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Propulsion strategy in running in children and adolescents with cerebral palsy

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ABSTRACT

Background: Running is a fundamental movement skill important for participation in physical activity. Children with cerebral palsy (CP) who are classified at Gross Motor Function Classification Scale (GMFCS) level I and II are able to run but may be limited by neuromuscular impairments.
Research question: To describe the propulsion strategy (PS) during running of children and adolescents with CP.
Methods: This cross-sectional study used kinematic and kinetic data collected during running from 40 children and adolescents with unilateral or bilateral CP and 21 typically developing (TD) children. Maximum speed, peak ankle power generation (A2), peak hip flexor power generation in swing (H3) and PS (PS = A2/(A2 + H3)) were calculated. Linear mixed models were developed to analyze differences between groups.
Results: Maximum speed, A2 and PS were significantly less in children with CP GMFCS level I than in TD children and significantly less in children in GMFCS level II than level I. For children with CP, A2 and PS were significantly smaller in affected legs than non-affected legs. In affected legs, H3 was significantly larger in children in GMFCS level II than GMFCS level I but not different between TD children and children in GFMCS level II.
Significance: The contribution of ankle plantarflexor power to forward propulsion in running is reduced in young people with CP and is related to GMFCS level. This deficit appears to be compensated in part by increased hip flexor power generation but limits maximum sprinting speed.

1. Introduction

Running is a fundamental skill developed in childhood which is necessary for participation in many recreational and sporting activities. The ability to run requires each leg to alternately propel the body into a flight phase [1]. People with CP, GMFCS level I or II, usually develop the ability to run, although they may lack speed or coordination [2]. Power for forward progression in running is provided during ground contact, primarily by the ankle plantarflexors at push-off, referred to as A2 [3]. The hip extensors also generate power in early stance, primarily to maintain a stable trunk but also to move the body over the supporting leg [1].

The ankle plantarflexors have short, pennate muscle fibres with a long, compliant tendon [4,5] and are elongated in mid-stance before shortening at push-off [4]. These features make them ideally suited to the efficient storage and recycling of elastic energy, which reduces the work of the muscle fibres and therefore expenditure of metabolic energy [4–6]. Hence the plantarflexors provide a large percentage of propulsive power at slower jogging speeds [7–9]. At higher running speeds ground contact time is brief and the ability of the plantarflexors to generate more power in less time becomes limited [10]. Further increases in velocity are achieved by increasing cadence [10], primarily by a faster pull-through of the femur by the hip flexors in swing, referred to as H3 [7,8].

During running, the centre of mass is accelerated forwards only during the last 40% of stance phase, which constitutes the propulsive phase of stance [11]. For this reason, investigations of strategies for forward propulsion have focussed on power generated by the plantarflexors at the end of stance. The relative contribution of ankle power to forward velocity has been termed propulsion strategy (PS) and is calculated using the formula A2/(A2 + H3) [8,9]. A higher PS indicates a relatively greater contribution of the plantarflexors to forward velocity and therefore higher metabolic efficiency due to use of the stretch shortening cycle [4–6]. In typically developing (TD) individuals, PS

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decreases with increasing velocity as the hip flexors generate power to increase cadence after plantarflexor power generation has reached a maximum [9]. In TD children, PS of 0.75–0.81 has been reported in jogging and 0.69–0.75 in fast running [8,9].

It has been demonstrated that reduced ankle power generation at push-off results in increased hip flexor power generation in swing during impaired walking [12] and during running in children with developmental coordination disorder (DCD) [8]. The strategy normally used to achieve high velocities is utilized at lower velocities to compensate for reduced ankle power generation. It is unknown whether children with CP use the same compensation strategy during running. In a descriptive study of running in children with diplegic CP, reduced power generation at the ankle, yet similar power generation at the hip, was reported in children with diplegic CP compared to children who were TD [2]. This suggests that ankle power is the limiting factor in running in people with CP, rather than simply a generalized reduction in limb strength and coordination. Determining whether ankle power generation is the limiting factor in running in people with CP would be useful as it would provide direction for intervention.

