MEDIAL GASTROCNEMIUS SPECIFIC FORCE OF ADULT MEN WITH SPASTIC CEREBRAL PALSY

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Abstract

Introduction: Muscle weakness determines functional impairment in spastic cerebral palsy (SCP). Measurement of specific force (SF) allows for strength comparison with unimpaired populations (controls) accounting for neural (activation and coactivation), architectural (fascicle length and pennation angle), and structural differences (moment arm length).

Methods. Medial gastrocnemius (MG) SF (and its determinants) was assessed in both paretic and non-paretic legs of 11 men with SCP and 11 age-matched controls during plantarflexion maximal voluntary isometric contraction (MVIC).

Results. SCP fascicles were 28% longer than controls (P<0.05)**.** Pennation angle of SCP was 41% smaller than controls. The PCSA of SCP MG was 47% smaller than controls (P<0.05). There was no difference in SF between controls and SCP.

Discussion: Weakness in SCP is primarily attributable to deficits in agonist activation and muscle size; consequently, SF measured in the MG is similar between SCP and controls.

Keywords: Cerebral Palsy; Muscle architecture; PCSA; Medial gastrocnemius; Specific force; Ultrasonography.

Introduction

Muscle weakness in children with spastic cerebral palsy (SCP) has been shown to originate from impaired neural signalling, smaller muscle size, and altered architecture in the paretic musculature [1-4]. Such weakness of the paretic muscles have been shown to contribute to differences in gait patterns [5] and to limit motor control performance [6,7]. Although muscle weakness may limit the performance of daily tasks, only a few studies have addressed the underlying determinants of weakness specifically in adults with SCP.

It has been reported that larger deficits in weakness exist in the more distal paretic muscles of the lower limbs in individuals with SCP [8]. With this in mind, Elder et al. [1] reported that isometric plantarflexion (PF) torque of the paretic limb relative to the anatomical cross sectional area (ACSA; $Nm \cdot cm^{-2}$) in children with hemiplegic SCP was ~40% lower than either the non-paretic limb or individuals without neurological impairment. Similarly, while such findings are crucial to furthering our understanding of the determinants of muscle weakness, it has been well documented how ACSA measurements underestimate the true physiological cross sectional area (PCSA) of pennate muscles [9-11]. In support of these findings, correlations between muscle force during PF maximal voluntary isometric contraction (MVIC) and PCSA have been shown to be considerably higher than correlations with ACSA ($r = 0.72$ vs $r = 0.92$, respectively[12]).

Although muscle size is the greatest determinant of muscle strength, architectural characteristics of pennate muscles are also known to influence contractile function. Changes in architecture as a result of resistance training [13] and bed rest interventions [14,15] have been suggested to impact the force output of a muscle in individuals without neurological

impairment. In children with spastic hemiplegic SCP, the resting fascicle lengths of the paretic muscle in the gastrocnemius have been reported to be smaller when compared to the muscle of children without neurological impairment [2] and the contralateral non-paretic limb [3]. On the other hand, resting fascicle pennation angle of the paretic medial gastrocnemius (MG) did not differ when compared to the non-paretic muscle of individuals with SCP and the dominant limb of control participants without neurological impairment [2]. Conversely, during PF MIVC trials of the MG in young adult men and women, the paretic fascicle length was not different from that of normal control participants [16]. It is for this reason that measures of contractile area in SCP should consider the possible morphological differences of the muscle, for example PCSA. Indeed, Barber et al [16] have shown how MG PCSA can account almost entirely for differences in PF MVIC torque between those with and without CP. However, a more complete assessment of the intrinsic strength of the muscle (and the neural and morphological determinants) would involve the measurement of specific force.

Specific force, defined as the fascicle force/PCSA, is a measure of intrinsic muscle strength, that accounts for these aforementioned architectural and morphological characteristics of the muscle, plus the moment arm length and neural determinants of strength (agonist activation and coactivation) [17,9,10]. Moment arm length is a primary determinant of the effective translation of muscle force to torque [18,19]. Despite the excessive plantar flexion and hypothetical impact this may have on the Achilles tendon moment arm [20], particularly given the joint deformation in the ankle [21], there appears to be some preservation of the muscle-joint configuration, at least in terms of indirect measures of the moment arm in children with SCP [22]. There is at present however, no information on moment arm lengths in the paretic and non-paretic limbs of adults with SCP.

