American Journal of Physical Medicine & Rehabilitation Articles Ahead of Print
DOI: 10.1097/PHM.0000000000002684

Impact of body-height increase on gastrocnemius muscle stiffness in children with cerebral palsy: A one-year prospective cohort study

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Author Disclosures:

a. Competing Interests:

None of the authors have any competing interests to disclose.

b. Funding, grants, or equipment provided for the project from any source:

This research did not receive any specific fundings, grants, or equipment from any funding agencies in the public, commercial, or not-for-profit sectors.

c. Financial benefits to the authors:

None of the authors received any financial benefits requiring disclosure.

d. Details of any previous presentation of the research, manuscript, or abstract in any form:

This research has not been presented at any conferences, and the manuscript and the abstract have not been published in any other journals.

Data Availability Statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Abstract

Objective: To investigate whether the impacts of height increase on gastrocnemius muscle (GM) stiffness are greater in children with spastic cerebral palsy (CP) than in those with typical development (TD).

Design: This one-year cohort study enrolled children (CP, 23; TD, 23) who underwent two measurements conducted at entry and after one year. Lateral and medial GM-strain ratios representing muscle stiffness were obtained using elastography.

Results: All regression equations (dependent variable, rate of change [RoC] of height; independent variable, RoC of the GM-strain ratios) were significant and all R^2s in children with CP (all $p < 0.001$; lateral GM's $R^2 = 0.81$; medial GM's $R^2 = 0.74$) were greater than those in children with TD ($p < 0.001$ and $R^2 = 0.49$; medial GM's $R^2 = 0.49$). The coefficients of equations in children with CP were significantly larger than those in TD ($p < 0.05$).

Conclusion: The greater R^2 values in CP than TD could explain how the variation in height predicts the variations in GM stiffness more accurately in CP than in TD. GM stiffness worsens more in children with CP than that in TD.

Key words: body height; cerebral palsy; elasticity imaging techniques; muscle

What Is Known

Most children with cerebral palsy (CP) show increased gastrocnemius muscle (GM) stiffness. The muscles of children with typical development (TD) stiffen as height increase. However, the muscles in children with CP show differential stretching compared to those in children with TD. Although the GM stiffness can increase with body-height, the relationship between the GM stiffness and body-height in children with CP has not been previously delineated in detail.

What Is New

The study presents evidence suggesting a stronger association between body-height and the GM stiffness in children with CP compared to typically developing children.

Introduction

Cerebral palsy (CP) encompasses a group of permanent disorders of movement and posture development caused by non-progressive brain lesions.[1] Although the underlying brain lesions are non-progressive, musculoskeletal impairments can show progression.[1] Spastic CP is one subtype of CP which accounts for 80% of children with CP.[2] Spasticity, defined as a velocity-dependent resistance to stretch due to reduced inhibition of the stretch reflexes, is a key feature in children with spastic CP.[3] Spasticity causes secondary alterations in muscle architecture,[4] including increased muscle stiffness, which can restrict joint range of motion (RoM) and gross motor function.[4,5] Most children with CP display increased stiffness of the gastrocnemius muscle (GM),[5,6] which causes restriction of ankle dorsiflexion (DF) -ROM, equinus deformity, and lower body function and structure.[5] Previous studies have reported that the risk of increased GM stiffness progresses with age.[6] Therefore, regular assessments of the GM stiffness are important.

A method for the measurement of increased GM stiffness using ultrasound elastography has been established in previous studies.[5] Ultrasound elastography, an ultrasonography technique, has the advantage of allowing the accurate measurement of individual muscles.[5] Strain elastography is a form of ultrasound elastography that involves the evaluation of muscle stiffness through the calculation of the muscle strain. However, children with CP undergo

rehabilitation not only in hospitals with the requisite facilities for strain elastography, but also in schools and homes that do not. As such, the identification of the specific conditions in when increased GM stiffness tends to worsen in children with CP, even in environments where strain elastography is not available, would facilitate the initiation of early treatment and prevent its worsening.

In children with CP, height can be an easily measurable indicator of increased GM stiffness. Children's height increases with age,[7] and increasing height indicates that long bones grow along the long axis.[8] Growing long bones cause increased muscle tension in the long axis.[8] This increased tension can stiffen muscles in children with typical development (TD).[8] Increased muscle stiffness in children is influenced by several factors, including muscle contractures caused by restricted joint movement and physical activity, and changes of muscle architecture caused by increased extracellular matrix;[4] however, increasing height is also an important factor affecting increased GM stiffness. Moreover, as the muscles in children with CP are less easily stretched along the long axis than those in children with TD,[9] increased height and increased GM stiffness could be associated in children with CP. Children's heights are measured regularly at schools in Japan under the Japanese School Health and Safety Act (Act number 56 of 1958). Non-medical professionals can also track height in children longitudinally, allowing easy collection of data. The clarification of whether GM stiffness tends to increase during the period

of increasing height in children with CP would enable prediction of the period at which muscle stiffness in these children tends to increase based on a regular evaluation of their height. However, the relationship between the increase in height and muscle stiffness remains unclear. Furthermore, although worsening of increased muscle stiffness can decrease the DF-RoM, the relationship between increasing height and decreasing DF-RoM has not been clarified.