The three aims of the present study were therefore to (1) describe the PS and components of the PS (A2 and H3) of affected and non-affected legs of children and adolescents with CP across a range of running speeds from jogging to sprinting; (2) determine whether the PS and components of the PS (A2 and H3) are affected by the level of gross motor function as delineated by the GMFCS; and (3) determine whether PS, A2, H3 and maximum speed were different in children with CP compared to children who were TD. It was hypothesized that:

(1) Maximum speed of running would be fastest in TD participants followed by participants classified at GMFCS level I then GMFCS level II.
(2) In participants with CP the affected limb would demonstrate a lower PS, lower A2 and higher H3 than that of the unaffected limb.
(3) In participants with CP both the affected and non-affected limbs would demonstrate a lower PS, lower A2 and higher H3 than that of the TD participants, and that this difference would be largest in the GMFCS II group.

2. Methods

2.1. Participants

Baseline data of participants with CP who were recruited from a community service provider for a larger study conducted in 2015 investigating the effect of a training programme on running in children aged 9–18 years was utilized. The study was approved by the Ethics Committees of Princess Margaret Hospital for Children, Perth, Western Australia (201405SEP) and Curtin University, Perth, Western Australia (HR 219/2014). The trial was prospectively registered with the Australian New Zealand Clinical Trials Notification ACTRN12614000467639. Informed consent was given by the parent/guardian and where applicable, the participant.

The comparative TD data was obtained from a sample of convenience, from participants who were recruited from a cohort of active children aged between 10 and 12 years of age, as part of a different study. Informed consent was given by the parent/guardian and where applicable, the participant.

2.2. Gait data collection

Reflective markers 10 mm in diameter were placed on the skin of participants by an experienced physiotherapist using a modified Cleveland Clinic Foundation marker protocol [13]. Participants in the CP study wore their usual sport shoes; calcaneus and metatarsal markers were placed on the shoes over the relevant landmark. Orthotics extending above the malleoli were not permitted. TD participants performed their trials barefoot. A regression equation was used to calculate the hip joint centre [14]. Knee joint centres were calculated as the midpoint between the medial and lateral femoral condyles [15]. Ankle joint centres were calculated as the midpoint between the medial and lateral malleoli [15]. Inertial and geometric properties of the segments were based on previously published models [16,17].

After a warm up, participants were asked to run at three speeds along a straight walkway in the Curtin University Motion Analysis Laboratory. The speeds were: (1) jog “like a warm-up or like a jog around the oval at school”; (2) run “faster than jogging, but not your fastest” and (3) sprint “like you are in a race”. Ten metres were available before and after the force plates to allow for acceleration and deceleration. At least five trials at each speed were collected unless the participant was too fatigued to continue. A two-minute sitting break was permitted between speeds if required. Kinematic data were recorded by an 18-camera motion capture system at 250 Hz (Vicon T-series, Oxford Metrics, UK). Synchronized ground reaction forces (GRF) were collected at 1000 Hz using three in-ground force platforms in series (AMTI, Watertown, MA). Marker trajectories were labelled and filled using Vicon Nexus 2.5 (Vicon Motion Systems, Oxford, UK).

2.3. Data processing

All data were processed using Visual 3D™ version 6 (C-Motion, Inc.). Kinematic and force plate data were filtered at 18 Hz using a zero-lag 4th order Butterworth filter. An inverse kinematic model with 6-degrees of freedom and specified joint translation boundaries was used [39]. Normalized speed (defined as velocity of the pelvis divided by height), A2, H3 and PS were calculated for each stride. The location of the centre of pressure along the *Y* axis of the foot was identified at initial contact and foot-strike classified as rear-foot (posterior third of the foot), mid-foot (middle third) or fore-foot (anterior third).

2.4. Statistical analysis

For variables A2, H3 and PS all trials were used for analysis. For maximum speed, the fastest trial speed achieved by each participant was used. A2 and H3 were transformed using a Box-Cox transformation to correct right skewedness. A linear mixed model was developed in Statistical Analysis Software (SAS) for each variable. Model validity and optimization were confirmed by meeting convergence criteria and by the Akaike Information Criterion and Bayesian Information Criterion values. Interactions between fixed effects were assessed and excluded if not significant at $p < 0.05$. For all models, random effects were Subject and Subject*Side.