In terms of neural impairment in SCP, increased coactivation of the antagonist [16] and reduced activation of the agonist [23] are known to contribute to strength decrements between individuals with and without SCP. Therefore, given the established neural [23], architectural [16], and possible

joint differences [20] of individuals with SCP, the aim of this study was to determine whether differences in strength at the fascicle level persist when these morphological and neurological factors are accounted for through the calculation of specific force. Consistent with other neuromuscular conditions that have shown evidence of lower specific force or muscle quality (e.g. sarcopenia [9] and disuse [24]), it was hypothesized that the MG specific force of the paretic limb would be lower than the non-paretic limb and the dominant limb of individuals without neurological impairment (hereafter, this group will be referred to as 'controls').

Materials and Methods

Participants

Twenty-two active and ambulant men gave written informed consent to participate in the study. Eleven of the participants had spastic hemiplegic CP [age = 21.2 (3.0) years, stature = 1.79 (0.10) m, mass = 70.0 (12.5) kg], and 11 control participants had no history of musculoskeletal or neurological impairment [age = 21.8 (2.2) years, stature = 1.81 (0.04) cm, mass = 79.0 (8.4) kg]. Each participant with SCP rated between II and III on the modified Ashworth scale and had been formally classified independently by individuals from the Cerebral Palsy International Sports and Recreation Association (CPISRA). All participants with SCP rated as level 1 on the Gross Motor Function Classification System (MGFCS). Both the paretic and non-paretic legs were tested in each participant with CP, whereas the dominant limb was assessed in the control participants. All participants were free from lower limb injury and had not received any form of medication to reduce the effects of spasticity within the last year. None of the participants had a history of any surgical procedures on their lower limbs that would have affected the data collection from the sites assessed. The study was approved by the local ethics committee at Manchester Metropolitan University and conformed to the standards set by the latest revision of the Declaration of Helsinki[25].

Protocol

Participants attended the laboratory on 2 occasions. During the first visit, familiarization was carried out, which included a series of 6 PF MVIC followed by a series of submaximal percutaneous electrical stimulations. During the second visit, participants were assessed for resting measures of muscle size and moment arm length, which were then followed by the MVIC tests.

Strength measurements

The PF MVIC torque was recorded with the participants secured to an isokinetic dynamometer (Cybex Norm, Cybex International Inc., NY, USA) in the seated position with the back angle reclined at 65 deg. The participant's knee was secured in full extension with Velcro straps, which were positioned proximal to the knee. All participants were able to achieve full knee extension in this position. The medial malleolus was aligned visually with the dynamometer's central axis of rotation, and 2 Velcro straps were used to secure the foot to the footplate in order to minimize heel displacement. The participant's hips were also secured to the seat to limit extraneous movement during PF MVIC trials. All participants warmed up by performing 3 submaximal isometric contractions with the ankle angle at 0 deg (the individual's anatomical zero), each of which were separated by a 1 min rest period. In this instance, 0 deg was defined as the foot at 90 deg to the tibia. After the warm up, the participant's ankle remained fixed at 0 deg, and 2 PF MVICs were obtained, separated by a 2 min rest period. Throughout all MVIC trials, participants were encouraged verbally to exert as much force as possible, and online visual feedback was provided on a monitor. Dorsiflexion (DF) MVIC was completed after the PF MVICs using the same testing posture and protocol in order to calculate tibialis anterior

(TA) coactivation. All but 2 of the SCP participants were able to achieve the 0 deg ankle position [maximum dorsiflexion ROM was -5.30 (0.48) deg for SCP participants[26]]. For these 2 participants "0 deg" was measured at 4 and 6 deg plantarflexion. It should be noted that fascicle length was longer in SCP during PF MVIC (see results), and passive torque at zero degrees was no different than in control participants [Control 19.5 (6.80) Nm, Paretic 22.7 (10.3) Nm]. Furthermore, based on the MVIC torque angle relation presented previously[16], the influence of this more plantarflexed position in these 2 participants could be estimated to have reduced their PF MVIC by ~5 Nm, and was considered unlikely to have influenced the significance of the results presented below.