Therefore, in the present study, we investigated the relationship between the increase in height and GM stiffness and the decrease in DF-RoM. We further hypothesized that GM stiffness would increase and DF-RoM would decrease with increasing height in children with CP compared with those with TD.

Methods

Design

This prospective, single-center, two-arm cohort study included children with CP and TD who underwent evaluation of height, GM stiffness, and maximum passive DF-RoM twice with an interval of one year.

Participants

Children diagnosed with spastic CP by a pediatrician were enrolled, in addition to children with TD living in the local community as volunteers. All participants were recruited between April 2020 and March 2022.

The inclusion criteria for children with CP were as follows: (1) age < 18 years old, (2) diagnosis of spastic hemiplegia, diplegia, or quadriplegia, (3) inpatients or outpatients treated at the Akita Prefectural Center on Development and Disability. The inclusion criteria for children with TD were as follows: (1) age < 18 years old, (2) children and their parents who saw the flyers recruiting the volunteers for the study distributed to the local community around the Akita Prefectural Center on Development and Disability, and who agreed to participate in the study on a voluntary basis.

The exclusion criteria in children with CP were as follows: (1) any surgery on the lower limb in the preceding 12 months, (2) receipt of any pharmacotherapy for decreasing spasticity in the preceding six months, (3) any lower limb injuries in the preceding six months, (4) and any inability to tolerate positioning for the study based on a previous study.[5] The exclusion criteria in children with TD were as follows: (1) any surgery on the lower limb in the preceding 12 months, (2) any lower limb injuries in the preceding six months, (3) and any inability to tolerate

positioning for the study based on previous studies.[5]

Prior to the current study, a pilot study enrolling 10 children with CP and 10 with TD was conducted to determine the appropriate sample size. The *z*-test, which can analyze whether the regression equations are parallel in both groups,[10] was conducted to assess whether the impact of an increase in height on GM stiffness and DF-RoM differed between groups. The rate of change (RoC) of height was included as the dependent variable. The RoCs of GM stiffness and DF-RoM were thus included as independent variables. The sample size was calculated using G*Power statistical software (ver. 3.1.9.7; Heinrich Heine University Düsseldorf, Düsseldorf, Germany).[11] The effect size was calculated as 0.89 and the power was calculated as 80%. Accordingly, for the effect size of 0.89, a power of 80%, and significance level of 5%,[12] the appropriate sample size was determined to be 46 participants across both groups. Consequently, we enrolled 60 participants (30 children with CP and 30 children with TD) who met the inclusion criteria to accommodate dropouts. However, 10 of the enrolled children (three children with CP and seven children with TD) could not participate owing to scheduling conflicts. Therefore, 50 children (27 children with CP and 23 children with TD) were finally recruited. The study protocol was approved by the Institutional Medical Ethics Committee of the Akita Prefectural Center on Development and Disability (approval number: 2023-05). The study was conducted in accordance with the Declaration of the World Medical Association and

the principles of the Declaration of Helsinki of 1975, as revised in 2013. All participants and their parents provided written informed consent for inclusion in the study. This study conforms to all of the STROBE guidelines and reports the required information accordingly (see Supplementary Checklist, http://links.lww.com/PHM/C683).

Procedures

All measurements, both at entry and after one year, were conducted by a single physiotherapist with five years of research experience in pediatric physiotherapy and ultrasonography. The timings of the measurements were adjusted to the individual schedules of the participants, to avoid taking measurements after physiotherapy, sports, and other exercises, as exercises that stretch the muscles can influence muscle stiffness.[13,14] Demographic characteristics including age, weight, and sex, as well as data on surgical history, previous pharmacotherapy, Gross Motor Function Classification System level,[15] and type of paralysis were obtained from the medical records.

The children's height was measured using a tape measure. However, the height of children with CP with hip and knee joints flexion contractures cannot be measured accurately.[16,17] Thus, height was calculated by summing the lines connecting six points on the body (top of the head, mastoid process, greater trochanter, median point of the lateral knee joint, lateral malleolus of

the fibula, and bottom of the foot), to avoid the effect of joint contractures.[16,17]

GM stiffness was evaluated using strain elastography (Noblus; Hitachi, Ltd., Tokyo, Japan) and a linear transducer (10 MHz). Measurements in children with CP were conducted on the more affected side, defined as the leg with a lower DF-RoM and for which the value of R2−R1 and the X score from Modified Tardieu Scale at planter flexor muscles was larger.[5,18] R1 indicates an RoM in which a sudden increase of muscle resistance is felt during a fast passive stretch.[18] R2 indicates an RoM at a slow maximum passive RoM.[18] A large value of R2−R1 indicates that the muscle is affected by spasticity.[18] The X score describes the types of muscle resistance when passive stretches are applied during the assessment (grade 0–5).[18] The measurements in children with TD were conducted on the side of the legs with a lower DF-RoM.[5] As a reference material for stiffness, an acoustic coupler (EZUTECPL1; Hitachi, Ltd., Tokyo, Japan) with an elastic modulus of 22.6 (standard deviation: 2.2) kPa was placed on the linear transducer. During the procedure, participants hung their feet over the edge of the bed in the prone position. The hip and knee joints were set at an extension angle of 0°. All measurements were conducted in a quiet room, during which participants were asked to refrain from speaking. Stiffness was measured in the lateral (LGM) and medial (MGM) GMs. The measurement points were located at the proximal 30% of the lower leg, from the head of the fibula to the lateral malleolus.[5,19] Measurements were performed in accordance with the methods described in previous