Firstly, the affected and non-affected legs of the participants with CP were compared. Non-affected legs were the non-affected leg of participants with hemiplegic CP, while affected legs were both legs of those participants with bilateral CP and the affected leg of participants with hemiplegic CP. For PS and transformed A2 the fixed effects were, limb status (affected/non-affected), GMFCS level and normalized speed. In the transformed H3 model there was a significant three-way interaction, hence affected and non-affected legs were analyzed separately. For affected legs, fixed effects were GMFCS level and speed, and for non-affected legs fixed effects were GMFCS level, normalized speed and GMFCS level*normalized speed.

Secondly, the affected legs of the participants with CP were compared to the legs of the TD participants. Fixed effects were GMFCS level, normalized speed and GMFCS level*normalized speed for PS, transformed A2 and transformed H3.

Thirdly, the non-affected legs of the participants with CP were compared to the legs of the TD participants. For transformed A2 and PS fixed effects were GMFCS level and normalized speed. For transformed H3 fixed effects were GMFCS level, normalized speed and GMFCS level*normalized speed.

Table 1
Participant demographics.

	CP cohort	TD cohort
Age, mean (SD)	12 years, 11 months (2 years, 9 months)	10 years, 2 months (6 months)
Gender, *n* (%)		
Male	25 (62%)	15 (68%)
Female	15 (38%)	7 (32%)
GMFCS level, *n* (%)		
Level I	25 (62%)	
Level II	15 (38%)	
CP distribution, *n* (%)		
Unilateral		
GMFCS I	11	
GMFCS II	8	
Total	19 (47%)	
Bilateral		
GMFCS I	14	
GMFCS II	7	
Total	21 (53%)	

	CP cohort		TD cohort
	GMFCS I	GMFCS II	
Footstrike pattern			
Jog			
Rearfoot	50%	55%	38%
Midfoot	23%	19%	26%
Forefoot	15%	23%	29%
Inconsistent	13%	3%	7%
Run			
Rearfoot	45%	50%	24%
Midfoot	26%	39%	40%
Forefoot	26%	11%	33%
Inconsistent	4%	0%	2%
Sprint			
Rearfoot	24%	48%	2%
Midfoot	37%	28%	41%
Forefoot	28%	20%	50%
Inconsistent	11%	4%	7%

SD = standard deviation; *n* = number; GMFCS = Gross Motor Function Classification System; % = percent.

3. Results

Participant characteristics are summarized in Table 1. Baseline data of 43 participants in the intervention study were available, three participants were excluded from the current study due to absence of a flight phase.

3.1. Maximum speed

Participants' maximum running speed was significantly slower in GMFCS I than in TD ($t = -5.65$; $p < 0.001$), and significantly slower in GMFCS II than GMFCS I ($t = -3.64$; $p < 0.001$) (Table 2).

3.2. A2

A2 was smaller in affected legs than non-affected legs ($F = 19.91$; $p < 0.001$) and smaller in GMFCS II than GMFCS I ($F = 9.61$; $p = 0.002$). In affected legs, A2 was smaller in GMFCS I than TD ($t = -10.75$ $p < 0.001$) and smaller in GMFCS II than GMFCS I ($t = -9.53$; $p < 0.001$). In non-affected legs, A2 was smaller in GMFCS I than TD ($t = -2.01$; $p = 0.045$) and smaller in GMFCS II than GMFCS I ($t = -3.01$; $p = 0.002$). Speed had a significant effect on A2, which increased exponentially with increasing speed in all limbs ($p < 0.001$) (Fig. 1). As normalized speed was considered as a fixed effect in the linear models, significant differences in A2 found between groups were independent of speed.

3.3. H3

In affected legs, H3 was significantly larger in GMFCS II than GMFCS I ($t = 2.36$; $p = 0.018$) but not significantly different between TD and GFMCS II ($t = -0.79$; $p = 0.427$). In non-affected legs, H3 was not significantly different between GMFCS I and II ($F = 2.84$; $p = 0.093$) or between TD and CP legs ($F = 2.77$; $p = 0.063$). Speed had a significant effect on H3, which increased exponentially with increasing speed in all limbs ($p < 0.001$) (Fig. 1).