Agonist Activation

In order to account for any deficit in MVIC torque in the quantification of specific force, 2 supramaximal stimuli were applied to the muscle (pulse width, 50 μs). The first simulus was applied during MVIC. The second was applied approximately 2 s after the first when the torque had returned to levels equivalent to that observed prior to MVIC, from which voluntary activation levels of gastrocnemius were calculated [23,17]. The stimulus was delivered by applying 2 percutaneous stimuli (DSVDigitimer Stimulator; Digitimer, Herts., UK) to the gastrocnemius using rubber stimulation pads (size ranging from 38 mm x 89 mm to 76 mm x 127 mm; Versastim; Conmed, NY, USA), both of which were placed transversely distal to the popliteal crease and myotendinous junction of the soleus. The amplitude of the stimuli was determined prior to interpolation while the participant was in a relaxed state, administering twitches starting from 50 mA and increasing in increments of 50-100 mA, until no further increase in twitch torque was quantified. The voluntary activation level of each participant was assessed using the trial that produced the highest contractile torque. Agonist activation was calculated by dividing the supramaximal twitch torque during MVIC by the post MVIC twitch

torque, consistent with Morse et al. [9]. If there was a deficit in muscle activation (a value <100%) and assuming a linear relationship between MVIC torque and agonist activation [27,28], a correction was made with PF MVIC, which was calculated as: (PF MVIC torque / 100) x deficit in voluntary activation. This value was subsequently added to the MVIC torque along with torque contributions in coactivation to estimate PF MVIC net torque.

Coactivation

TA electromyographic (EMG) activity was recorded using 2 pre-gelled, unipolar, 10 mm, Ag-AgCl percutaneous electrodes (Medicotest, Denmark). Boundaries of the TA were determined using ultrasonography to ensure accurate placement of each electrode along the mid-sagittal axis of the muscle and to reduce cross-talk. Two electrodes were placed distally at two-thirds of the TA length, and a reference electrode was placed over the lateral epicondyle of the femur. Prior to placement of the electrodes, the area was shaved and cleaned with an alcohol swab to remove residual skin cells and oils and reduce skin impedance. Raw EMG data were recorded at 2000 Hz, with high and low band-pass filters set at 10 and 500 Hz, respectively, with a notch filter set at 50 Hz. The integral of the root mean square of the raw signal 0.5 s either side of the MVIC PF torque was used to quantify the level of muscle coactivation. The torque produced by the DF during PF MVIC was estimated by assuming a linear relationship between torque and EMG activity, as previously reported [29]. The relative contribution of antagonist coactivation from the DF MVIC was added to estimate PF net torque along with any correction in agonist activation as aforementioned.

Muscle volume

B-mode ultrasonography (AU5, Esaote, Italy) was used to obtain several axial-plane images of the MG to measure ACSA [30]. The MG proximal insertion and the MTJ were marked to identify 50% of muscle length. Strips of Micropore™ tape were placed axially across the mid-line of the MG at approximately 3.5 cm intervals. These strips of tape were used as echo absorptive markers that project a shadow onto the ultrasound image to provide a positional reference into the scanned structures. With the probe in an axial plane, a recording of the probe moving from the medial to the lateral border of the MG was obtained. Individual images were extracted from the recording offline and used to reconstruct the muscle by overlapping anatomical landmarks and external markers, as has been described previously [30]. Image J software (version 1.34; National Institutes of Health) was used to measure the ACSA of the reconstructed MG, from which volume was estimated as described below.

The use of ultrasonography in the measurement of ACSA has been validated against MRI in the rectus femoris (R^2 = 0.90, CV = 6.7% [31]) and vastus lateralis (R^2 = 0.98, CV = 1.7% [30]). However, no current data exist on techniques for using a single ACSA measurement to predict MG muscle volume. Based on a previous approach[32], retrospective analysis of MRI scans from an adult male population were carried out to allow more accurate predictions of muscle volume in this study. Briefly, the MG from 11 adult men [age 24.7 (4.7) years, height 1.79 (0.08) cm, mass 76.9 (12.4) kg] had been scanned previously in the transverse plane in 10% increments, from 10-90% of MG muscle length using a 0.2 T MRI (E-Scan, ESAOTE Biomedica, Genova, Italy). At each 10% increment, the ACSA of the MG was measured (OsiriX medical imaging software, OsiriX, Altlanta, USA) and presented relative to the maximum ACSA. A third order polynomial curve was then fitted through the ACSA at each section relative to the maximum ACSA (equation 1, as follows). The MG volume was then estimated by integrating the regression equation over the measured length of the muscle at intervals equivalent to 10% of measured muscle length. Compared to MG volumes acquired from this subgroup using contiguous ACSA measures along the muscle length at 1cm intervals