studies.[5,20] The two-screen mode was used for the measurements (Fig. 1). The transducer was rhythmically pressed, and the examiner confirmed that the pressure was within the appropriate range on the pressure graph (Fig. 1). The regions of interest were set on both the muscles and acoustic couplers. On the muscle side, the entire muscle on the monitor was selected to be as large as possible. On the acoustic coupler side, the height of the region of interest was set to the entire coupler, while the width was set to the width of the region of interest of the muscle. The strain of the muscle and that of the acoustic coupler were calculated using Noblus. Muscle stiffness was further obtained as the strain ratio (the amount of strain of the acoustic coupler divided by that of the LGM or MGM). The increased muscle stiffness compared with that in the control group resulted in a higher strain ratio of the muscle. Strain elastography measurements were conducted three times for each muscle. The average of the three strain ratios was used as the representative value.

The maximum passive DF-RoM was measured in 1°-increments using an international full circle goniometer (R-360-W, TIGER Medical Instruments Co., Ltd., Osaka, Japan). Participants were positioned on a bed in the supine position in the knee extension posture. The goniometer axis was located on the lateral malleolus.[21] The stationary arm was located along the lateral aspect of the fibular bone, while the movement arm was located along the lateral aspect of the fifth metatarsal bone.[21] The examiner dorsiflexed only the talocrural joints,[21,22] with the knee

joint at 0° extension (Fig. 2). Finally, the maximum RoM at which the participants did not feel uncomfortable was measured.[21,22] DF-RoM measurements were conducted thrice per measurement period, and the average of these three values was used as the representative value.

Statistical analyses

SPSS version 24 (IBM Corp., Armonk, NY, USA) was used for data analyses. Intraclass correlation coefficients (ICC) and coefficients of variation (CV) were calculated to confirm whether the strain elastography and DF-RoM measurements showed appropriate repeatability and reliability. If the ICC values were > 0.90 and CV values were < 10%, the repeatability and reliability were determined to be appropriate.[23]

Unpaired *t*-test and Mann–Whitney *U*-test were applied to confirm the significance of differences between children with CP and TD in terms of height, strain ratios for muscle stiffness, DF-RoM, and other demographic characteristics. The chi-squared test was applied to confirm the significance of differences between the groups in terms of sex.

The RoC was calculated as follows:

$$RoC\ \ (\%) = \frac{[(second\ \ value) - (first\ \ value)]}{first\ \ value} \times 100. \qquad (1)$$

Regression analyses were further performed to obtain regression lines for both groups. The

dependent variable was the RoC of height. The independent variables were the RoC of GM stiffness and that of DF-RoM.

The *z*-test was applied to examine the potential existence of parallelism between the regression lines in both groups.[10] The equation for the *z*-test is as follows:

$$z = \frac{x_1 - x_2}{\sqrt{\left[\left(SE_{x_1}\right)^2 - \left(SE_{x_2}\right)^2\right]}} \quad (2)$$

where $'x_1'$ and $'x_2'$ represent the coefficients in both groups and $'SE_{x_1}'$ and $'SE_{x_2}'$ represent standard errors associated with the coefficients in both groups.[10] A p-value of < 0.05 was considered statistically significant. Post-hoc power analysis to calculate the statistical power was conducted to confirm whether the analyses had reliably detected the differences in the effect of increased body-height on increased GM stiffness between the CP and TD groups.

Results

Following the sample size estimation based on the pilot study, this study enrolled 50 participants (27 children with CP and 23 children with TD). However, four children with CP who underwent surgery on their legs and received pharmacotherapy to decrease their spasticity were excluded from the statistical analyses. Finally, 23/27 (85%) children with CP and 23/23 (100%) with TD were included in the analyses (Fig. 3). All ICC values for the strain-ratio and DF-RoM

measurements were > 0.90, and all CVs were < 10%. The *z*-test indicated a statistical power of 61%.

The demographic characteristics of the two groups are shown in Table 1. There were no significant differences in age (p = 0.116), sex (p = 0.074), height in the first (p = 0.132) or second (p = 0.119) measurements, and RoC of height (p = 0.874) between the two groups.

We observed significant differences in the RoC of the LGM- and MGM-strain ratios and that of the DF-RoM between children with CP (mean LGM-strain ratio, 107.9%; mean MGM-strain ratio, 113.1%; mean DF-RoM, −145.4%) and those with TD (mean LGM-strain ratio, 40.2%; mean MGM-strain ratio, 40.3%; mean DF-RoM, −4.4%) (LGM, p = 0.002; MGM, p < 0.001; DF-RoM, p < 0.001).

