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Medial gastrocnemius structure and gait kinetics in spastic cerebral palsy and typically developing children

A cross-sectional study

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Abstract

To compare medial gastrocnemius muscle-tendon structure, gait propulsive forces, and ankle joint gait kinetics between typically developing children and those with spastic cerebral palsy, and to describe significant associations between structure and function in children with spastic cerebral palsy.

A sample of typically developing children (n = 9/16 limbs) and a sample of children with spastic cerebral palsy (n = 29/43 limbs) were recruited. Ultrasound and 3-dimensional motion capture were used to assess muscle-tendon structure, and propulsive forces and ankle joint kinetics during gait, respectively.

Children with spastic cerebral palsy had shorter fascicles and muscles, and longer Achilles tendons than typically developing children. Furthermore, total negative power and peak negative power at the ankle were greater, while total positive power, peak positive power, net power, total vertical ground reaction force, and peak vertical and anterior ground reaction forces were smaller compared to typically developing children. Correlation analyses revealed that smaller resting ankle joint angles and greater maximum dorsiflexion in children with spastic cerebral palsy accounted for a significant decrease in peak negative power. Furthermore, short fascicles, small fascicle to belly ratios, and large tendon to fascicle ratios accounted for a decrease in propulsive force generation.

Alterations observed in the medial gastrocnemius muscle-tendon structure of children with spastic cerebral palsy may impair propulsive mechanisms during gait. Therefore, conventional treatments should be revised on the basis of muscle-tendon adaptations.

Abbreviations: aGRF = anterior ground reaction force, bE = belly excursion, bL = belly length, BW = body weight, CI = confidence interval, CP = cerebral palsy, ES = effect size, fE = fascicle excursion, fL = fascicle length, GRF = ground reaction force, MDF = maximum dorsiflexion, MG = medial gastrocnemius, MTJ = myotendinous junction, NP = negative power, P = power, PP = positive power, RJA = resting joint angle, ROM = range of movement, SCP = spastic cerebral palsy, SD = standard deviation, SE = standard error, TD = typically developing, tE = tendon excursion, tL = tendon length, vGRF = vertical ground reaction force.

Keywords: ankle joint, ground reaction force, joint power, muscle architecture

1. Introduction

Cerebral palsy (CP) has been defined as "a group of permanent disorders affecting 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."[1] Spastic type CP (SCP) is the most common type of CP and has been reported to result in short muscle-tendon units, muscle hyper-resistance, weakness, and impaired sensory-motor control.[2] These neuromuscular deficits have been frequently observed at the medial gastrocnemius (MG) muscle-tendon unit of children with SCP,[2] and have been associated to impaired muscle-tendon function during gait.[3,4] Consequently, children with SCP present altered gait,[2] including abnormal ground reaction forces (GRF)[5] and ankle power and work performance[3] that would ultimately affect support and progression.[6–8] However, these interrelations are still not well understood.[2]

Regarding MG muscle-tendon structure of children with SCP, studies have consistently reported longer Achilles tendons,[9,10] and reduced MG muscle belly volume, cross-sectional area, thickness, and length with respect to typically developing (TD) children.[11] Conversely, reports on fascicle length differences between TD children and those with SCP have been inconclusive.[11] Fascicle length has been considered an important functional parameter which determines fascicle excursion and in turn the range through which muscles can develop force and power, maximum shortening speed, and length at which passive forces are developed.[12] Accordingly, significant correlations have been found between knee extensor fascicle length and rate of

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force development and impulse in TD children, but not in children with CP.[13] Authors argued that longer fascicles, indicative of a greater number of sarcomeres in series, would allow for greater fiber shortening velocity which would have direct influence on rate of force development and impulse.[13] However, it appears that regulation of sarcomere length does not occur similarly in TD children and those with SCP.[14]

Studies on TD children have observed that with growth, MG muscles and fascicles lengthen proportionally with tibia length.[15] In response to this chronic stretch, fascicles have been reported to adapt through in-series sarcomere addition in order to maintain and optimize force production capacity.[16] However, children with SCP have shown fewer and longer sarcomeres compared to TD children despite having similar fascicle lengths, which has suggested impaired muscle adaptation to longitudinal growth.[17] Thus, we may not infer ankle joint power and work performance solely through fascicle length. Moreover, conventional isometric testing may be inadequate when comparing children with SCP and TD children due to different optimal joint positions.[18,19] Thus, more functional tests such as isokinetic testing[20] or 3D motion capture analysis may better elucidate ankle joint power and work performance limitations of children with SCP.[3]

Furthermore, passive mechanisms have been shown to contribute substantially to joint kinetics in TD subjects.[21] Hence, impaired passive mechanisms observed in children with SCP[22–26] may further affect GRF generation and ankle joint power and work performance during gait. In this sense, children with SCP have shown limited dorsiflexion,[22–24] greater ankle joint stiffness,[22] greater MG muscle belly stiffness,[23] and MG fascicle stiffness[24] with respect to TD children. Moreover, the greater MG fascicle stiffness reported for children with SCP was accompanied by reduced fascicle strain.[24] Similarly, children with SCP and equinus gait have shown reduced MG fascicle strain,[10,27] and TD subjects with greater ankle stiffness have shown reduced elongation of fascicles and aponeuroses at maximum angles compared to those with lower ankle stiffness.[28] Thus increased resistance to stretch during passive ankle dorsiflexion in SCP may be attributed at least in part to the inability of the fascicles to elongate with added force.[24] Therefore, we believe that relative fascicle and muscle excursions may be related to power performance during gait. Conversely, no differences in tendon stiffness between TD children and those with SCP have been reported.[23]