3.4. Propulsion strategy

PS was smaller in affected legs than non-affected legs ($t = -3.06$; $p = 0.002$) and smaller in GMFCS II than GMFCS I ($t = -4.11$; $p < 0.001$). In affected legs, PS was smaller in GMFCS I than TD ($t = -2.54$; $p = 0.011$) and smaller in GMFCS II than GMFCS I ($t = -7.16$; $p < 0.001$). In non-affected legs, PS was not significantly different between TD and GMFCS I ($t = -0.74$; $p = 0.458$) but smaller in GMFCS II than GMFCS I ($t = -2.23$; $p = 0.026$). Speed had a significant effect on PS, which decreased with increasing speed in all limbs ($p < 0.001$). PS decreased more quickly in TD than GMFCS I affected legs ($t = 2.71$; $p = 0.007$) and decreased more quickly in GMFCS I affected legs than GMFCS II affected legs ($t = 3.93$; $p < 0.001$) (Figs. 1 and 2).

4. Discussion

The main finding of the present study is that the PS for running in children and adolescents with CP is smaller than the PS of TD children and adolescents. The PS is smaller in affected legs than non-affected legs and smaller in GMFCS II than GMFCS I (Fig. 2). A smaller PS indicates a relatively smaller contribution of the plantarflexors compared with the hip flexors to forward progression of the body. At slower running speeds, in affected legs, children with CP utilize the hip flexors

Table 2
Power and propulsion strategy by GMFCS level – mean of raw data across all speeds.

Group	Affected	Maximum normalized speed Mean (SD)	A2 Mean (SD)	H3 Mean (SD)	PS Mean (SD)
TD	TD	3.57 (0.21)	15.56 W kg^{-1} (4.95 W kg^{-1})	7.87 W kg^{-1} (5.71 W kg^{-1})	0.69 (0.12)
GMFCS I	Affected	2.89 (0.52)[a]	8.64 W kg^{-1} (3.91 W kg^{-1})[a]	3.33 W kg^{-1} (0.67 W kg^{-1})[a]	0.67 (0.11)[a]
	Non-affected		11.10 W kg^{-1} (3.83 W kg^{-1})[a]	3.37 W kg^{-1} (0.73 W kg^{-1})	0.73 (0.09)
GMFCS II	Affected	2.36 (0.44)[ab]	6.00 W kg^{-1} (2.93 W kg^{-1})[ab]	2.76 W kg^{-1} (0.59 W kg^{-1})[b]	0.59 (0.14)[ab]
	Non-affected		7.89 W kg^{-1} (3.93 W kg^{-1})[ab]	2.06 W kg^{-1} (0.64 W kg^{-1})	0.64 (0.19)[ab]

A2 = peak ankle plantarflexor power generation in stance; H3 = peak hip flexion power generation in swing; normalized speed = velocity/height; m = metres; s = second; SD = standard deviation; PS = propulsion strategy A2/(A2 + H3); TD = typically developing; GMFCS = Gross Motor Function Classification Scale; W = Watts; kg = kilogram; [a]significantly different to TD ($p < 0.05$); [b]significantly different to GMFCS I ($p < 0.05$).