(e.g. [33]), there was no significant difference in estimated MG volume [301 (65) cm^3 and 294 (83) cm^3]. The bias (± 95% confidence limits) tended towards negative, but was low (-7.63 ± 22.4 cm^3), equivalent to 3% of the measured volume. There was also a significant correlation between measured and predicted MG volume (r^2 = 0.86). Therefore, this regression-based approach was adopted using the previously validated ultrasonography measure of ACSA to estimate MG muscle volume.

It should be acknowledge that this regression is based on a healthy adult male population, and although the similar muscle length between the participants [controls = 25.7 (2.0) cm, SCP 24.5 (3.75) cm] suggests some homogeneity, differences in the distribution of ACSA along the length of the muscle (if present) could not be accounted for.

$$y = -3.6395x^3 + 1.838x^2 + 1.8061x \quad \text{(Equation 1)}$$

MG ACSA relative to maximum MG ACSA (y, where ACSA max = 1) expressed relative to muscle length (x, where 100% of muscle length = 1), from which seMGental volumes were estimated and summed to calculate MG volume from measured ACSA at 50% of muscle length.

Muscle architecture

At the point of peak PF torque during the MVIC trials, real time ultrasonography was used to record fascicle length and pennation angle during contraction synchronized with the measured PF torque values. The 5 cm, 7.5 Hz linear array probe was held on the mid-sagittal plane of the MG equidistant between the proximal and distal tendon insertions previously established by ultrasonography. Additionally, the probe was held perpendicular to the surface of the skin to obtain several visible fasciculi ranging from the superficial to the deep aponeuroses. After the PF MVIC trials were completed, the recording of the highest torque trial was analyzed offline using Image J software. Fascicle length was measured as the length between the

241 superficial and deep aponeuroses [34]. Pennation angle was defined as the insertion angle of
242 the fascicle into the deep aponeurosis [17]. Fascicle length and pennation angle were measured
243 at the time point of maximum PF torque, as it has been reported that pennation angle is
244 underestimated and fascicle length is overestimated during rest conditions by 18.1 deg and
245 17.0 mm, respectively [34]. Thus, in order to accurately calculate the intrinsic force-generating
246 capacity of the MG, data must be obtained during contraction, not during rest [17,35,9]. The
247 dimensions of the window used for analysis were 4.15 cm x 3.5 cm; in some cases fascicle
248 length was estimated using linear extrapolation if a whole image of the fascicle was not
249 available for direct measurement.

250

251 *PCSA*

252 The PCSA was estimated as the ratio of MG muscle volume to fascicle length [36,17].

253

254 *Moment arm length*

255 The tendon excursion method was used to estimate moment arm length during a passive
256 stretch trial on an isokinetic dynamometer by passively rotating the ankle to calculate tendon
257 excursion while in a seated position. The medial malleolus was visually aligned with the
258 dynamometer's central axis of rotation. Prior to the experimental trial, end dorsiflexion end
259 range of motion was identified by the experimenter by rotating the ankle at 1 $deg \cdot s^{-1}$, starting
260 from 15 deg PF, until discomfort caused participants to cease the stretch in dorsiflexion. This
261 velocity was chosen in relation to previous findings which elicited minimal neural activity
262 throughout passive stretch trials in individuals without neurological impairment [37,38]. During the
263 passive stretch, B-Mode ultrasonography was used to determine the displacement of the MG
264 MTJ throughout the passive stretch. MTJ displacement was measured relative to an

265 acoustically-reflective marker (a thin strip of Micropore^{TM} tape) secured to the skin proximal to
266 the MG MTJ.