Impaired MG musculotendinous adaptations observed in children with SCP may contribute to GRF generation and ankle joint power performance during gait. However, this has not been assessed to date. Thus, the aims of the present study were: to compare MG muscle-tendon structure and passive strain of children with SCP and TD children; to compare GRF and ankle joint power performance during gait of children with SCP and TD children; and to describe associations between MG muscle-tendon structure and passive excursions, and ankle joint power and propulsive forces during gait in children with SCP.

2. Methods

2.1. Participants

A convenience sample of children with SCP was obtained from those who visited the movement analysis laboratory of our hospital for a routine gait analysis and met the following inclusion criteria: diagnosed SCP; hemiplegic or diplegic; independently ambulant GMFCS I-II; no previous orthopedic or neuro-surgery[10];no botulinum neurotoxin type A injections[29] or casting[30] 6 months prior assessment; and within 5 to 12 years of age.[15] This age range was selected as MG length has been linearly associated to tibia length within this range.[15] In addition, a group of TD children were recruited for comparison which met the following inclusion criteria: no lower-limb impairment or impairments affecting locomotion such as impaired postural control system; and within 5 to 12 years of age. Children and/or parents or legal guardians signed an informed consent prior testing. Recruitment and testing were carried out on the same day. This study was approved by the Ethics Committee on Clinical Research of our hospital (R-0021/15) and was in accordance with the Declaration of Helsinki on human research.

2.2. Procedures

Regarding MG muscle-tendon structure assessments, subjects were instructed to lay prone on an examination table, with their feet hanging from the edge, and their knees fully extended. Once the subject was ready, fibula length, resting ankle joint angle (RJA), and maximum dorsiflexion (MDF) were measured. Fibula length was measured from the head of the fibula to the lateral malleolus through an anthropometric tape. Furthermore, RJA and MDF were measured with a manual goniometer by placing its axis at the lateral malleolus with one goniometer arm aligned with the head of the fibula and the other parallel to the lateral base of the foot. Subsequently, a strapping device was fixed to the subject's forefoot and used to move the ankle into MDF and fix the position (Fig. 1). Assistance was used to immobilize the hip on the testing side.

A 2D B-mode ultrasound imaging device (F31, Hitacki, Aloka) with a 13 MHz linear array transducer (UST-5413) was used to take images of the MG muscle-tendon unit. An extended field of view technique was used to follow the path of MG fascicles, muscle belly, and Achilles tendon. This technique has been reported to be highly valid and reliable for the measurement of fascicle length, more so than conventional trigonometric relations.[31] A substantial amount of gel was placed on the ultrasound probe and pressure was kept minimal throughout assessments. Three ultrasound images were taken at each ankle joint position (RJA and MDF) and for each structure (fascicle, belly, and tendon) resulting in a total of 18 images for further analysis. Subjects were instructed to relax throughout recordings and if movement or muscle contraction was detected through visual inspection that trial was discarded and repeated.

The probe position was standardized following previously reported error reduction methods.[32] In order to accurately identify the MG muscle-tendon trajectory the following landmarks were marked: medial fourth of femur intercondylar distance, mid-way of the MG width, and myotendinous junction (MTJ) of the MG.[33] The mid-way of the MG width was marked when a transverse plane ultrasound image was centered on the MG and the deep aponeurosis was parallel to the image horizontal. Then, the ultrasound probe was rotated 90° and the path of the MG was followed down to the MTJ of the MG, position from which muscle fascicle images were taken.[32] From the later position, the ultrasound probe was moved following a straight line up to the medial condyle of the femur to obtain an image of the muscle belly. Similarly, Achilles tendon images were reconstructed from the proximal edge of the calcaneus to the MG MTJ.