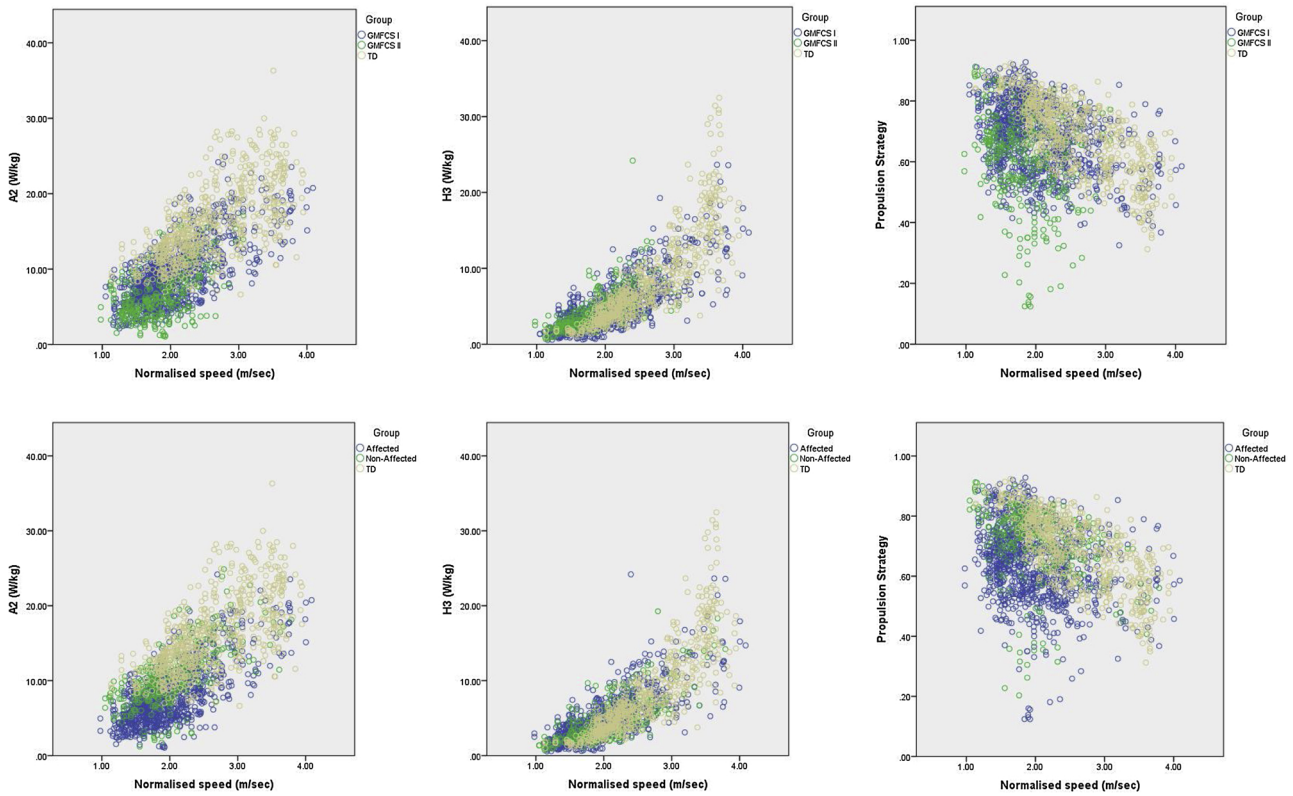

Fig. 1. Peak ankle plantarflexor power generation in stance (A2), Peak hip flexion power generation in swing (H3) and Propulsion Strategy A2/(A2 + H3) (PS), by normalized speed (velocity/height). W = Watts; kg = kilogram; normalized speed = speed/height; m = metre; s = second; GMFCS = Gross Motor Function Classification Scale; TD = Typically developing.

(a proximal strategy) to compensate for reduced plantarflexor power (Fig. 1), which has been reported previously in walking in children with CP [12] and in running in children with DCD [8]. In the present study, A2 was smaller in GMFCS I than TD and smaller in GMFCS II than GMFCS I. Conversely, there was no difference in H3 between groups in non-affected legs, and in fact H3 was larger in GMFCS II than GMFCS I in affected legs at the same speed. This suggests that in affected legs of children with CP, GMFCS level II, H3 is increased to compensate for a smaller A2. This is a strategy that is normally used at higher speeds by people who are TD [10]. In other words, children with CP utilize a normal proximal strategy at slower speeds to compensate for a distal power deficit.

The second main finding of this study is that reduced ankle plantarflexor power in children with CP appears to be the limiting factor for sprinting velocity. In the present study, maximum running velocity was slower in GMFCS I than TD and slower in GMFCS II than GMFCS I. This