The regression equations, in which the dependent variable was the RoC of the LGM-strain ratio and the independent variable was the RoC of height, were significant in both groups (Fig. 4a). The equation in the CP (p < 0.001) group was as follows:

$$RoC\ \ of\ \ LGM\ \ (\%) = 12.1 + 33.2 \times \left(RoC\ \ of\ \ height\ \ (\%)\right). \quad (3)$$

The equation in the TD (p < 0.001) group was as follows:

$$RoC\ \ of\ \ LGM\ \ (\%) = -18.5 + 19.9 \times \left(RoC\ \ of\ \ height\ \ (\%)\right). \quad (4)$$

The *p*-value obtained from the *z*-test was 0.008. The regression lines in both groups showed no significant parallelism.

The regression equations, in which the dependent variable was the RoC of the MGM-strain ratio and the independent variable was the RoC of height, were significant in both groups (Fig. 4b).

The equation in the CP ($p < 0.001$) group was as follows:

$$RoC\ \ of\ \ MGM\ \ (\%) = 39.0 + 25.7 \times \left(RoC\ \ of\ \ height\ \ (\%)\right). \quad (5)$$

The equation in the TD ($p < 0.001$) group was as follows:

$$RoC\ \ of\ \ MGM\ \ (\%) = -3.4 + 14.8 \times \left(RoC\ \ of\ \ height\ \ (\%)\right). \quad (6)$$

The *p*-value obtained from the *z*-test was 0.009. The regression lines in both groups showed no significant parallelism.

The regression equations, in which the dependent variable was the RoC of the DF-RoM and the independent variable was the RoC of height, were significant in both groups (Fig. 4c).

The equation in the CP ($p < 0.001$) group was as follows:

$$RoC\ \ of\ \ DF\text{-}RoM\ \ (\%) = -41.9 - 35.9 \times \left(RoC\ \ of\ \ height\ \ (\%)\right). \quad (7)$$

The equation in the TD ($p < 0.001$) group was as follows:

$$RoC\ \ of\ \ DF\text{-}RoM\ \ (\%) = 13.2 - 8.2 \times \left(RoC\ \ of\ \ height\ \ (\%)\right). \quad (8)$$

The *p*-value obtained from the *z*-test was 0.001. The regression lines in both groups showed no significant parallelism.

Discussion

Overall, the results of the present study revealed that GM stiffness and DF-RoM worsened with increasing height in children with CP compared with children with TD. These results indicate that it is possible to identify the period when the GM is most prone to stiffness in children with CP by measuring height, even without performing ultrasonography.

Both the CP and TD groups exhibited common changes in GM stiffness. The regression equations, in which the RoC of height was set as the dependent variable and the RoC of muscle stiffness was set as the independent variable, were significant in both groups, with positive coefficients. These results indicate that GM stiffness increased with an increase in height in both groups. An increase in height indicates growth of the tibia along the long axis,[8] which stretches the GM, causing it to become stiffer.[24-26] As such, our analyses confirmed that GM stiffness increases with increasing height in both groups.

The *z*-test was conducted to clarify whether there was a difference between the coefficients of the two groups. When the RoCs of the LGM and MGM stiffness were set as the dependent variables and the RoCs of height was set as independent variable, the coefficients in children with CP were significantly larger than those in children with TD. The difference in the coefficient could indicate that the GM stiffness increases linearly with height in children with CP compared with those with TD. Indeed, prior studies reported that height was lower in children with CP than in those with TD.[27,28] However, no significant differences in height or the RoC of height were observed between the two groups. As such, the difference in the amount of increase in height between the two groups is unlikely to affect the RoC of the GM stiffness.

The reason for the impact of the increase in height on GM was larger in children with CP than in those with TD could be explained by the characteristics of the spastic muscle. Indeed, the sarcomere length of the spastic muscle has been shown to be longer than that in muscles with TD.[4] Prior studies have reported that a lengthened sarcomere indicates that the myofibrils and muscles are overstretched.[4,29] Stretching an overstretched muscle beyond its original length can be difficult. Indeed, one previous study that conducted muscle biopsies and compared muscle extensibility in CP and TD reported that the muscle in the participants with CP was more difficult to stretch in the long-axis direction than that in the participants with TD.[9] The reason why GM stiffness in children with CP is likely to increase with height could be that the GM

cannot adapt to the tension increased by height in children with CP.

Overall, the results of the present study showed that an increase in height was significantly correlated with restriction of the DF-RoM, in addition to GM stiffness. Moreover, when the RoC of the DF-RoM was set as dependent variable and the RoC of height was set as independent variable, the coefficient was significantly larger in children with CP than in those with TD. Previous studies have shown that increased GM stiffness restricts the DF-RoM in children with CP.[4] Increasing GM stiffness with height may be a factor that decreases the DF-RoM in children with CP.