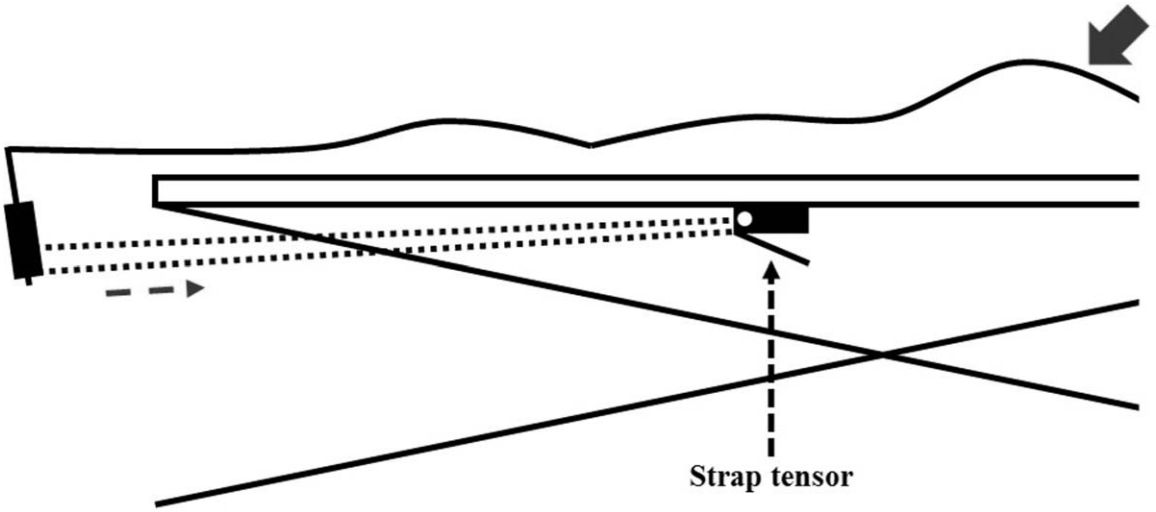

Figure 1. Representative diagram of ankle joint position fixation. Thick arrow indicates direction of manual force applied at the hip for immobilization. Dashed arrow indicates direction of pull.

Moreover, a 3D motion capture system with 8 optoelectronic cameras (Smart DX 6000, BTS Bioengineering, Milan, Italy) synchronized with two force platforms (Kistler 9286AA, Winterthur, Switzerland) was used to measure GRFs and joint kinetics. Sampling frequency was set at 200 Hz for both motion and force recordings. A set of reflective markers were placed over the skin on discrete anatomical sites in order to build a Helen Hayes Model.[34] After calibration, technical markers were removed and the subject was asked to walk normally throughout the calibrated volume until at least 10 walking trials were completed. No instructions regarding foot placement were given. Assessments were carried out on the affected limb for hemiplegic children and on both limbs for diplegic children.

2.3. Data analysis

Images taken were transferred to an image processing software for measurement (ImageJ 1.50b, National Institutes of Health, Bethesda, MD). Variables extracted at each position were: fascicle length (% fibula length) measured from deep to superficial aponeurosis on a clearly visible fascicle located at a distance of one-third muscle belly length proximal to the MTJ (fL)[9,10,35,36]; tendon length (% fibula length) measured from the proximal edge of the calcaneus to the MTJ (tL); belly length (% fibula length) measured from the MTJ to the medial condyle of the femur (bL); fascicle to belly length ratio (fLbL Ratio); tendon to fascicle length ratio (tLfL Ratio); and tendon to belly length ratio (tLbL Ratio). Furthermore, from fL, tL, and bL measurements at RJA and MDF, the following variables were extracted: relative fascicle excursion (%) measured as the percent change in fL from RJA to MDF relative to fL at RJA (fE); relative tendon excursion (%) measured as the percent change in tL from RJA to MDF relative to tL at RJA (tE); andrelative muscle belly excursion (%) measured as the percent change in bL from RJA to MDF relative to bL at RJA (bE). The mean of 3 measurements (one measurement per image) was used for further analysis.

Gait kinetic data was exported and processed to extract the following variables: Peak negative power (W/kg) (NP peak); peak positive power (W/kg) (PP peak); total negative power (W/kg) (NP total); total positive power (W/kg) (PP total); net power (W/kg) (P net); push-off positive power (W/kg) (PP push-off); peak vertical GRF at push-off (%BW) (vGRF peak); peak anterior GRF at push-off (%BW) (aGRF peak); total vertical GRF (%BW) (vGRF total); and total anterior GRF (%BW) (aGRF total). Furthermore, normalized gait velocity (% height/s) was measured.

2.4. Statistical analysis

The Statistical Package for the Social Sciences (SPSS 22.0, IBM Corp., Armonk, New York) was used for statistical analyses. In order to compare sample characteristics, RJA, MDF, range of motion (ROM), and MG muscle-tendon structure of TD children and those with SCP we ran a student *t* test after checking the data for assumption compliance. For those variables which did not meet the student *t* test assumptions, a Mann-Whitney *U* test was used instead after checking the data for assumption compliance. Furthermore, a one-way ANCOVA test for independent means was carried out after checking the data for assumption compliance to check for differences between TD children and those with SCP regarding fE, tE, and bE while controlling for ROM effects on differences. Similarly, in order to compare gait kinetics of TD children and those with SCP, a one-way ANCOVA test for independent means was carried out in order to control for gait velocity effects on differences. Finally, in order to establish associations between muscle structure and gait kinetics, we ran hierarchical multiple regression analyses controlling for gait velocity after checking data for assumption compliance. Velocity covariate was entered first as an individual block, and each muscle architecture variable was entered second as another individual block. A third individual block with ROM was entered before excursion variables in order to control for the effects of ROM on excursion. Throughout all the statistical analyses a Shapiro-Wilk test was used to determine whether data was normally distributed.[37]

3. Results

Reported means are mean (SD). A sample of children with SCP [n = 29 and 43 limbs, age 8.64 years (2.18), height 131.47 cm (11.95), weight 28.24 kg (7.45), 41.4% hemiplegic and 58.6% diplegic, 62.0% GMFCS I and 38.0% GMFCS II) was compared to a sample of TD children (n = 9 and 16 limbs, age 9.16 years

Table 1

Passive ankle joint kinematics and medial gastrocnemius muscle-tendon structure of typically developing children and those with spastic cerebral palsy.

