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Does acute passive stretching increase muscle length in children with cerebral palsy?

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ABSTRACT

Background: Children with spastic cerebral palsy experience increased muscle stiffness and reduced muscle length, which may prevent elongation of the muscle during stretch. Stretching performed either by the clinician, or children themselves is used as a treatment modality to increase/maintain joint range of motion. It is not clear whether the associated increases in muscle–tendon unit length are due to increases in muscle or tendon length. The purpose was to determine whether alterations in ankle range of motion in response to acute stretching were accompanied by increases in muscle length, and whether any effects would be dependent upon stretch technique.

Methods: Eight children (6–14 y) with cerebral palsy received a passive dorsiflexion stretch for 5 × 20 s to each leg, which was applied by a physiotherapist or the children themselves. Maximum dorsiflexion angle, medial gastrocnemius muscle and fascicle lengths, and Achilles tendon length were calculated at a reference angle of 10° plantarflexion, and at maximum dorsiflexion in the pre- and post-stretch trials.

Findings: All variables were significantly greater during pre- and post-stretch trials compared to the resting angle, and were independent of stretch technique. There was an approximate 10° increase in maximum dorsiflexion post-stretch, and this was accounted for by elongation of both muscle (0.8 cm) and tendon (1.0 cm). Muscle fascicle length increased significantly (0.6 cm) from pre- to post-stretch.

Interpretation: The results provide evidence that commonly used stretching techniques can increase overall muscle, and fascicle lengths immediately post-stretch in children with cerebral palsy.

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1. Introduction

Children with cerebral palsy (CP) show increased muscle stiffness and reduced muscle length, which may contribute to reduced function. Stretching is commonly used in the treatment and management of children with spastic CP and is considered to be an important part of preventing or delaying the onset of contractures (National Institute for Health and Clinical Excellence, 2012). The assumptions made in clinical practice are that repeated bouts of stretching over periods of weeks or months can increase muscle length and reduce stiffness (Herbert, 2004; Odeen, 1981) by providing the necessary stretch stimulus that allows the muscle to lengthen in line with bone growth.

The increased stiffness (hypertonicity) of the muscle can have both neural and mechanical components. Spasticity (neural) and reduced muscle length (mechanical) can both theoretically be addressed with stretching, although the mechanisms by which these changes occur are not fully understood (Guissard and Duchateau, 2006). Regarding the former, the reduction in neural hypertonia may be related to reduced motor neuron excitability or reduced neural input to motor neurons through both pre- (e.g. input from Ia afferents) and/or post-synaptic mechanisms (Hummelsheim et al., 1994). The consequence of this for a spastic muscle may be a decrease in tonic reflex activity or an increase in the threshold of tonic stretch reflex, thus allowing an increase in joint range of motion (RoM) and muscle–tendon unit length with stretch (Calota et al., 2008). Regarding the latter, stretching may affect mechanical hypertonia by causing an inducing effect to increase muscle fascicle length (Coutinho et al., 2004). This plasticity of muscle has been demonstrated in several animal studies, where daily stretching over a period of several weeks was sufficient to increase the number of in-series sarcomeres (Williams, 1990).

Regardless of the mechanism involved, it seems that repeated elongation of the muscle during stretch is the key to inducing changes, both neural or mechanical (Williams, 1990). There is no consensus either in clinical practice or in the literature, with regards to the appropriate time of application, duration or frequency of stretch. However, before determining the appropriate stimuli for long-term adaptations, it should first be established whether spastic muscles are indeed able to receive a stretch during changes in joint RoM.

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Acute stretching has been shown to cause short-term increases in RoM in adults (McNair and Stanley, 1996), but the underlying mechanisms are inconclusive. Whilst some studies suggest that a passive ankle dorsiflexion stretch induces length changes of the gastrocnemii (Blazevich et al., 2012; Morse et al., 2008) other studies suggest that increases are largely accounted for by changes in Achilles tendon length (Herbert et al., 2002; Kubo et al., 2002). Muscle elongation for a given stretch intensity is governed by muscle and tendon stiffness, which are determined by their dimensions as well as material properties. In addition, muscle stiffness is also determined by neural factors (i.e., spasticity). For this reason, acute adaptations to muscle length in response to stretching could be different in children with CP compared to healthy populations. Both Wren et al. (2010) and Barber et al. (2012) showed that tendons are longer in children with CP compared to their typically developing peers. Whilst this dimensional difference is not necessarily associated with differences in tendon stiffness (Barber et al., 2012), children with CP have been shown to have a greater tendon slack length (Barber et al., 2012). During stretching, rotation of the joint may cause tendon slack to be taken up without any elongation of the muscle.