267

268 The total change in MTJ displacement was divided by the change in ankle range of motion
269 (rad), to predict the moment arm length for each individual. This technique has previously
270 been validated using cadavers when assessing the moment arm length of the Achilles
271 tendon[39]. *In vivo*, the tendon excursion technique shows high agreement with the center of
272 rotation approach (R^2 = 0.76) but may underestimate by 2-8% compared to the MRI based
273 measures of the latter [40,41]. As previously mentioned, we observed no significant difference in
274 passive DF end ROM between the participant groups [Control -8.40 (0.16) deg, Paretic -5.30
275 (0.48) deg [26]]. Based on current measures of the Achilles tendon moment arm over the PF
276 ROM[41], this 3 deg difference in ROM would be equivalent to an underestimation of the moment
277 arm in SCP by 21 mm.

278

279 *Achilles tendon force*

280 Tendon force was calculated by dividing the net plantarflexion torque by the Achilles tendon
281 moment arm length [9,10].

282

283 *Fascicle force*

284 In order to estimate MG fascicle force, PF MVIC net torque was multiplied by the relative
285 contribution of the MG PCSA in the triceps surae muscle group. The relative PCSA of the PF
286 muscles have previously been used to determine the relative contribution of each muscle,
287 whereby the relative PCSA of the MG was found to account for 15.4% of the Achilles tendon

force [12]. Therefore, the force generated by the MG was calculated by determining ratio of MG contribution to Achilles tendon force. At present there are no complete data on the relative PCSA in the triceps surae of adults with SCP, therefore it is not possible to test the assumption that the MG contributes 15% of the Achilles tendon force. However, in the triceps surae of children with SCP there is a degree of homogeneity to the relative atrophy of these muscles. Compared to age-matched controls, the MG, soleus (SOL), and lateral gastrocnemius (LG) were 42, 39, and 36% smaller, respectively [42]. The calculation of specific force is therefore presented with the knowledge that at least in terms of muscle ACSA, there seems to be some degree of similarity in the relative differences between SCP and controls in the triceps surae.

The force generated by the MG muscle was subsequently divided by the cosine of the pennation angle measured during contraction to determine MG fascicle force.

Specific force

Specific force was calculated by dividing MG fascicle force by MG PCSA.

Statistics

All statistical analyses were performed using SPSS software (Version 19, SPSS Inc., Chicago Illinois). To ensure the data were parametric, the Shapiro-Wilk and Levene tests were utilized to assess the distribution and variance of the data. As there were no breaches of these statistical assumptions, independent *t*-tests were used to assess baseline anthropometric data between CP and control groups. To minimize type I error of the main outcome measures (as could occur with repeated ANOVA tests), a MANOVA was used to compare the differences and interactions in the joint torque, force, neural, and architectural variables (listed in Tables

311 1-3) of the paretic vs. non-paretic limbs vs. the dominant limb of control individuals. Statistical
312 significance was accepted at the $P < 0.05$ level, and all data are presented as mean (SD).

313

314 **Results**

315 Adults with SCP and matched control individuals were of similar age ($P = 0.575$), stature ($P =$
316 0.604), and body mass ($P = 0.061$).

317

318 *Torque and moment arm properties*

319 The PF MVIC torque produced by the paretic limb was 33% less than the non-paretic limb
320 and 46% lower than the control group (Table 1). No difference in PF MVIC torque was
321 identified between the non-paretic limb and control group ($P = 0.178$). During the PF MVIC
322 trial, net torque from the paretic limb was 30% lower than the control group (Table 1). However,
323 no difference was identified between the paretic and non-paretic net PF MVIC torque ($P =$
324 0.892), nor between the non-paretic limb and control groups ($P = 0.193$). No differences were
325 identified in DF MVIC torque ($P = 0.653$) or moment arm length ($P = 0.281$) between groups
326 (Table 1).

327

328 *MG muscle size and architecture*

329 There was no difference between the paretic and non-paretic MG fascicle lengths ($P = 0.070$)
330 and non-paretic and control MG fascicle lengths ($P = 0.929$; Table 2). However, the paretic
331 fascicles were 28% longer than in the control group (Table 2). The pennation angle at which
332 the fascicles joined the deep aponeurosis during PF MVIC in the paretic MG was 31% less

than the non-paretic limb and 41% smaller than the control group (Table 2). No difference in pennation angle was identified between the non-paretic limb and control group (P = 0.095).