	Typically Developing		Spastic Cerebral Palsy		
	Mean (SD)	95% CI	Mean (SD)	95% CI	ES
RJA (deg)	−27.00 (5.35)	−30.09 to −23.91	−31.60 (11.01)	−34.99 to −−28.22	0.46
MDF (deg)	21.71 (11.42)	15.12–28.31	9.56 (11.87)	5.91–13.21	1.03[a]
ROM (deg)	48.71 (14.91)	40.10–57.32	41.16 (11.59)	37.60–44.73	0.61
RJA fL (% fibula)	13.91 (1.85)	12.92–14.89	11.40 (2.36)	10.68–12.13	1.12[b]
RJA tL (% fibula)	54.16 (7.36)	50.24–58.09	59.14 (5.08)	57.58–60.71	0.86[c]
RJA bL (% fibula)	64.21 (6.15)	60.80–67.61	56.42 (5.66)	54.68–58.16	1.35[b]
MDF fL (% fibula)	19.77 (2.55)	18.41–21.13	16.19 (3.61)	15.08–17.30	1.06[c]
MDF tL (% fibula)	52.21 (6.80)	48.58–55.83	57.99 (5.17)	56.40–59.58	1.02[c]
MDF bL (% fibula)	70.01 (7.06)	66.24–73.77	61.06 (6.99)	58.91–63.21	1.28[b]
RJA fLbL ratio	0.22 (0.03)	0.20–0.24	0.20 (0.04)	0.19–0.21	0.45
MDF fLbL ratio	0.28 (0.03)	0.27–0.30	0.26 (0.05)	0.25–0.28	0.44
RJA tLfL ratio	3.97 (0.87)	3.51–4.44	5.50 (1.66)	4.99–6.01	1.03[b]
MDF tLfL ratio	2.70 (0.64)	2.36–3.05	3.85 (1.30)	3.45–4.25	0.99[b]
RJA tLbL ratio	0.86 (0.19)	0.75–0.96	1.06 (0.18)	1.01–1.12	1.14[b]
MDF tLbL ratio	0.76 (0.17)	0.67–0.85	0.97 (0.19)	0.91–1.03	1.13[b]

bL = belly length, CI = confidence interval, ES = pooled effect size), fL = fascicle length, MDF = maximum dorsiflexion, RJA = resting joint angle, ROM = range of motion, SD = standard deviation, tL = tendon length. Significant differences between groups are [a]($P < .05$), [b]($P < .001$), and [c]($P < .01$),.

(2.66), height 133.90 cm (16.95), weight 29.94 kg (9.95)]. Three diplegic children in the SCP group had only their most affected limb tested due to lack of time. There were no statistically significant differences between groups regarding sex, age, height, or weight.

3.1. Passive ankle joint kinematics

Children with SCP had significantly reduced MDF with respect to TD children, $t(55) = 3.359$, $P = .001$, $d = 1.033$. No statistically significant differences between groups were found for RJA or ROM. Table 1 shows a detailed report of results regarding passive ankle joint kinematics.

3.2. Muscle architecture

Regarding muscle architecture, children with SCP had shorter MG fascicles than TD children at RJA, $t(57) = -3.829$, $P = .000$, $d = 1.121$; and at MFD, $t(57) = -3.631$, $P = .001$, and $d = 1.063$. Similarly, children with SCP had shorter muscle bellies than TD children at RJA, $t(56) = -4.485$, $P = .000$, $d = 1.345$; and at MDF, $t(57) = 4.356$, $P = .000$, and $d = 1.276$. Conversely, children with SCP had longer tendons than TD children at RJA, $t(57) = 2.948$, $P = .005$, $d = .863$; and at MDF, $t(57) = 3.495$, $P = .001$, and $d = 1.024$. Furthermore, regarding MG tLfL ratios, scores for children with SCP were higher than for TD children at RJA, $U = 120$, $z = -3.820$, $P = .000$; and at MDF, $U = 128$, $z = -3.683$, $P = .000$, using an exact sampling distribution for U. Similarly, MG tLbL ratios of children with SCP were higher than for TD children at RJA, $t(56) = 3.797$, $P = .000$, $d = 1.138$; and at MDF $t(57) = 3.873$, $P = .000$, and $d = 1.134$. No statistically significant differences between groups were found for fLbL ratios and fE, bE, and tE. Tables 1 and 2 show a detailed report of results regarding muscle architecture and relative excursions respectively.

3.3. Gait kinetics

Children with SCP walked more slowly than TD children $t(56) = 2.529$, $P = .014$, $d = .747$, thus, gait velocity was controlled for. Children with SCP presented a significantly greater NP peak than TD children, $F(1, 55) = 31.651$, $P = .000$, partial $\eta^2 = .365$, $d = 1.010$. Conversely, children with SCP presented a significantly smaller PP peak than TD children, $F(1, 55) = 41.238$, $P = .000$, partial $\eta^2 = .429$, and $d = 2.056$. In line with peak power parameters, children with SCP had more NP total than TD children, $F(1, 55) = 11.757$, $P = .001$, partial $\eta^2 = .176$, $d = .979$. Furthermore, children with SCP had significantly less PP push-off than TD children, $F(1, 55) = 21.610$, $P = .000$, partial $\eta^2 = .282$, and $d = 1.632$. Accordingly, children with SCP presented less P net than TD children, $F(1, 55) = 18.226$, $P = .000$, partial $\eta^2 = .249$, $d = 1.514$, despite showing more NP total.