With regards to muscle properties, several dimensional and mechanical differences have been reported between children with CP and typically developing children. These include differences in muscle length (Malaiya et al., 2007), cross sectional area (Elder et al., 2003) and alterations in connective tissue (Smith et al., 2011). In addition, in a spastic muscle, the increased gain or lower threshold of the stretch reflex may cause the muscle to be activated even during low levels of stretch. If the net result of these factors was an increase in muscle stiffness, this could prevent the muscle from elongating in response to stretch. Even if muscle elongation does occur during stretch, it is not clear whether any change in muscle length will be due to the elongation of the muscle fascicles or the surrounding connective tissue. Morse et al. (2008) demonstrated that although acute stretching increased the length of the gastrocnemii, this lengthening was caused by changes in the connective tissue alone and not increases in fascicle length. In a hypertonic muscle this effect may be exaggerated due to a greater abundance of connective tissue in the muscle (Smith et al., 2011). In contrast, Barber et al. (2011) showed that although children with CP have stiffer muscle fascicles than typically developing children, some fascicle elongation did occur. Thus, it is not clear whether or not muscles and fascicles elongate during acute passive stretching in children with CP. If this was not the case, the effectiveness of long term stretching to increase muscle length or reduce muscle stiffness in children with spastic CP would be questionable. Therefore, the first purpose of the study was to examine whether short-term increases in RoM in children with CP as a result of acute stretching would be due to transient changes in medial gastrocnemius muscle and fascicle length, and/or Achilles tendon length.

In addition to the knowledge of the mechanism underlying stretch-induced increases in RoM, stretch technique is another important factor to consider in clinical practice. Several techniques are used by physiotherapists and taught to parents/carers, which may be classified into two broad categories; passive stretches administered manually by a physiotherapist ("PT-stretch"), often with the patients lying supine, or standing stretches performed by the individuals themselves ("self-stretch"). These two techniques are the most commonly used stretches in clinical practice to increase RoM and muscle extensibility, and have been developed based on guidelines related to the aims of physical therapy (Bandy and Sanders, 2001).

If the primary goal of stretch is to elongate the muscle, two potential reasons exist as to why these techniques could have different effects. Due to the voluntary nature of the standing self-stretch, it might be difficult for children with CP to coordinate muscle activity to maintain the body position required for the muscle to be stretched (Rose and McGill, 2005), which could negatively impact the effectiveness of the stretch. Conversely, during the standing self-stretch proprioceptive inputs from the foot sole and/or altered input from the vestibular system, which may serve to suppress the H-reflex (Alrowayeh et al., 2005), could reduce the activation level of the muscle and thereby increase the muscle's extensibility. From a basic science perspective, an understanding of the effect stretch technique has on muscle extensibility is important as it would provide further insights into these conflicting mechanisms. From an applied perspective, such an understanding would help to inform clinicians as to which technique is most effective in terms of achieving the greatest potential stretch of the muscle. Therefore, the second purpose of this study was to investigate the effect of stretch technique on muscle extensibility in children with spastic CP.

2. Methods

2.1. Participants

Eight children with clinically diagnosed spastic diplegic CP (3 males, 5 females; mean age 10.2 y SD 3.2 y (range = 6–14 y) mean stature 138.4 SD 18.4 cm; mean mass 37.3 SD 13.2 kg) were recruited through the British National Health Service (NHS) paediatric physiotherapy services. Five patients were classified as Level 2, and 3 patients were classified as Level 1 on the Gross Motor Function Classification System as assessed by a physiotherapist (Palisano et al., 1997). No children had received any form of orthopaedic surgery or Botulinum toxin injection prior to participation in the study. The study was approved by institutional as well as the relevant local NHS Ethics Committees. The study was conducted in accordance with the Declaration of Helsinki. Written parental consent was obtained in addition to written assent from the children.