The ACSA of the paretic MG was found to be 20% and 27% smaller than the non-paretic and control group MG, respectively (Table 2). No differences were identified between the non-paretic and control group MG ACSA (P = 0.601). The paretic MG volume was 28% smaller than that of the non-paretic and 30% smaller than the control group (Table 2). Similarly, the PCSA of the paretic MG was 41% and 47% smaller than the non-paretic and control group, respectively (Table 2). However, no difference was identified between non-paretic limb and control group when assessing MG volume (P = 0.574) and MG PCSA (P = 0.323). No difference between groups was identified when assessing MG length (Table 2; P = 0.095).

Force measurements

Achilles tendon force and MG force of the paretic limb was 41% lower than the control limb (Table 3). No difference in the non-paretic Achilles tendon force and MG force was established when compared to the paretic limb (both P = 0.100) and control group (both P = 1.000). The paretic MG fascicle force was 41% less than the non-paretic limb and 52% lower than the control MG fascicle force (Table 3). No difference between the non-paretic MG fascicle force and control group was identified (P = 0.370). Lastly, no difference between the groups was established when assessing MG specific force (P = 0.393, Table 3).

Discussion

This study assessed the specific force of the MG in active individuals with SCP. The purpose was to establish whether the specific force of the paretic MG and the variables used in its calculation differed when compared to the non-paretic limb and control participants. Contrary to the hypothesis, the main finding of the study was that there was no difference between the *in vivo* specific force of the paretic and non-paretic MG of active individuals with SCP and the muscle of control participants. Although specific force of the MG was the same across all groups, paretic fascicle force was 41% and 52% lower than the non-paretic and control group, respectively.

Consistent with the results of our previous work [23], the SCP participants demonstrated significantly lower levels of activation than unimpaired counterparts. The aim of the electrical stimulation in the present study was not to calculate the level of the activation deficit but to account for any neural contribution to weakness and allow for a more accurate measure of MG specific force. By accounting for the differences in MG activation and TA coactivation across the paretic, non-paretic, and control groups, the net PF MVIC torque remained 30% lower in the paretic limb when compared to the control group, but it was not different from the non-paretic limb. Prior to this correction, the paretic PF MVIC torque was 46% and 33% lower compared to the control group and non-paretic limbs, respectively. As previously discussed [23], the 3 times higher coactivation and 38% lower agonist activation of the CP group, therefore contributes to about 16% of the difference in PF MVIC strength between CP and controls. The remaining 30% is attributable to morphological or architectural properties of the muscle.

The majority of research concerning muscle weakness in individuals with SCP measures the MVIC torque generated by a muscle or group of muscles [16,43,1,4]. A limitation when assessing torque is that moment arm lengths between limbs and/or groups of individuals are not taken into account. As individuals with SCP may have structural deformities in the paretic limb as a result of increased tone of the muscle throughout maturation [44], it is possible that the internal structures between the paretic, non-paretic, and control limbs may be more prominent in adults, compared to pediatric populations. It has been established that the Achilles tendon moment arm increases in length with plantarflexion [45], and although previously hypothesized to be different in the ankle following gait kinematics [20], indirect measures at the wrist suggest some preservation of the moment arm in children with SCP [22]. In our study, based on the consistent foot angle of 0 deg, we observed no significant difference in the Achilles tendon moment arm between the SCP and control groups. Nevertheless, when the *in vivo* forces were calculated in the paretic Achilles tendon and MG, they were found to be 41% weaker than the control group, but with no difference between the paretic and non-paretic limbs. Although the difference in Achilles tendon moment arm observed in our participants was not significantly different, based on the measured values and the assumed underestimation (see methods), the paretic Achilles tendon moment arm is between 0.51-0.72 cm larger than controls. With all else being equal when calculated using the mean PF MVIC net torque in this SCP population, reducing the moment arm length by 0.51-0.72 cm would theoretically increase the tendon force in the SCP limb by 212-120 N, or approximately 5-10% of the measured value. As a result, assessment of specific force using joint torque rather than tendon force would overestimate the true force-producing capacity of the contractile mass. Accounting for moment arm lengths, muscle architecture, and neural properties facilitates assessment of the intrinsic material force-producing capacity of muscle *in vivo* [9,35].

