PASSIVE MUSCLE MECHANICAL PROPERTIES OF THE MEDIAL GASTROCNEMIUS IN YOUNG ADULTS WITH SPASTIC CEREBRAL PALSY

[1]Lee Barber, [1]Rod Barrett and [2]Glen Lichtwark
[1]Musculoskeletal Research Program, Griffith Health Institute, Griffith University, Gold Coast, Australia;
email: l.barber@griffith.edu.au, web: www.griffith.edu.au/health
[2]School of Human Movement Studies, University of Queensland, Brisbane, Australia

SUMMARY
Individuals with spastic cerebral palsy (SCP) exhibit restricted joint range of motion and increased joint stiffness due to structural alterations of their muscles. Little is known about which muscle-tendon structures are responsible for these alterations. The aim of this study was to investigate the passive mechanics of the medial gastrocnemius (MG) muscle at both the joint and muscle fascicle level in young adults with SCP and compare this to typically developed (TD) individuals. Nine ambulant SCP (17±2 years) and ten TD individuals (18±2 years) participated in the study. Freehand 3D ultrasound was used to measure MG muscle volume. An isokinetic dynamometer rotated the ankle through its range while joint torque and ultrasound images of the MG muscle fascicles were simultaneously measured providing ankle torque-angle and fascicle strain data. For each individual we calculated MG force, physiological cross sectional area (PCSA), normalised ankle torque-angle and force-fascicle length data and corresponding stiffness. The MG muscles in SCP were significantly smaller in volume, experienced lower fascicle strain and were stiffer in absolute terms and when normalised for PCSA than for TD. The reduced MDF in the SCP participants may be attributed to greater muscle fascicle stiffness.

INTRODUCTION
Spastic cerebral palsy (SCP) is a group of non-progressive motor impairment syndromes that occur secondary to lesions of the brain in the early stages of development [1]. Spasticity is the key feature of SCP, and is neural in origin. However, it is clear that spastic muscle also undergoes significant structural alterations which contribute to muscle weakness, restricted joint range of motion and increased joint stiffness [2]. Passive mechanical properties of musculo-articular complexes have been studied experimentally in typically developed (TD) individuals [3] and subjects with upper motor neuron lesions with associated spastic muscle [4,5,6]. In the only study to date that has investigated individuals with SCP, the ankle joint of children aged 4-9 years with SCP was reported to be more stiff [6]. Mechanical properties have not been investigated at the fascicle level in individuals with SCP. The aim of this study was to investigate the passive mechanics of the medial gastrocnemius (MG) muscle *in vivo* at both the joint level and muscle fascicle level in young adults with SCP and compare this to TD individuals.

METHODS
Nine ambulant young adults with SCP, (6 males, 3 females, aged mean (1 SD) 17 (2) years, range 15-21 years) and ten TD young adults (5 males, 5 females, aged 18(2) years, range 15-20 years) participated in the study. MG volume was measured using freehand 3D ultrasound [7]. An isokinetic dynamometer rotated the ankle through its range while joint torque and ultrasound images of the MG muscle fascicles were simultaneously measured. Ankle stiffness was calculated as the slope of the torque-angle loading curve between 30-100% of maximal torque. Ankle torque was normalised by body mass and height. Slack angle was defined as the angle at which ankle torque exceeded 0 Nm. Joint angle was normalised to the slack angle. MG force was calculated as a function of ankle torque and the estimated Achilles tendon moment arm [8]. MG fascicle stiffness was calculated as the slope of the MG force-fascicle length loading curve between 30-100% of maximal force. Slack length of the MG fascicles was defined as the fascicle length when force exceeded 0 N. Physiological cross sectional area (PCSA) was estimated from the ratio of MG muscle volume to fascicle slack length. MG force was normalised by muscle PCSA and fascicle strain was calculated by the change in fascicle length (maximal fascicle length - slack length) as a percentage relative to the slack length. A between group general linear model was used to test differences in outcome measures between the SCP and TD groups.

RESULTS AND DISCUSSION
Absolute and normalised ankle torque-ankle angle and MG force-fascicle length data are presented in Figure 1. Outcome measures for the SCP and TD groups are presented in Table 1. At the joint level, the SCP group showed significantly reduced maximum dorsiflexion range of motion $6(1)^{o}$ versus $21(1)^{o}$ ($p<0.01$) and increased normalised ankle joint stiffness compared with TD individuals. These findings are in agreement with the previous study investigating the passive stiffness in ambulant children with SCP [6]. At the muscle level MG volume was 40% smaller in the SCP group but MG fascicle stiffness normalised for PCSA was 3.2 times greater than the TD group. In addition, fascicle strain was significantly less in the SCP group. Greater MG fascicle stiffness, in spite of much reduced muscle volume may suggest that the material properties of the fascicle and/or the tendon are different in the SCP versus the TD participants.

There was no significant correlation between the joint level stiffness and the fascicle level stiffness but in the SCP group MDF was correlated with fascicle strain ($r = 0.74$, $p = 0.02$), and MDF was negatively correlated with normalised fascicle stiffness ($r = -0.77$, $p = 0.02$) but not in the TD group.

CONCLUSIONS

The SCP participants had reduced ankle range of motion and increased normalised ankle stiffness compared to the TD individuals. While the MG muscles were significantly smaller in volume in the SCP group the MG fascicles exhibited significantly less strain and were stiffer in absolute terms and when normalised for PCSA. The data from the present study suggests that the reduced MDF during passive movement in individuals with SCP may be attributed to the inability of the muscle fascicles to elongate with added passive force.