2.2. Experimental design

Participants attended the physiotherapy clinic on one occasion. During this time, each participant underwent a series of passive ankle dorsiflexion stretches using both the self-stretch and PT-stretch techniques (described below). The order of stretch techniques was administered in a random order. Both techniques were applied to the right and left legs, the order of which was also randomised. For each participant, the first stretch technique was initially performed on the ipsilateral leg, followed by the same stretch technique applied to the contralateral leg. After a period of rest, the ipsilateral leg was stretched again using the second stretch technique, followed by a stretch of the contralateral leg using the same second stretch technique (Fig. 1). Within this context we ensured that the rest period was a minimum of 60 min between the two different stretch techniques on the same leg. This period has previously been shown to be sufficient to eliminate the acute effects of stretch on muscle and tendon properties in healthy individuals (Magnusson et al., 1996a). The majority of this 60 minute rest period was taken up with testing the contralateral leg. For the remainder of the rest period participants were seated with the muscle in a relaxed position. Passive stretches were performed five times on each leg with each technique. Each stretch was held at maximum RoM for 20 s, followed by a 60 s rest period between stretches. Maximal ankle dorsiflexion was assessed before and after each of the five passive stretches using the PT-stretch technique outlined below.

2.3. Procedure for stretching

2.3.1. PT-stretch

For the PT-stretch, participants lay supine on a foam mat, with the physiotherapist positioned to the side of the participant — opposite to the leg being stretched. To gain the initial stretch position, the leg was lifted with the knee flexed to 90°. To initiate a stretch of the gastrocnemius muscle, the physiotherapist's hand was cupped around

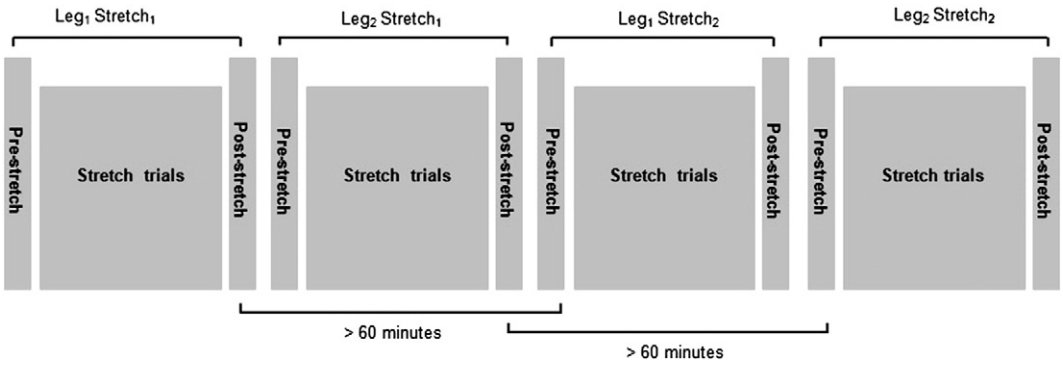

Fig. 1. Experimental design.

the heel, with the palm of the hand flat against the foot. The knee was supported and slowly guided into full extension. This position was stabilised and maintained by pressure at the proximal tibia. Once the knee was locked in an extended position, the ankle was slowly dorsiflexed, with pressure from the hand and forearm on the plantar surface of the forefoot. Joint rotation continued to be applied by the physiotherapist until feedback from the participant indicated the point of discomfort. This point was considered to be maximal dorsiflexion. Once in a maximal stretch position, the joint was held for a period of 20 s. A similar method was used to assess maximum dorsiflexion angle before and after a set of five passive stretches. However, here, the stretch was held only until maximum dorsiflexion had been reached.

2.3.2. Self-stretch

For the self-stretch, children were instructed to stand facing the wall. To gain the initial stretch position, participants were positioned at approximately arms length from the wall with their hands flat on the wall at shoulder height. The leg to be stretched was placed behind the body, and the contralateral leg was flexed and placed in front of the body for support. To maintain an upright posture, participants were instructed to keep their hips facing the wall and to draw the belly button inwards. This pulled the pelvis towards the centre line of the body, maintaining a straight line between the back leg, hips and trunk. For the stretch, participants were asked to ease the back leg away from the wall keeping the knee in an extended position, and pressing the heel into the floor. Once in this stretched position as determined by the participant and physiotherapist, this position was maintained for 20 s. Each participant performed the self-stretch five times on each leg (see Fig. 1). Maximum dorsiflexion was determined before and after the sequence of five stretches as described in the previous section. For all participants, the same physiotherapist was responsible for implementing all passive stretches, and provided all detailed instructions and demonstrations during the self-stretches.