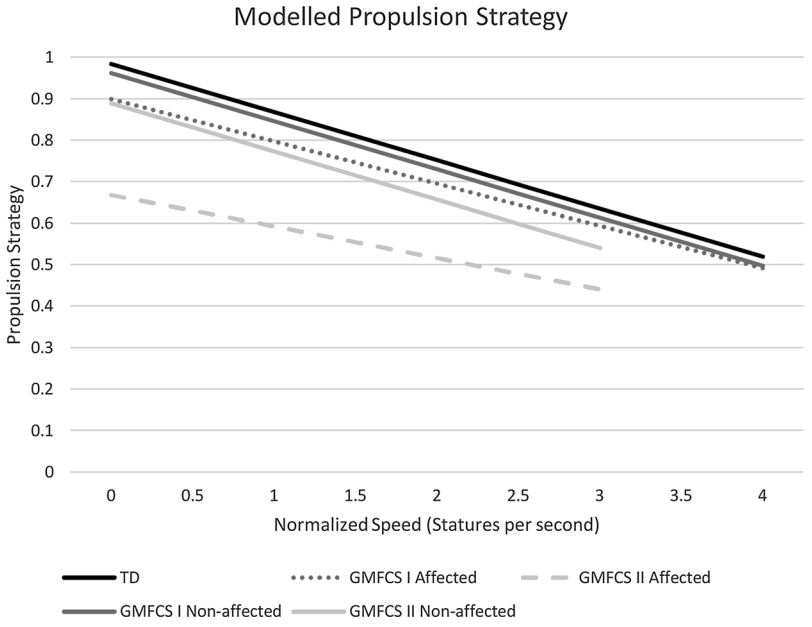

Fig. 2. Modelled Propulsion Strategy.

pattern matches the pattern of A2 in both affected and non-affected legs, which was also smaller in GMFCS I than TD and smaller in GMFCS II than GMFCS I. All groups increased H3 with increasing speed, however this occurred earlier in GMFCS II affected legs compared to GMFCS I affected legs, as demonstrated by both the increased magnitude of H3 in GMFCS II affected legs compared to GMFCS I affected legs at the same speed, and by the slower decrease of PS in GMFCS II affected legs than GMFCS I affected legs (Fig. 2). As the proximal strategy was already employed at slower speeds in GMFCS level II affected legs to compensate for reduced A2, there was less capacity to increase H3 to increase sprinting velocity. Ankle plantarflexor power generation has previously been identified as a limiting factor for running speed in TD sprinters [18] and in children with DCD [8].

A deficit in plantarflexor power in children with CP may be explained by muscle changes which limit the ability to utilize the storage and recycling of elastic energy, termed the stretch-shortening cycle (SSC). This is an important mechanism in running for metabolic efficiency [6]. The SSC is suited to long muscle fascicles and a stiff tendon [19]. During sprinting the plantarflexor muscle fibres act almost isometrically to enhance tendon stretch and recoil [5]. In young people with spastic CP the achilles tendon is longer and thinner than in the TD population which is likely to reduce the efficacy of the SSC [20]. The gastrocnemius muscle fascicles are shorter [20,21] and have a reduced ability to elongate which restricts dorsiflexion range [22], while spasticity may also restrict dorsiflexion range [23]. Hypothetically, the combination of hyperreflexia and reduced compliance of the musculotendinous unit in people with CP could be favourable for the storage and recycling of elastic energy, and therefore the activity of running. Children with CP, GMFCS level II are more likely to be able to run if they have gastrocnemius spasticity [24]. In the present study, children in GMFCS level II had greater maximum A2 with higher levels of spasticity compared to lower levels (Fig. 3). This finding suggests that gastrocnemius spasticity assists running in this group, although their maximum speed was slower than children in GMFCS level I. The largest maximum A2 in children in GMFCS level I occurred with moderate levels of spasticity. Those with either mild or strong spasticity had reduced maximum A2 compared to moderate spasticity (Fig. 3). These findings have implications for interventions which reduce spasticity, such as botulinum toxin injection and tone-reducing medication.

Spastic muscles have been reported to have reduced muscle belly volume, cross sectional area, thickness and length [21] compared to healthy muscles which is likely to result in a reduced capacity for force generation. Muscle weakness in CP may also result from poor muscle recruitment and activation [25]. In running, the ability to generate force rapidly is important [10,26] as ground contact time is very short. Rate of force development has been reported to be reduced in children with CP compared to TD children [26]. Increasing the power (force x velocity) of the plantarflexors in children with CP has been reported to improve walking velocity, cadence and step length [27]. Power training has been reported to be more effective than traditional strength training in increasing muscle fascicle length, muscle belly cross sectional area and velocity of movement in youth with CP [28]. The present study has demonstrated a plantarflexor power deficit in children and adolescents with CP during running which agrees with previous findings and suggests that increasing power generation of the plantarflexors would improve running performance in this population. It is yet to be determined whether power training of the plantarflexors can improve A2, increase maximum speed or reduce proximal compensation during running. This finding should also be considered in context when making decisions regarding interventions with potential to reduce plantarflexor power, such as botulinum toxin injection, calf lengthening surgery and the use of ankle–foot orthoses.