In children with SCP, the morphology of the paretic MG during rest showed that fascicle length was 18% shorter compared to the non-paretic contralateral limb [3] and 16% shorter compared to age-matched controls [2]. Based on these previous findings, deficits in paretic fascicle length would imply that the number of sarcomeres in series is typically lower than in participants without SCP. However, in contrast to previous architectural data, we found that fascicle length during PF MVIC was 8% longer in SCP than in the control group. It is likely that the contrasting results we obtained reflect the nature of the measurement technique. Where previous studies have reported muscle architecture at rest, MG fascicle length in this study was measured at peak PF MVIC torque, as is consistent with the calculation of specific force [17]. Due to the spasticity in the MG muscle, the paretic foot of participants with SCP is typically in an equinus position. Where previous studies have reported shortened fascicles in SCP, it is likely that this is due to them being measured at a more plantarflexed or "relaxed" position [2]. In addition, as we measured MG fascicle length during PF MVIC, any difference in the tendon properties or force produced would influence the relative shortening experienced by individuals with and without SCP, as has been observed in the elderly [35]. At present however, tendon stiffness comparisons between those with and without SCP show no difference in Achilles tendon strain during MVC[16]. Although it should be noted in their comparisons Barber et. al., [16] conducted tendon measurements at maximal dorsiflexion (-6 deg in SCP, -21 deg in controls), in men and women. Therefore, direct comparisons regarding fascicle shortening may not be possible with the men in our study who performed PF MVC at 0 deg ankle angle and who had no difference in maximal dorsiflexion angles.

When neural and architectural factors were accounted for in this study, there was a difference in MG fascicle force between SCP and control participants that was almost entirely accounted for by the difference in PCSA, as evidenced by similar values for specific force between SCP and controls. As PCSA takes into account the volume and fascicle length of the muscle, it

provides a more accurate measurement of the true contractile area of pennate muscle [11]. We found that the paretic MG PCSA was ~45% smaller than the non-paretic limb and control group, similar to the observed difference in MG fascicle force (52% smaller). Such differences indicate that the paretic MG muscle has fewer sarcomeres in parallel compared to the non-paretic and control MG muscle, and any weakness at the whole muscle level is unlikely to be influenced by a decrease in the quality of the muscle at the fascicle level. Indeed, no difference in specific force was observed between the SCP and control group or between the paretic and non-paretic limbs of the SCP participants. This would initially appear to be in contrast to previous work which established that the size/strength relationship of muscles in individuals with SCP is reduced compared to individuals without neurological impairment (e.g. PF MVIC/ACSA torque [1]). However, as previously stated, based on the architectural differences between SCP and the control group, and the substantial neural contribution to reduced joint torque, the estimation of the size/strength relationship (e.g. MVIC/ACSA) may be erroneous unless these factors are considered. Indeed, where PCSA and coactivation have previously been included, the size/strength relationship appears no different between SCP and controls[16]. It is pertinent to consider however, that where lower size/strength indices have previously been reported in SCP even though neural factors are likely to contribute, there is an element that may be attributed to alterations in the collagen content of the extracellular matrix[46]. It is possible that inclusion of an elevated collagen content (or other non-contractile elements) in the calculation of muscle size would likely result in a lower size/strength relationship (e.g. MVC/ACSA[1]). This is particularly relevant, as the participants of Elder et. al., [1] were likely more impaired than in our study (most of those in the former study had undergone surgical procedures to the Achilles tendon compared to none of our participants), and it has been shown that contracture severity is linked to collagen content [46]. This is also reflected in the work of Barber et. al., [16] who showed no difference in PF MVC/PCSA in young adults with SCP classified on the MGFSC as level 1, or least impaired. At least in our study and the work of Barber et. al., where no difference in specific force is observed, it is consistent with the

direct observations that histological structure is similar between SCP and control muscle [47]. Indeed, despite the fact that collagen content has been reported to be elevated in SCP muscle, there is no evidence of its mislocalisation within the muscle [46].

Although consistent with the observations of others [16], several factors may potentially impact on the calculation of *in vivo* specific force reported in the present investigation. In the present study, moment arm was estimated during rest, whereas specific force should represent the data obtained during MVIC. The difference in Achilles moment arm length during MVIC is approximately 1.5 cm longer than resting measures in individuals without neurological impairment [45]. In addition, fascicle length was measured at a comparable length rather than the angle at which peak torque occurred [35] due to the fact that we used 0 deg ankle angle to control for the different resting angles between participants. However, as the plantarflexors are on the ascending limb of the force/length relationship, it is likely that specific force is underestimated [35]. To compare between individuals that have limited dorsiflexion ROM, a consistent joint angle was chosen for measurement of specific force at 0 degrees. As previously mentioned, compared to SCP, Achilles tendon strain at PF MVIC is no different than controls[16]. Nevertheless, the interaction of the muscle and the tendon has yet to be addressed in adult males with SCP at a matched ankle angle, consistent with the measurement of specific force.