Finally, regarding GRF, children with SCP generated less vGRF total than TD children, $F(1, 55) = 8.867$, $P = .004$, partial $\eta^2 = .139$, $d = .659$. Accordingly, children with SCP presented reduced vGRF peak at push-off with respect to TD children, $F(1, 55) = 21.450$, $P = .000$, partial $\eta^2 = .281$, $d = 1.410$. Finally,

Table 2

Relative medial gastrocnemius fascicle, muscle, and Achilles tendon excursions of typically developing children and those with spastic cerebral palsy.

	Typically Developing			Spastic Cerebral Palsy			
	Unadjusted Mean (SD)	Adjusted Mean (SE)	95% CI	Unadjusted Mean (SD)	Adjusted Mean (SE)	95% CI	ES
fE (%)	41.56 (9.81)	40.76 (3.07)	34.60–46.92	41.94 (11.64)	42.21 (1.72)	38.75–45.66	0.03
tE (%)	−2.36 (4.54)	−1.73 (1.16)	−4.05–0.59	−2.63 (4.38)	−2.85 (0.66)	−4.18 to −1.51	0.06
bE (%)	9.02 (5.37)	8.33 (1.36)	5.60–11.05	8.16 (5.10)	8.37 (0.74)	6.89–9.86	0.17

bE = relative muscle belly excursion, CI = confidence interval, ES = pooled effect size, fE = relative fascicle excursion, SD = standard deviation, SE = standard error, tE = relative tendon excursion.

Table 3

Gait kinetics of typically developing children and those with spastic cerebral palsy.

	Typically Developing			Spastic Cerebral Palsy			
	Unadjusted Mean (SD)	Adjusted Mean (SE)	95% CI	Unadjusted Mean (SD)	Adjusted Mean (SE)	95% CI	ES
NP total (W/kg)[a]	0.66 (0.21)	0.63 (0.07)	0.49–0.78	0.92 (0.30)	0.93 (0.04)	0.84–1.02	0.98[$]
PP total (W/kg)	17.00 (3.99)	15.36 (1.27)	12.81–17.90	13.70 (6.24)	14.33 (0.76)	12.80–15.86	0.58
P net (W/kg)	9.19 (4.45)	8.12 (1.35)	5.43–10.82	0.87 (5.83)	1.28 (0.81)	−0.35–2.90	1.51[†]
PP total push-off (W/kg)	16.28 (3.77)	15.24 (0.89)	13.45–17.03	9.90 (3.96)	10.30 (0.54)	9.22–11.38	1.63[†]
NP peak (W/kg)[b]	0.85 (0.09)	0.88 (0.04)	0.79–0.96	0.60 (0.19)	0.59 (0.03)	0.54–0.65	1.01[†]
PP peak (W/kg)	2.04 (0.61)	1.90 (0.09)	1.72–2.09	1.14 (0.36)	1.19 (0.06)	1.08–1.31	2.06[†]
vGRF total (%BW)	5035.99 (119.44)	5074.78 (58.65)	4957.24–5192.31	4881.36 (264.70)	4866.58 (35.33)	4795.77–4937.39	0.66[$]
aGRF total (%BW)	269.85 (49.11)	258.59 (21.99)	214.52–302.66	273.12 (96.45)	277.41 (13.25)	250.86–303.96	0.04
vGRF peak (%BW)	107.61 (6.06)	107.78 (1.81)	104.16–111.40	97.87 (7.17)	97.81 (1.09)	95.63–99.99	1.41[†]
aGRF peak (%BW)	18.92 (2.96)	18.13 (0.95)	16.24–20.03	13.98 (4.18)	14.28 (0.57)	13.14–15.42	1.27[$]

aGRF = anterior ground reaction force, BW = body weight, CI = confidence interval, ES = pooled effect size, NP = negative power, P = power, PP = positive power, SD = standard deviation, SE = standard error, vGRF = vertical ground reaction force. [a]Data under reflect+logarithmic transformation, [b]Data under inverse transformation.
Significant differences between groups are [†] ($P < .001$), [$] ($P < .01$).

despite no statistically significant differences between groups regarding aGRF total, children with SCP presented reduced aGRF peak at push-off with respect to TD children, F (1, 55) = 11.674, $P = .001$, partial $\eta^2 = .175$, d = 1.270. Table 3 shows a detailed report of results regarding GRF and ankle joint power performance. Furthermore, differences can be visually examined on Figure 2.

3.4. Correlations

Regarding passive ankle joint positions, after controlling for the effects of velocity on gait kinetics, greater RJA accounted for a 25.4% increase in NP peak, F(1,39) = 13.890, $P = .001$; an 8.0% increase in PP peak, F(1,39) = 5.248, $P = .027$; and a 14.3% increase in aGRF peak at push-off, F(1, 39) = 7.866, $P = .008$. Moreover, greater MDF accounted for a 12.0% reduction in NP total, F(1,39) = 5.650, $P = .022$; a 10.0% reduction in PP total, F(1,39) = 6.024, $P = .019$; and a 25.2% reduction in NP peak, F(1,39) = 13.804, $P = .001$.