2.4. Data processing

For both techniques, participants were positioned equidistantly between six infrared LED motion capture cameras (Motion Analysis, Santa Rosa, USA). The cameras were positioned on both sides of the participant. Reflective markers were placed bilaterally on the heads of the first and fifth metatarsals, the medial and lateral malleoli, and lateral and medial femoral epicondyles. Further markers were placed on the calcanei as well as the greater trochanters of the right and left femur. Finally, two markers were placed on the handle of the ultrasound probe, perpendicularly to the field of view (see Fig. 2). All kinematic data were filtered using a low-pass, fourth-order, zero-lag Butterworth filter with a cut-off frequency of 5 Hz, as determined by residual analysis (Winter, 1990). The relative ankle angle was defined as the angle between the shank and foot. To account for eventual movements out of the sagittal plane, the angle was calculated using the 3D coordinates. For this purpose, the 3D locations of the midpoints between the 1st and 5th metatarsals, the lateral and medial malleoli as well as the lateral and medial femoral epicondyles were calculated. The relative ankle angle was calculated from these three 3D coordinates using the law of cosines.

During each trial, muscle and tendon elongation were measured by tracking the displacement of the medial gastrocnemius muscle–tendon junction, using B-mode ultrasonography Megas GPX (Esaote, Genova, Italy; 45 mm Linear array probe, 10 MHz transducer scanning). The video transmission was digitally captured at 25 Hz using a video converting frame grabber Canopus ADVC-55 (Grass Valley, Paris, France). A layer of water-based gel (Henleys Medical Supplies Ltd., Hertfordshire, UK) applied between the ultrasound probe and skin enhanced acoustic transmission without depressing the dermal surface.

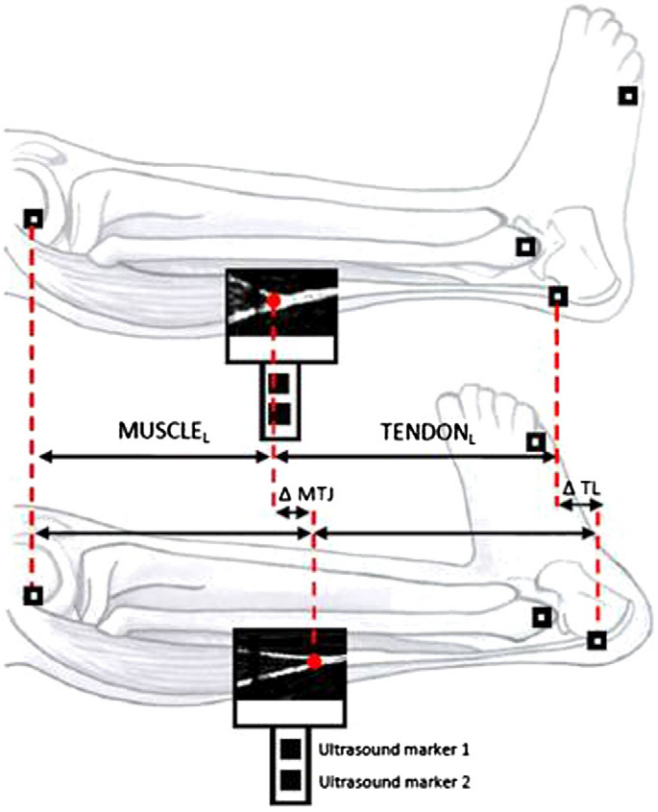

Fig. 2. Experimental setup for measuring muscle and tendon lengths.

The probe was placed perpendicularly to the skin surface above the muscle–tendon junction of the medial gastrocnemius and orientated to reveal a line running between the aponeuroses of the medial gastrocnemius and soleus muscles. The probe was fixed in position using a custom made holder. Peak Motus tracking software (Peak Performance, Cambridge, UK) was used to manually digitise 2D coordinates of the muscle–tendon junction from the ultrasound images. Medial gastrocnemius muscle fascicle length was also quantified in six children, using open source digital measurement software ImageJ (NIH, Bethesda, USA) on the existing ultrasound trials. These measurements were made at approximately the mid belly of the muscle as changes at this site have been shown to be relatively uniform (Lichtwark et al., 2007). Three optimal and identifiable fascicles were selected and measured from deep to superficial aponeuroses. These fascicles were tracked in each frame of the pre- and post-stretch trials, and an average of the three fascicles was used for subsequent analysis. The ultrasound data were synchronised with kinematic data by means of an electrical trigger (Trigger module SP-U03, Delsys Inc., Ltd., Boston, USA). Digitised muscle–tendon junction position data were filtered using a low-pass fourth-order zero-lag Butterworth filter with a 3.25 Hz cut-off frequency. Filtered motion analysis data were down-sampled to 25 Hz to match the sampling frequency of the ultrasound data.

