in medial gastrocnemius MTU length can be attributed to the concomitant medial gastrocnemius fascicle lengthening in the CP group. In children with CP, however, MTU and tendon lengthening begins from initial foot contact, when energy is absorbed by the plantar flexors, and results in the MTU and tendon stretch occurring

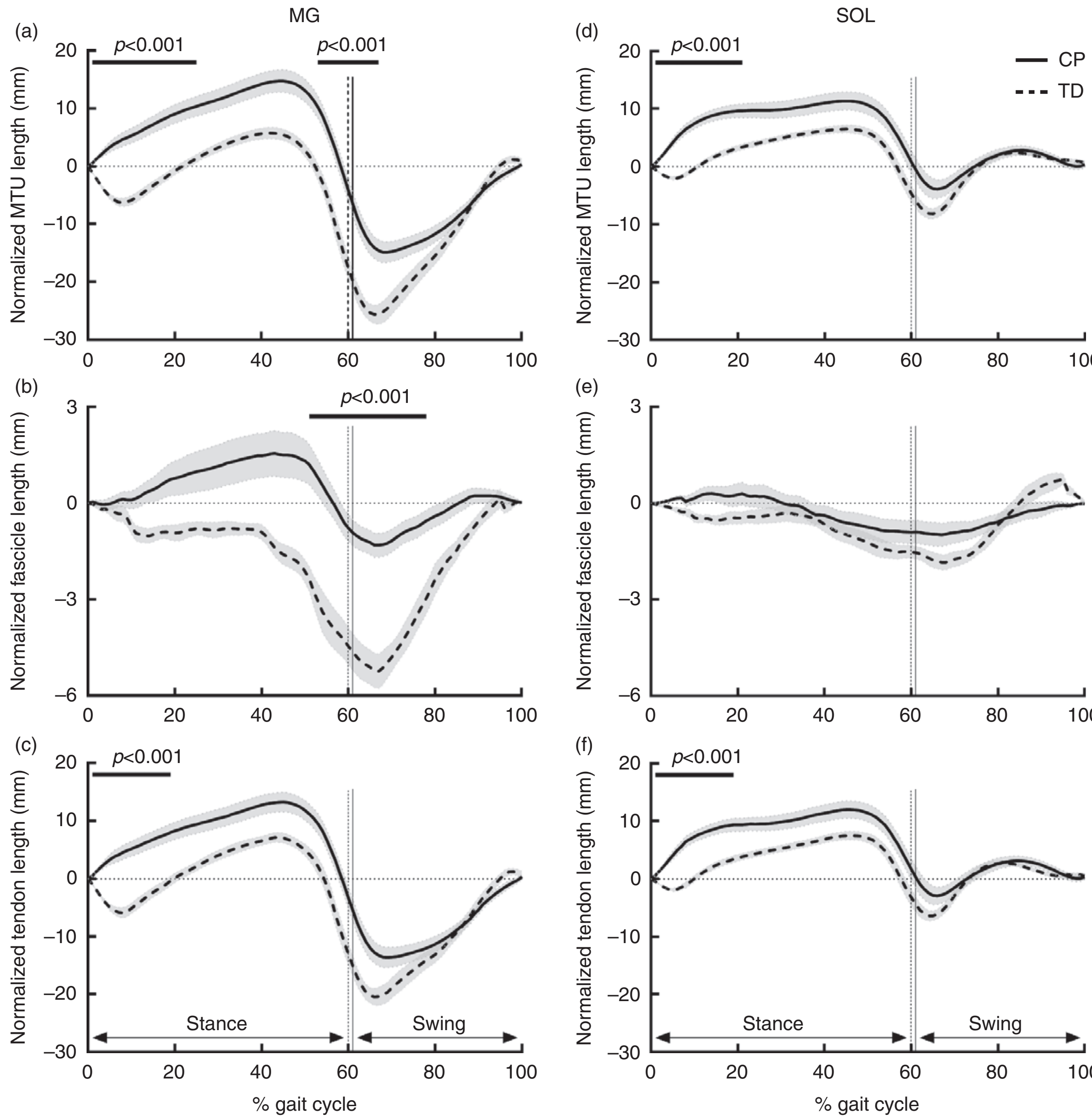

Figure 3: (a) normalized medial gastrocnemius (MG) muscle-tendon unit (MTU) length; (b) normalized MG fascicle length; (c) normalized MG tendon length; (d) normalized soleus (SOL) MTU; (e) normalized SOL fascicle length; (f) normalized SOL tendon length during the walking gait cycle (0–100% foot contact to foot contact, vertical line toe-off). MTU, fascicle, and tendon lengths for the MG and SOL were normalized to the MG and SOL MTU, fascicle and tendon lengths at initial foot contact. CP group (solid), TD group (dashed), mean (standard error of measurement). Positive values are lengthening; negative values are shortening. Lines and *p*-values above plots show significant difference between groups during the proportion of the gait cycle. CP, cerebral palsy; TD, typically developing.

over a greater period of the gait cycle than in typically developing children.