The primary strength of the present study is that our results revealed a significant correlation between increased body height and increased GM stiffness. Moreover, we found that this correlation was stronger in children with CP than children with TD. No study has yet focused on the impact of the increase in height on GM stiffness and DF-RoM. Children with TD experience a maximum increase in the time from age 1 to 2 years, with a subsequent maximum increase between 9 and 15 years of age in girls and between 10 and 17 years of age in boys.[7] The regression equations in the present study showed that a 1% increase in height caused a 45.3% increase in LGM stiffness, a 64.7% increase in MGM stiffness, and a 77.8% decrease in DF-RoM. Careful observation of longitudinal changes in height at these ages could assist in

considering the need to treat increased GM stiffness and restricted DF-RoM. Heights can be easily measured by medical staff such as nurses and community physiotherapists in schools without performing ultrasonography. The current study offers novel insight that even non-medical staff can determine the period when GM stiffness is likely to increase through the monitoring of longitudinal changes in height in children with CP.

Study limitations

One of the limitations of the present study was that it was not possible to analyze whether the participants underwent measurement during, before, or after their growth spurts. Further studies should therefore perform stratified analyses to determine whether participants are being investigated during the phase of a peak increase in height. Cohort studies with longer follow-up durations could investigate participants during, before, and after growth spurts.

Another limitation of the present study is that age, body height, and sex could act as confounding variables. Although we found no significant differences in age, body height, or sex between the two groups, the CP group trended towards being older and smaller, with a lower proportion of girls than the TD group, which may have influenced the results. In children with CP, differences in the affected body parts and level of Gross Motor Function Classification System could have affected the study results. Further studies stratifying patients by these

confounding variables may be needed.

Studies with low statistical power have a reduced chance of detecting a true effect.[30] The statistical power of the present study was calculated as 61%; this low statistical power indicates that if there is a true difference on the effect of increased body-height on GM stiffness, then our study has a 61% of determining a difference between CP and TD. The statistical power of the study is higher than the medians of the statistical power of other previous neuroscience and biomedical science studies.[30] However, this statistical power remains lower than the 80% which is generally considered to be the appropriate level.[30] This low statistical power indicates that further larger cohort studies are required to confirm our results.

Conclusions

The present study indicates that GM stiffness increases linearly in children with CP more clearly than in children with TD, and that the DF-RoM decreases linearly with increasing height in children with CP more than in children with TD. These results indicate the existence of a correlation between increasing height and worsening GM stiffness in children with CP. It is also possible that monitoring increases in height in children with CP could help to identify the preliminary stage of worsening GM stiffness.

Acknowledgments

The authors would like to thank Editage (www.editage.com) for the English language editing.

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Figure Legends

Fig. 1. The image shown in the strain elastography monitor.

Measurements were conducted in two-screen mode, with the strain elastography color-coded image on the left side and the B-mode image in black and white on the right side (a). Strain elastography images are shown in red (indicating soft tissue) to blue (indicating stiff tissue) (b). The examiner rhythmically pressed the transducer towards the GM within an appropriate pressure range. The examiner confirmed that the pressure was within the appropriate range on the pressure graph (c). The upper limit of the pressure force was set to 0.7 kPa. (The reader may refer to the web version of the article to facilitate the interpretation of the references to color in this figure legend.)

Abbreviation: GM, gastrocnemius muscle.

Fig. 2. Measurement of the ankle dorsiflexion range of motion.

Participants were placed in the supine position with a knee extension of 0°. The long axes of the tibia and the second metatarsal bone were aligned in the sagittal plane (a). The examiner dorsiflexed the ankle joint of the participants and measured the maximum RoM at which the participants did not feel uncomfortable in 1°-increments using a goniometer (b).

Abbreviation: RoM, range of motion.

Fig. 3. Flow chart of participant inclusion in this study.

Abbreviations: CP, cerebral palsy; TD, typical development.

Fig. 4. Comparison of regression equations between children with cerebral palsy and those with typical development, with the rate of change of height as the independent variable.

Children with CP are represented as black dots and children with TD are represented as white dots in dots in (a), (b), and (c). (a) shows the comparison of the regression with the RoC of LGM-strain ratio between the children with CP and those with TD. The coefficients in children with CP and TD were 33.2 ($p < 0.001$) and 19.9 ($p < 0.001$), respectively. Coefficients were significantly higher in children with CP than in those with TD ($p = 0.008$). (b) shows the comparison of the regression with the RoC of MGM-strain ratio between the children with CP and those with TD. The coefficient in children with CP and TD were 25.7 ($p < 0.001$) and 14.8 ($p < 0.001$), respectively. The coefficient was significantly higher among children with CP than in those with TD ($p = 0.009$). (c) shows the comparison of the regression with the RoC of DF-RoM between the children with CP and those with TD. The coefficients in children with CP and TD were −35.9 ($p < 0.001$) and −8.2 ($p < 0.001$), respectively. The coefficient was significantly higher in children with CP than in those with TD ($p = 0.001$).

Abbreviations: CP, cerebral palsy; TD, typically development; RoC, rate of change; LGM, lateral gastrocnemius muscle; MGM, medial gastrocnemius muscle; DF-RoM, ankle dorsiflexion range of motion; CP, cerebral palsy; TD, typical development.