ACKNOWLEDGEMENTS

This work was supported by funding from the National Health and Medical Research Council, Australia (Biomedical Postgraduate Scholarship Grant ID: 481953).

REFERENCES

1. Bax M, et al., *Developmental Medicine & Child Neurology*. **47**:571-6, 2005.
2. Gracies JM, *Muscle & Nerve*. **31**:535-51, 2005.
3. Gajdosik RL, *Clinical Biomechanics*. **16**:87-101, 2001.
4. Gao F, et al., *Arch Phys Med Rehabil*. **90**:819-26, 2009.
5. Hoang P, et al., *Clinical Biomechanics*. **24**:291-8, 2009.
6. Alhusaini AA, et al., *Developmental Medicine & Child Neurology*. **52**:e101-6, 2010.
7. Barber L, et al., *Journal of Biomechanics*. **42**:1313-9, 2009.
8. Grieve D, et al., *Biomechanics VI-A*. **2A**:405-12, 1978.

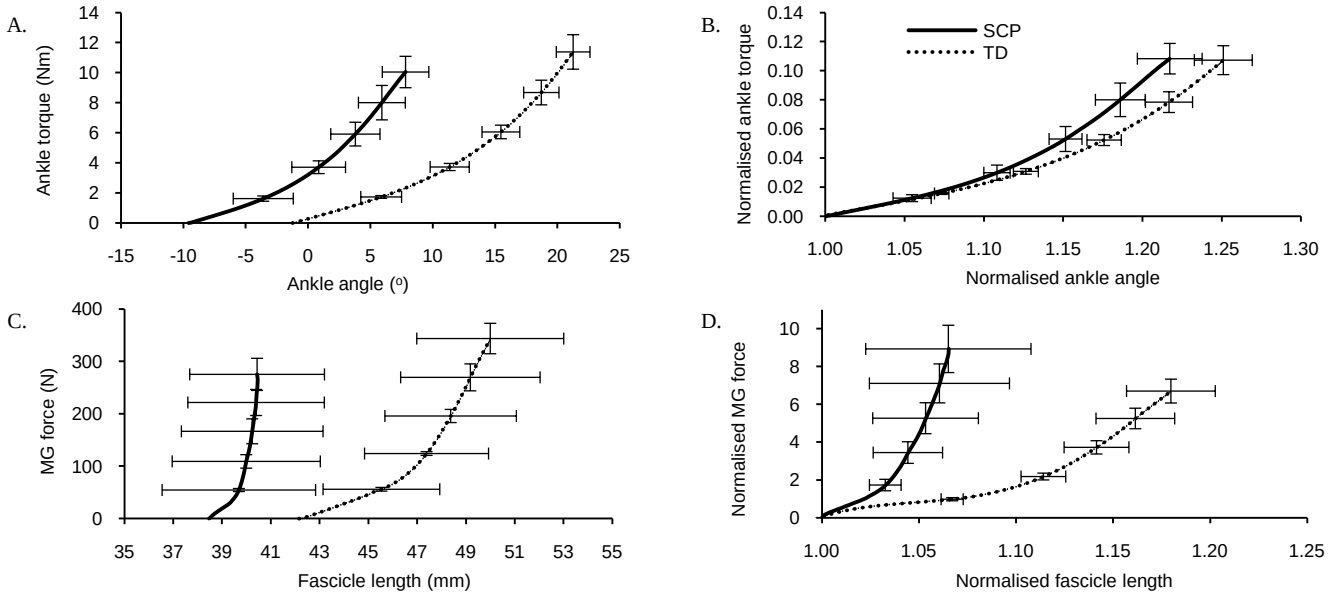

Figure 1. A. Ankle torque versus ankle angle, B. Normalised ankle torque versus normalised ankle angle, C. MG force versus fascicle length, and D. Normalised MG force versus normalised fascicle length in the SCP and TD groups. Data are mean (1 SEM). Positive ankle joint angle indicates dorsiflexion.

Table 1: Joint level and fascicle level outcome measures (absolute and normalised) for the SCP and TD groups. Data are mean (1 SEM). * $p<0.05$. ES, effect size.

	SCP	TD	p	ES
Joint level				
MG volume (ml)	134 (20)*	223 (21)	< 0.01	-1.5
Slack angle (°)	-8.4 (2)*	-1.0 (2)	< 0.01	-1.47
Ankle stiffness (Nm/°)	0.73 (0.1)	0.51 (0.04)	0.64	0.9
Normalised ankle joint stiffness (Ankle stiffness/kg/m)	0.65 (0.1)*	0.43 (0.03)	0.41	1.0
Fascicle level				
PCSA (cm^2)	34 (3)*	53 (5)	< 0.01	-1.6
Slack length (mm)	38.6 (3)	42.3 (2)	0.35	-0.43
MG fascicle stiffness (N/mm)	88.4 (19)*	42.4 (6)	0.02	1.1
Normalised MG fascicle stiffness (MG fascicle stiffness/PCSA)	114.1 (33)*	35.3 (5)	0.02	1.1
Fascicle strain (%)	10.8 (1.6)*	20.3 (1.4)	< 0.01	2.0