Regarding muscle architecture, after controlling for the effects of velocity on gait kinetics, longer RJA fL accounted for a 17.5% increase in aGRF total, F(1,39) = 8.936, $P = .005$; and a 13.3% increase in aGRF peak at push-off, F(1,39) = 7.183, $P = .011$. Similarly, longer MDF fL accounted for a 17.9% increase in aGRF total, F(1,39) = 9.132, $P = .004$; and a 16.3% increase in aGRF peak at push-off, F(1, 39) = 9.195, $P = .004$. Finally, longer RJA tL accounted for a 9.1% reduction in aGRF total, F(1,39) = 4.178, $P = .048$. Regarding muscle architecture ratios, greater RJA fLbl ratios accounted for a 14.5% increase in aGRF total, F (1,39) = 7.130, $P = .011$; and a 13.8% increase in aGRF peak at push-off, F(1, 39) = 7.517, $P = .009$. Similarly, greater MDF fLbL ratios accounted for a 17.2% increase in aGRF total, F(1,39) = 8.700, $P = .005$; and a 20.7% increase in aGRF peak at push-off, F(1, 39) = 12.542, $P = .001$. Moreover, greater RJA tLfL ratios accounted for 21.7% reduction in aGRF total, F(1,39) = 11.720, $P = .001$; and a 14.9% reduction in aGRF peak at push-off, F (1,39) = 8.251, $P = .007$. Similarly, greater MDF tLfL ratios accounted for a 20.2% reduction in aGRF total, F(1,39) = 10.660, $P = .002$; and a 16.6% reduction in aGRF peak at push-off, F(1,39) = 9.438, $P = .004$. Finally, greater RJA tLbL ratio accounted for a 9% reduction in aGRF total, F(1,39) = 4.125, $P = .049$; and a 10.3% reduction in vGRF peak at push-off, F (1,39) = 4.504, $P = .040$. Table 4 shows a detailed report of results regarding correlation analyses.

4. Discussion

The present study showed that children with SCP had a significantly altered MG muscle-tendon unit structure and impaired ankle joint kinetics with respect to TD children. We expected muscle-tendon unit structure would be related to ankle joint power performance during gait in children with SCP, however, no correlations were found. Nevertheless, relevant structural parameters were significantly related to aGRF generated at push-off, of great importance to support and progression during gait.

In accordance with previous studies of similar methodology, the present study observed that children with SCP had significantly reduced passive MDF.[22] This has been shown to determine differences in resistance to passive stretch in children with SCP where reduced MDF implied greater resistance to stretch.[38,39] Moreover, the present study observed altered MG muscle-tendon unit structure, which is in line with previous reports. Previous studies have reported that children with SCP had shorter MG muscle bellies[10,27,40] and longer Achilles tendons than TD children as seen in the present study.[10] However, previous studies have been inconclusive regarding fL differences between SCP and TD children.[11] While some studies have reported no differences in fL,[10,24,35,40] others support the present findings reporting that children with SCP have shorter MG fascicles than TD children.[27,41] Notwithstanding, the present study reported that children with SCP had greater tLfL and tLbL ratios than TD children. Similar differences in tLfL and tLbL ratios between TD children and those with SCP were extracted from previous studies.[10,27]

Regarding passive muscle-tendon mechanics, the present study observed no statistically significant differences in fE, bE, or tE between TD children and those with SCP. Similarly, a recent study reported no differences between TD children and those with SCP for bE.[27] However, contrary to our results, previous reports have observed children with SCP had reduced tE[27] and fE[10,27,42] compared to TD children. This may be attributed to the fact that our sample of children with SCP comprised children with and without dynamic equinus and mild MDF limitation, while studies showing reduced MG tE and fE had SCP samples of children with dynamic equinus and highly limited MDF angles with respect to TD children.[10,27,42] More specifically, the present study reported twice more MG fE than children with SCP walking in equinus.[10,27,42]