The behaviour of the CP and typically developing soleus fascicles followed a similar pattern across the gait cycle; however, in the CP group the MTU stretched significantly in early-stance, although the overall stretch is similar to the typically developing group at the end of mid-stance. Therefore, while the fascicle behaviour is similar in the two groups, the tendon is absorbing more energy in early support in the CP group. Soleus fascicle shortening was shown to occur before the MTU shortening in both CP and typical development, which has been previously demonstrated during walking in typically developing adults.[43,44] The shortening of the soleus fascicles generates mechanical work that stretches the tendon and continues to store energy, which can be recovered during push-off when the tendon recoils. In this case, it seems that the muscle function of the soleus for generating work during push-off seems similar in children with CP and typically developing children. Despite the fact that medial gastrocnemius and soleus are functionally synergistic at the ankle during walking, the simultaneous shortening of the soleus fascicles and lengthening of the medial gastrocnemius fascicles in the children with CP during mid-stance may be due to the reduced impairment of the soleus in individuals with CP compared to the medial gastrocnemius muscle.[7]

During late-stance gait in typically developing individuals, the ankle plantar flexors contribute to ankle power for push-off and to accelerate the limb into swing through active muscle contraction producing positive work and passive tendon recoil yielding elastic strain energy.[45] Our findings show that the normalized peak ankle power and positive work were significantly reduced by 52 per cent and 38 per cent respectively in the children with CP compared to the typically developing peers. The significantly reduced shortening of the medial gastrocnemius fascicles (and soleus fascicles, but not significant) from foot contact to toe-off in the participants with CP would result in a smaller contribution of the plantar flexor muscle contractile tissue to the net work produced. We therefore believe that the reduced ankle power during propulsion in ambulant children with CP is a result of reduced muscle fibre work performed across the stance phase, and that this may primarily be due to the reduced apparent strength of the plantar flexor muscles – i.e. reduced muscle volume[38] in conjunction to less muscle activation (based on EMG during gait).

There are certain points to consider in light of the current study. The findings are not generalizable to all speeds of walking or to walking on gradients. MTU, fascicle, and tendon behaviour of the medial gastrocnemius and soleus may vary with different conditions, as reported in typically developing individuals[17,46] and children with CP walking with a crouch gait pattern,[22] and needs to be examined in future studies. Conclusions about the contribution of the lengthening fascicles to passive force generation within the MTU and the specific contractile conditions of both the medial gastrocnemius and soleus cannot yet be established without further investigation. With the increased prevalence of musculoskeletal modelling to simulate the relationship of kinematics and muscle lengths in childhood CP gait abnormalities,[47] our findings of medial gastrocnemius and soleus muscle and tendon interactions during equinus gait should be considered to inform future walking simulations in this population. Surgical procedures described for lengthening the gastrocnemius-soleus muscle tendon unit in children with CP to normalize equinus gait improve gait function;[48,49] however, the impact on the interaction between the contractile and non-contractile elements in the calf muscle remains unknown. Interventions that promote increases in calf muscle strength, such as progressive resistance training[50] and electrical stimulation,[51] should be strongly considered from an early age in children with CP; however, training intensity and volume and recovery periods need to be closely monitored to minimize the potential negative impact of repeated eccentric contractions on tissue adaptations.[52]

CONCLUSION

The eccentric action of the CP medial gastrocnemius muscle fascicles during mid-stance walking is consistent with reduced strength of the impaired muscle. Neuromuscular adaptations of the impaired CP calf muscle may result in greater reliance on aponeurotic and tendon structures passive elastic energy storage and return for forward propulsion during equinus gait. The findings of the interaction between the contractile (fascicle) and non-contractile (tendon) elements in the calf muscle of children with CP walking with equinus gait informs surgical, pharmacological, and conservative management decision-making to improve walking ability in children with CP.

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