Muscle and tendon lengths were calculated from a combination of motion analysis and ultrasound data. Using the positions of the two markers from the ultrasound probe (Fig. 2), combined with the co-ordinates of the muscle–tendon junction in the ultrasound image, the global 3D position of the muscle–tendon junction was calculated in the inertial reference frame. During pilot testing performed on four healthy adults, the coefficient of variation for muscle–tendon junction location obtained from three separate measures was determined to be 1.35%.

Medial gastrocnemius muscle length was defined as the distance between the midpoint coordinates of the femoral epicondyles and the global 3D coordinates of the muscle–tendon junction. Achilles tendon length was calculated as the distance between the muscle–tendon junction to the calcanei (Fig. 2). Thus, both medial gastrocnemius and Achilles tendon were modelled as straight lines. This analysis was performed using custom written analysis software Matlab v7.14 (MathWorks, Cambridge, UK). To ensure the correctness of this algorithm, we used a sample data set to confirm that the programme's outcome measures were identical to those obtained "manually" by means of spreadsheet calculations.

2.5. Dependent variables

Muscle length, muscle fascicle length and tendon length were expressed firstly at a reference angle (defined as the relative ankle angle of 10º plantarflexion). This angle was calculated from the pre-stretch trials where the ankle was slowly and passively moved from a relaxed plantarflexed position into dorsiflexion. In subsequent analyses, we identified the three data points that were closest to this reference angle for a given trial. For all participants, the ankle angle corresponding to these data points did not deviate by more than one degree from the reference angle. Muscle and tendon lengths were then averaged across these three points. Ankle angle, muscle and fascicle lengths, and tendon length at maximum dorsiflexion were then measured before and after five passive stretches. Maximum dorsiflexion was assessed as described in Section 2.3.1. For each of these variables, we calculated the mean of right and left legs for the corresponding conditions.

2.6. Statistical analysis

To test whether the acute effects of the first stretch technique had diminished before the measurements of the second stretch technique with the same leg, a paired *t*-test on maximum dorsiflexion angle was conducted. Three time (reference angle, pre-stretch, post-stretch) by technique (self-stretch, PT-stretch) repeated measures ANOVAs were performed on ankle dorsiflexion angle, muscle length, and tendon length. A further time (pre-stretch, post-stretch) by technique (self-stretch, PT-stretch) ANOVA was performed on muscle fascicle length. To examine the effects of stretching per-se, we tested for a main effect of time. To test whether stretching effects would be dependent on stretch technique, we tested for a time by technique interaction. In the case of significance, follow up paired *t* tests with Bonferroni correction were performed. Statistical significance was accepted at $P<0.05$.

3. Results

The paired samples *t* test showed no significant difference in maximum dorsiflexion angle ($t_{(7)} = 1.12$, $P = 0.29$) between pre-stretch trials (i.e. before the two different stretch techniques were applied to the same leg). We also found that maximum muscle length increased progressively during the five stretches (Fig. 3), which confirmed the assumption that the muscle was indeed receiving a stretch across the five stretch trials.

The ANOVAs revealed that the main effects for technique and the time by technique interactions were non-significant for ankle angle, muscle and tendon length ($F_{2,14} = 0.34–2.98$, $P > 0.05$ 0.32–0.83) and fascicle length ($F_{2,12} = 0.21–1.69$, $P > 0.05$). The main effects for time were significant for ankle dorsiflexion angle ($F_{2,14} = 150.0$, $P < 0.001$), muscle length ($F_{2,14} = 268.27$, $P < 0.001$), tendon length ($F_{2,14} = 459.61$, $P < 0.001$) and fascicle length ($F_{2,12} = 640.76$, $P < 0.001$). Since there was no interaction effect, data were collapsed across techniques for post-hoc analyses. These tests revealed that all variables were significantly greater during the pre-stretch condition compared to rest ($P < 0.05$). Further, all variables were significantly greater during the post-stretch condition compared to the pre-stretch condition ($P < 0.05$) (Table 1) (see Fig. 5).