Conclusion

This study has shown that the paretic MG of physically active individuals with SCP has a similar specific force-generating capacity to the non-paretic muscle and the MG of control individuals. This study also demonstrates how the pennation angle and fascicle length of the

paretic muscle at MVIC is different from the control group. Nevertheless, weakness (while accounting for neural properties, moment arm lengths, and muscle architecture) observed in the paretic MG can be primarily attributed to a smaller PCSA rather than to the intrinsic material properties at the fascicle level.

Abbreviations

Anatomical cross sectional area (ACSA)

Dorsiflexion (DF)

Electromyography (EMG)

Medial Gastrocnemius (MG)

Lateral Gastrocnemius (LG)

Soleus (SOL)

Maximal voluntary Isometric contraction (MVIC)

Physiological cross sectional area (PCSA)

Plantarflexion (PF)

Spastic cerebral palsy (SCP)

Standard deviation (SD)

Tibialis anterior (TA)

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Tables

Table 1. Joint torque, moment arm, and neural properties of the paretic and non-paretic limbs of individuals with SCP and controls.

	Paretic limb	Non-paretic limb	Control group
PF MVIC (Nm)	102 (55.8)*†	153 (47.7)	190 (26.7)
Net PF MVIC (Nm)	139 (59.5)*	160 (46.9)	198 (27.3)
DF MVIC (Nm)	17.5 (8.58)	21.3 (11.9)	20.8 (10.6)
Moment arm (cm)	6.05 (1.69)	5.08 (0.98)	5.54 (1.56)

*Difference between paretic and control groups ($P < 0.001$). †Difference between paretic and non-paretic groups ($P = 0.039$). ‡Difference between paretic and non-paretic groups ($P < 0.001$).

624 **Table 2**. Muscle size and architectural characteristics of the MG muscle in the paretic and
625 non-paretic limbs of individuals with SCP and controls.

	Paretic limb	Non-paretic limb	Control group
Fascicle length (cm)	3.70 (0.62)*	3.14 (0.56)	2.89 (0.47)
Pennation angle (deg)	25.7 (4.08)†§	37.2 (7.59)	43.4 (7.00)
MG length (cm)	24.5 (3.75)	26.8 (3.23)	25.7 (2.00)
MG ACSA (cm^2)	12.0 (2.62)‡‖	15.0 (2.23)	16.5 (2.90)
MG volume (cm^3)	195 (56)*¶	269 (62)	279 (52)
MG PCSA (cm^2)	52.3 (11.6)†#	89.0 (28.1)	98.8 (23.8)

626 *Difference between paretic and control groups (P = 0.0004). †Difference between paretic and
627 control groups (P < 0.001). ‡Difference between paretic and control groups (P = 0.001).
628 §Difference between paretic and non-paretic groups (P = 0.001). ‖Difference between paretic
629 and non-paretic groups (P = 0.028). ¶Difference between paretic and non-paretic groups (P =
630 0.0005). #Difference between paretic and non-paretic groups (P = 0.0001).

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633 **Table 3**. Force measurements in the paretic and non-paretic limbs of individuals with SCP and
634 controls

	Paretic limb	Non-paretic limb	Control group
Achilles tendon force (kN)	2.26 (0.57)*	3.34 (1.59)	3.81 (0.32)
MG muscle force (N)	347 (88.2)*	515 (244)	586 (161)
MG fascicle force (N)	388 (104)†‡	662 (317)	814 (205)
Specific force ($N \cdot cm^{-2}$)	7.53 (1.84)	7.37 (2.08)	8.65 (2.99)

635 *Difference between paretic and control groups (P = 0.010). †Difference between paretic and
636 control groups (P < 0.001). ‡Difference between paretic and non-paretic groups (P = 0.024).

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641 **Conflict of interest**

642 The authors declare that they have no conflict of interest.

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