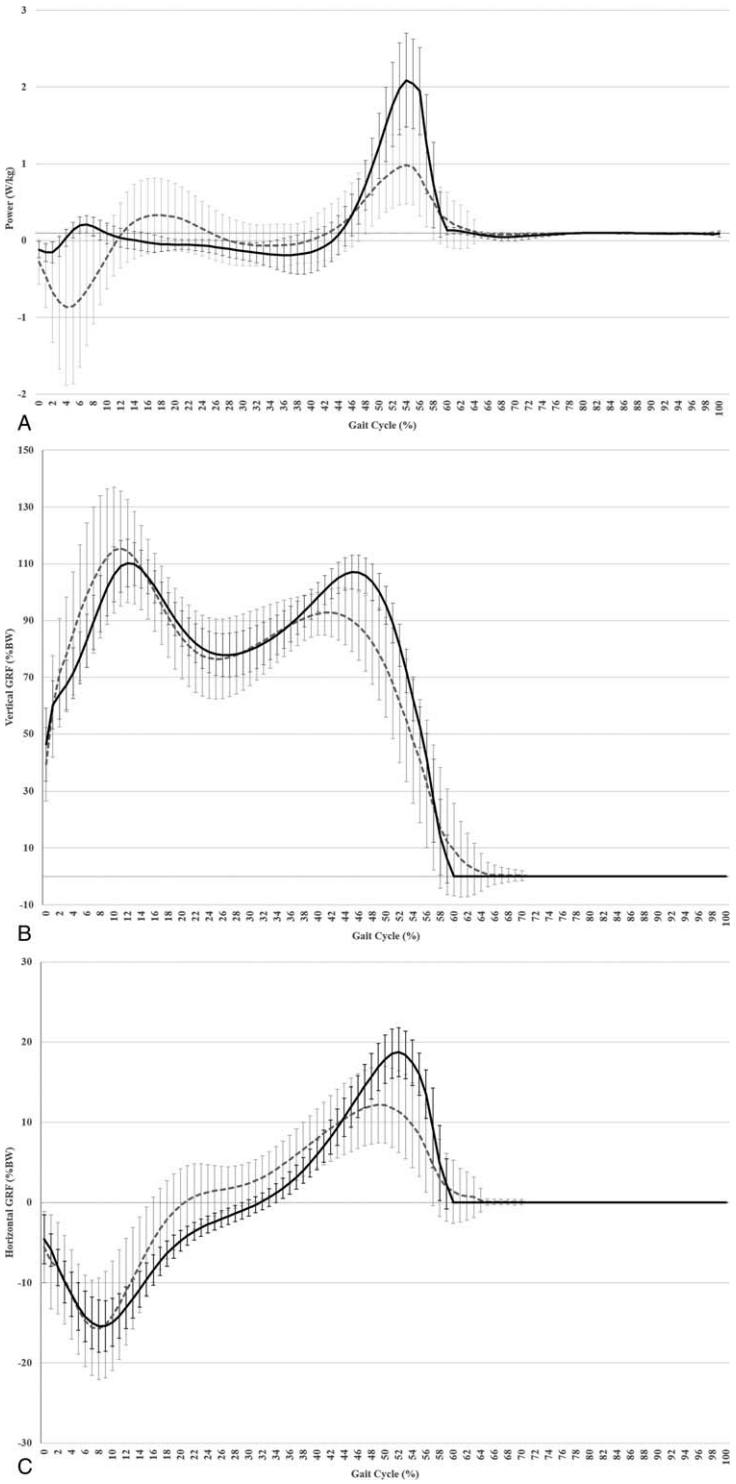

Figure 2. Ankle joint power (A), vertical (B), and horizontal (C) ground reaction force curves for typically developing children (continuous black line) and those with spastic cerebral palsy (dashed gray line).

Ankle joint power during gait reflected marked differences between TD children and those with SCP in most parameters measured. The present study reported increased early-stance NP peak and NP total as well as reduced PP peak and PP total, resulting in reduced P net for children with SCP with respect to TD children. Similar results have been previously reported for a sample of children with SCP and equinus gait.[3] Likewise, previous studies support that children with SCP have reduced PP peak with respect to TD children.[3,27,43–45] Furthermore, the reduction of P net is in accordance with the much greater hysteresis reported for children with SCP, indicating that less energy applied during loading is returned during unloading.[22] Regarding propulsive forces, the present study reported that children with SCP had reduced vGRF total and vGRF peak, and reduced aGRF peak which is in line with previous reports.[5,45]

Table 4

Part correlation coefficients (r) between passive ankle joint kinematics and medial gastrocnemius muscle structure, and gait kinetics in children with spastic cerebral palsy.

	NP total	PP total	P net	PP push-off	NP peak	PP peak	$_{v}$GRF $_{total}$	$_{a}$GRF $_{total}$	$_{v}$GRF $_{peak}$	$_{a}$GRF $_{peak}$
RJA	0.208	−0.259	−0.[068]	−0.045	**0.504b**	**−0.282**[a]	−0.061	−0.083	0.045	**−0.378**[b]
MDF	**0.346**[a]	**−0.317**[a]	0.009	0.067	**0.502**[b]	−0.253	−0.063	0.181	0.011	−0.093
ROM	0.158	−0.078	0.075	0.114	0.033	0.012	−0.006	0.269	−0.032	0.271
RJAfL	0.090	0.047	0.141	0.045	0.001	0.055	0.118	**0.419**[b]	0.092	**0.364**[a]
RJAtL	0.165	−0.151	0.004	−0.026	0.223	−0.037	−0.055	**−0.302**[a]	−0.297	−0.236
RJAbL	0.080	−0.085	−0.011	−0.008	0.075	−0.031	−0.085	0.246	0.243	0.176
MDFfL	0.092	0.086	0.184	0.056	−0.060	0.040	0.151	**0.423**[b]	0.058	**0.403**[b]
MDFtL	0.062	−0.034	0.026	0.034	0.186	−0.013	−0.072	−0.255	−0.137	−0.222
MDFbL	0.151	−0.049	0.099	0.046	0.121	−0.041	−0.099	0.230	0.156	0.128
fE	0.003	0.138	0.151	0.036	−0.193	−0.019	0.110	0.106	−0.079	0.132
tE	−0.167	0.187	0.033	0.103	−0.054	0.032	−0.012	0.056	0.252	0.111
bE	0.203	0.027	0.233	0.104	0.142	−0.051	−0.051	0.050	−0.124	−0.164
RJA fLbL $_{Ratio}$	0.060	0.084	0.151	0.042	−0.051	0.074	0.195	**0.381**[a]	−0.048	**0.371**[b]
MDF fLbL $_{Ratio}$	0.000	0.156	0.167	0.049	−0.183	0.076	0.264	**0.414**[b]	−0.042	**0.456**[b]
RJA tLfL $_{Ratio}$	0.015	−0.085	−0.076	−0.026	0.109	−0.047	−0.120	**−0.466**[b]	−0.145	**−0.386**[b]
MDF tLfL $_{Ratio}$	−0.015	−0.085	−0.106	−0.016	0.138	−0.031	−0.147	**−0.449**[b]	−0.081	**−0.408**[b]
RJA tLbL $_{Ratio}$	0.050	−0.035	0.013	0.002	0.089	0.005	0.011	**−0.300**[a]	**−0.321**[a]	−0.224
MDF tLbL $_{Ratio}$	−0.069	0.037	−0.030	0.009	0.017	0.038	0.002	−0.260	−0.182	−0.177