Measures of H3 in the present study were similar to values previously reported in the literature in children with CP [2,29] and children who were TD [2,30]. Measures of A2 in the present study were higher than those previously reported in the literature in children with CP [2,29] and TD children [2,8,30]. For example, A2 of 3–5 W kg^{-1} has previously been reported in children with CP running at 2.3–2.6 ms^{-1} [2,29], while the present study reports a mean A2 of 5.4 W kg^{-1} in GMFCS II and 7.5 W kg^{-1} in GMFCS I during jogging at 2.5–2.7 ms^{-1}. A2 of up to 14.4 W kg^{-1} has been reported in TD children sprinting at 4.2 ms^{-1} [8], while the present study reports A2 of 19.9 W kg^{-1} during sprinting at 5.1 ms^{-1}. We surmise that the higher values of A2 are due primarily to the faster self-selected speeds by participants in our study, compared to previous studies. The higher values of A2 compared to previously published results and the similarity of H3 to previously published results mean we can be confident that any significant lowering of PS is a true reflection of a deficit in plantarflexor power.

4.1. Limitations

A limitation of this study is that although data for both groups were collected in the same laboratory utilizing the same protocol, children with CP wore shoes while TD children were barefoot. Shoe-mounted markers tend to inflate ankle range of motion compared to skin-mounted markers in running [31]. Shod running tends to increase stride length [32] and increase the incidence of a rearfoot striking pattern [32,33] compared to barefoot running. Foot-strike patterns (Table 1) confirm that the CP group had a higher incidence of rearfoot strike than the TD group. Ankle plantarflexion moment has been reported to be 0.28 Nm/kg greater with forefoot strike pattern compared to rearfoot strike pattern [34] and ankle power generation has been reported to be the same [35] or slightly greater [32] in the barefoot condition compared to shod. Both these effects could exaggerate the differences between CP and TD groups. However, participants with CP in this study were on average two years older than the TD group and we did not control for age in our statistical analysis, although we did normalize speed by height. Both maximal speed and horizonal power increase with age in children (approximately 2 W kg^{-1} from pre- to mid-peak height velocity) [36,37] which in this study could reduce the differences between the CP and TD groups. Age and shod condition/striking pattern have opposing influences on the data and the magnitude of each is small compared to the differences found between groups in this study. This paper only considered intra-limb compensation. It is possible that there is an inter-limb compensation pattern and this would be worth investigating further.

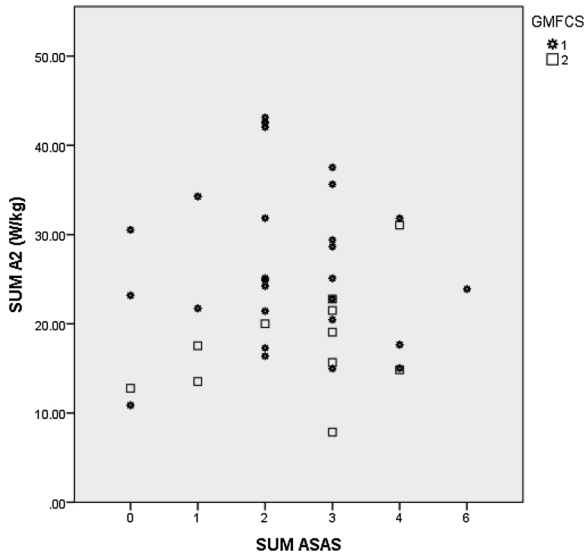

Fig. 3. Ankle Power Generation and Gastrocnemius Spasticity. A2 = Ankle power generation at push-off; ASAS = Australian Spasticity Assessment Scale [38]; GMFCS = Gross Motor Function Classification System; SUM = Left plus Right.

4.2. Conclusion

Children and adolescents with CP have reduced plantarflexor power generation at push off when running, which is compensated for by increasing hip flexor power generation in swing. It appears that reduced A2 limits maximum sprinting speed. Both A2 and maximum running speed are related to GFMCS level, with greater deficits in GMFCS II than GMFCS I.

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