Figure 1

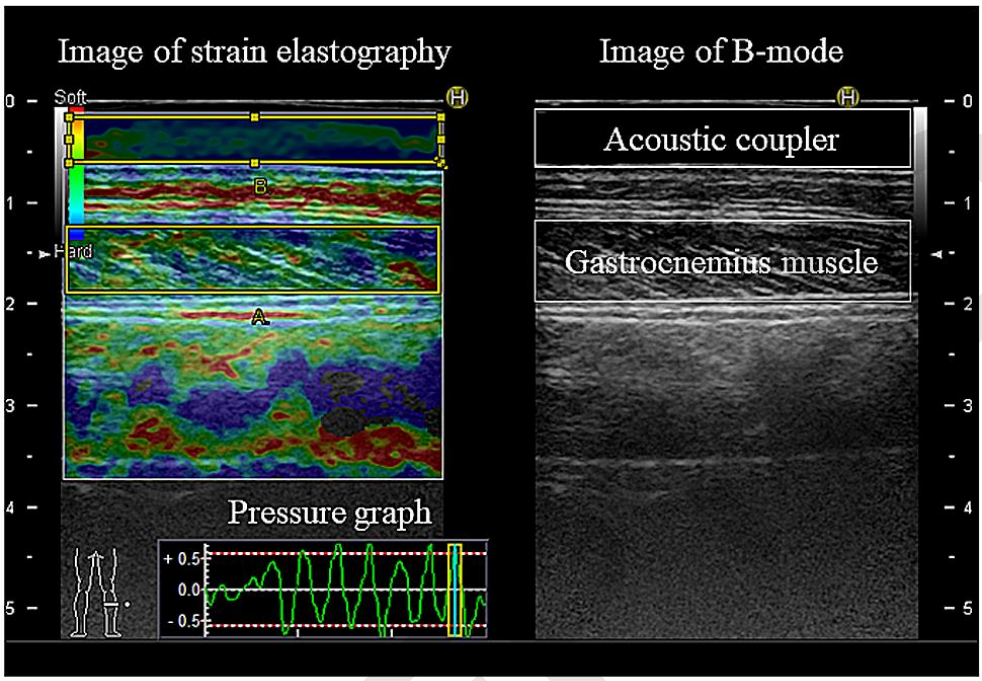

Figure 2

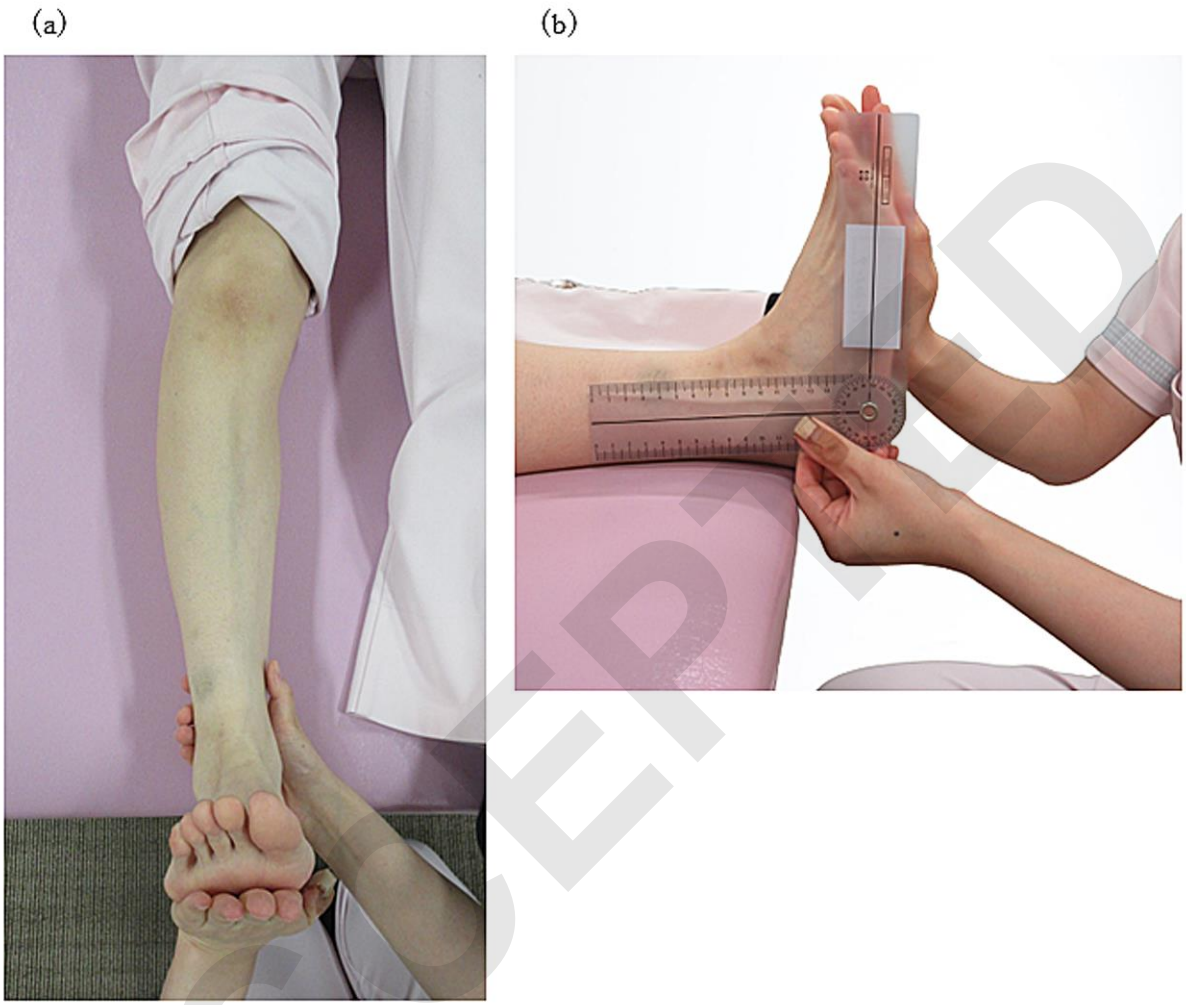

Figure 3

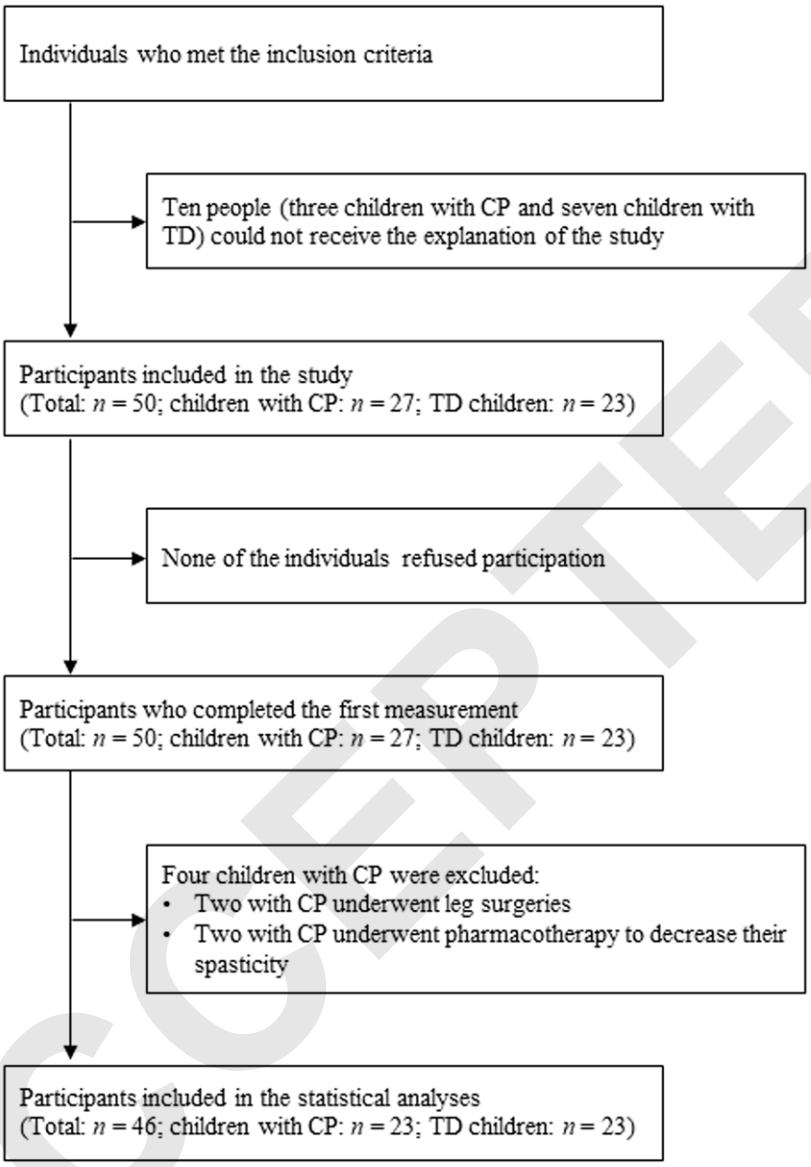

Figure 4

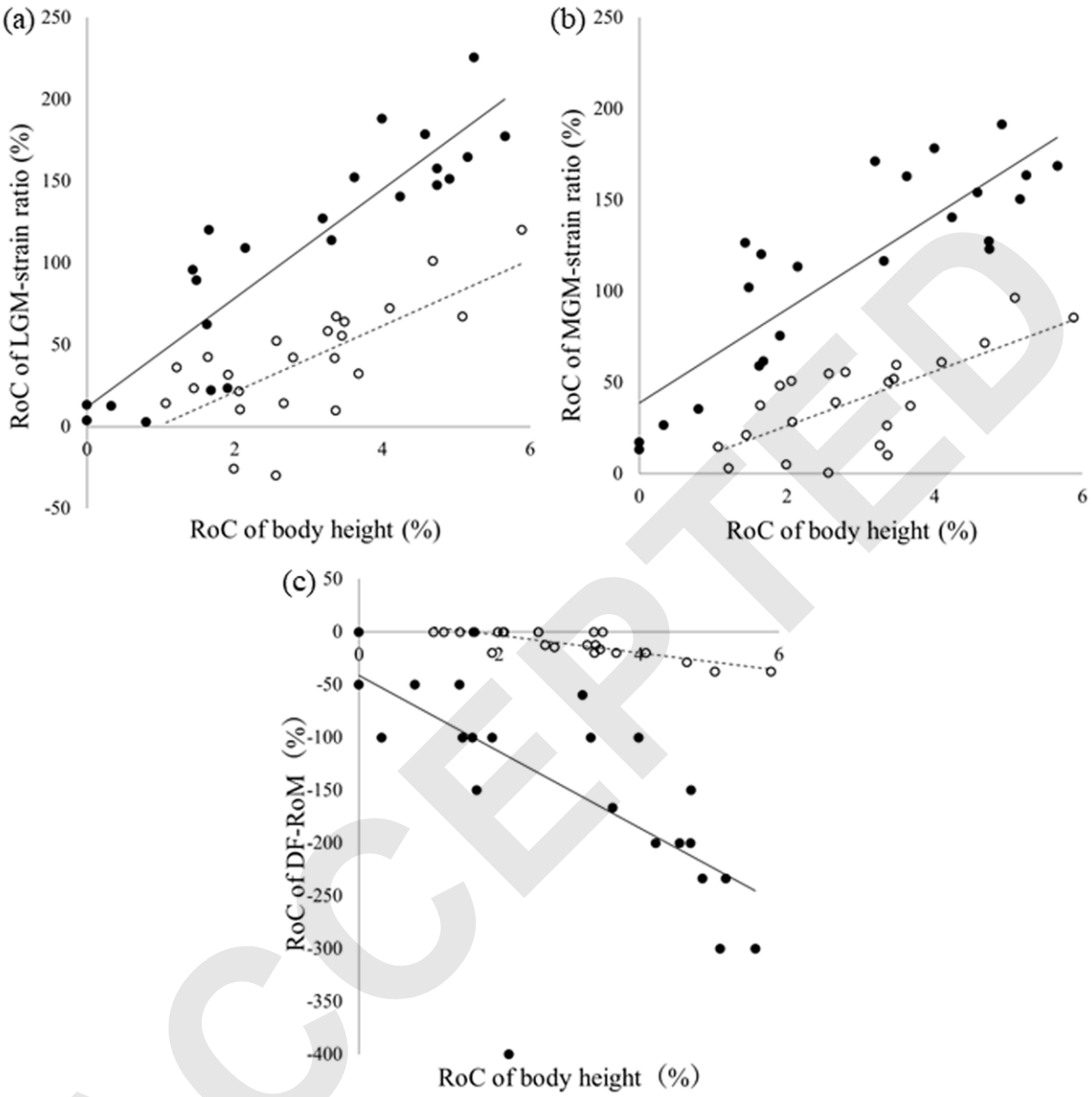

Table 1. Participant characteristics

	CP, n = 23 mean (SD)	TD, n = 23 mean (SD)	p-value
Age, years months	11 years 4 months (2 years 9 months)	10 years 5 months (2 years 5 months)	0.116*
Sex	10 females; 13 males	16 females; 7 males	0.074†
Gross Motor Function Classification System, n			