aGRF = anterior ground reaction force, bE = muscle belly excursion, bL = belly length, fE = fascicle excursion, fL = fascicle length, MDF = maximum dorsiflexion, NP = negative power, P = power, PP = positive power, RJA = resting joint angle, ROM = range of movement, tE = tendon excursion, tL = tendon length, vGRF = vertical ground reaction force. Significant correlations are [a]($P < .05$), [b]($P < .01$). Significant correlations in bold are [a]($P < .05$), [b]($P < .01$).

This is the first study to report on MG muscle-tendon unit structure contribution to GRFs and ankle joint power during gait in children with SCP. While this approach has been previously carried out, assessments were on the tibialis anterior of children with SCP, and only gait kinematics were analyzed.[46] Therefore, comparison with previous studies is limited. The present study reported the highest correlation coefficients for RJA and MDF on NP peak. Hence, a greater ankle RJA and a smaller MDF in children with SCP accounted for a significant increase in NP peak. This is in line with reports of greater power absorption in children with equinus gait and limited ankle MDF.[3] However, measures of MG muscle-tendon architecture or passive mechanics were not correlated to ankle joint power variables. We believe this might be attributed to the inherent differences between passive joint mechanics and active functional mechanics of gait. Hence, correlations or lack thereof should be considered with caution. Furthermore, the MG is a biarticular muscle, and as such, knee kinematics and kinetics would affect ankle performance during gait. Future studies should consider measuring ankle joint power analytically. Nevertheless, significant correlations with aGRFs generated at push-off were identified, with shorter fascicles at RJA and MDF accounting for significant reductions in aGRF total and aGRF peak. Accordingly, small fLbL ratios and large tLfL ratios accounted for significant reductions in aGRF total and aGRF peak.

These findings may be of great clinical relevance as most neuromuscular deficits associated with gait impairments are treated at the muscle-tendon level in children with SCP[2,47]. Subsequently, widely used treatments such as muscle-tendon lengthening,[48] botulinum neurotoxin type A injections,[29] casting,[30] prolonged orthotic use,[27] chronic stretching,[49] and different types of strengthening interventions[50] generate muscle-tendon adaptations which may in turn affect function. For example, despite functional gains outweighing muscle atrophy, prolonged use of ankle-foot braces has been shown to decrease fascicle length and reduce late stance ankle moment generation.[27] However, authors suggested concurrently improving muscle architecture for a more sustainable change in function, as seen for example through passive-stretching combined with active-movement training.[51] Thus, in depth understanding of the interrelations between treatment strategies, muscle-tendon unit adaptations, and function has become crucial for the proper management of the child with SCP.

There are several limitations which should be considered in the present study. First of all, because tests were time consuming, it was difficult to match the number of children in the TD group to that of SCP. The latter had come to the movement analysis laboratory for a follow-up analysis and less additional time was required for testing. Furthermore, the SCP sample was comprised of children with mild functional limitation, with or without dynamic equinus, and the neural component of hyper-resistance was not considered. Therefore, a more homogeneous sample taking stretch hyperreflexia into account may have better elucidated correlations of muscle-tendon structure to gait kinetics.[52] Regarding assessments, while ultrasound has been established as one of the most common methods to study real time MG muscle-tendon structure,[53] it is not without potential measurement errors.[32] Thus, for the present study, we incorporated previously reported error reduction techniques in the measurement protocol design. Unfortunately, though care was taken to maintain probe orientation throughout scans, this was not controlled for and measurement errors may have been introduced.

In conclusion, the present study supports previous findings of altered MG muscle-tendon unit structure in children with SCP. Furthermore, observations of impaired GRFs and ankle joint power during gait in children with SCP were reported and add to scarce literature on the matter. Finally the contribution of fL, fLbL ratio, and tLfL ratio to aGRFs generated at push-off should be taken into account, as these parameters have been shown to respond to usual MG muscle-tendon treatments.[10,27,29,49,50] Therefore, conventional treatment options should be revised on the basis of how they affect muscle architecture. Literature on the latter is scarce and should be considered for future studies, in

addition to studies which elucidate relationships between muscle architecture and function.

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