Level I	6		
Level II	4		
Level III	3		
Level IV	7		
Level V	3		
Body height			
First measurement, cm	137.7 (19.2)	144.6 (14.3)	0.132*
Second measurement, cm	141.5 (18.9)	148.8 (14.6)	0.119*
RoC, %	2.9 (1.8)	3.0 (1.2)	0.874‡
LGM stiffness			
First measurement	82.9 (52.1)	71.3 (53.3)	0.210*
Second measurement	181.2 (151.8)	92.5 (59.5)	0.019*
RoC, %	107.9 (67.4)	40.2 (35.1)	0.002*
MGM stiffness			
First measurement	83.6 (64.0)	125.1 (49.7)	0.002*
Second measurement	184.7 (165.8)	175.7 (74.8)	0.272*
RoC, %	113.1 (54.8)	40.3 (26.1)	< 0.001*
DF-RoM			
First measurement, °	−0.7 (2.5)	7.3 (2.4)	< 0.001*
Second measurement, °	−3.3 (3.2)	6.8 (1.9)	< 0.001*
RoC, %	−145.4 (101.5)	−4.4 (7.8)	< 0.001*

Abbreviations: CP, cerebral palsy; TD, typical development; RoC, rate of change; LGM, lateral gastrocnemius muscle; MGM, medial gastrocnemius muscle; DF-RoM, ankle dorsiflexion range of motion; SD, standard deviation; *, Mann–Whitney U-test; †, Chi-squared test; ‡, unpaired t-test.