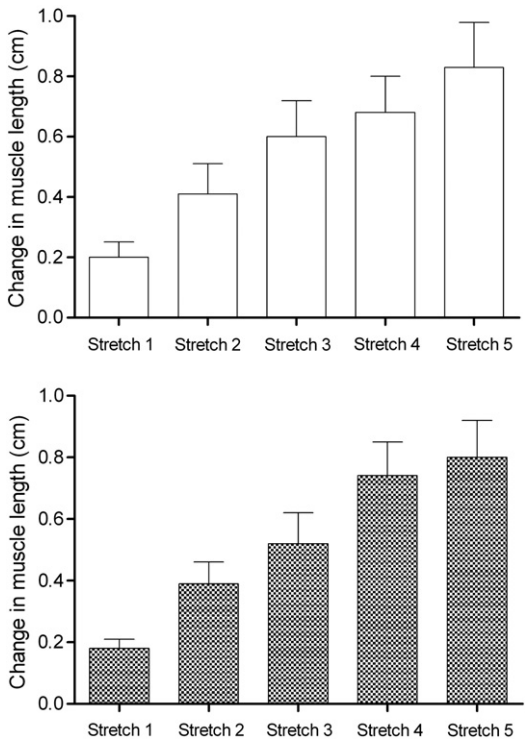

Fig. 3. Change in muscle length across the five stretch trials for the PT-stretch (top figure), and the self-stretch (bottom figure). This change is calculated from muscle length in the pre-stretch trials. Values are expressed as mean (SD).

Table 1
Absolute and relative changes in dorsiflexion angle, muscle length and tendon length. Values expressed as mean (SD). Asterisks represent significance (**$P < 0.001$).

	Absolute Change		Percentage change		$t_{(7)}$
	PT-stretch	Self-stretch	PT-stretch	Self-stretch	
Maximum ankle dorsiflexion (°)					
Rest to pre-stretch	18.0 (2.7)	18.4 (2.3)	21.9%	22.5%	21.02**
Pre- to post-stretch	9.8 (1.7)	9.3 (1.6)	11.9%	11.4%	16.56**
Muscle length (mm)					
Rest to pre-stretch	11.5 (1.6)	11.2 (1.8)	8.2%	8.0%	−15.07**
Pre- to post-stretch	8.3 (2.6)	8.0 (2.9)	5.8%	5.7%	−9.14**
Tendon length (mm)					
Rest to pre-stretch	9.9 (1.8)	9.0 (2.7)	6.2%	5.7%	−12.98**
Pre- to post-stretch	10.2 (1.9)	9.6 (1.6)	6.3%	6.0%	−17.38**
Muscle fascicle length (mm)					
Pre- to post-stretch	5.8 (0.6)	5.3 (1.5)	14.3%	12.9%	−17.33**

To illustrate the physiological significance of the effects of stretching on the dependent variables, we also report the relevant absolute and relative changes (Table 1). Following the stretching protocol, maximal dorsiflexion angle increased by approximately 12% pre- to post-stretch (Fig. 4). This change was accompanied by increases of muscle and tendon length of approximately 6% and a change in muscle fascicle length of 14% pre- to post-stretch. The absolute change in maximal dorsiflexion angle was approximately 10° and accompanied by an 8 mm change in muscle length and 10 mm in tendon length (Fig. 5).

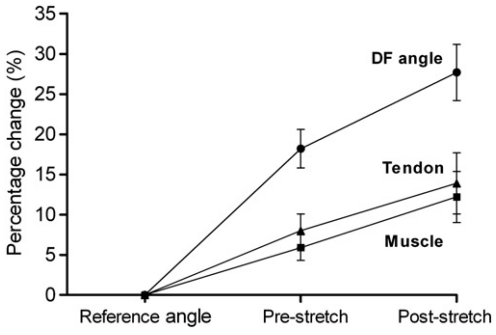

Fig. 4. Percentage change in ankle dorsiflexion angle (DF) (closed circles), muscle length (closed squares) and tendon length (closed triangles) at each testing condition for the collapsed data. Values are expressed as mean (SD).

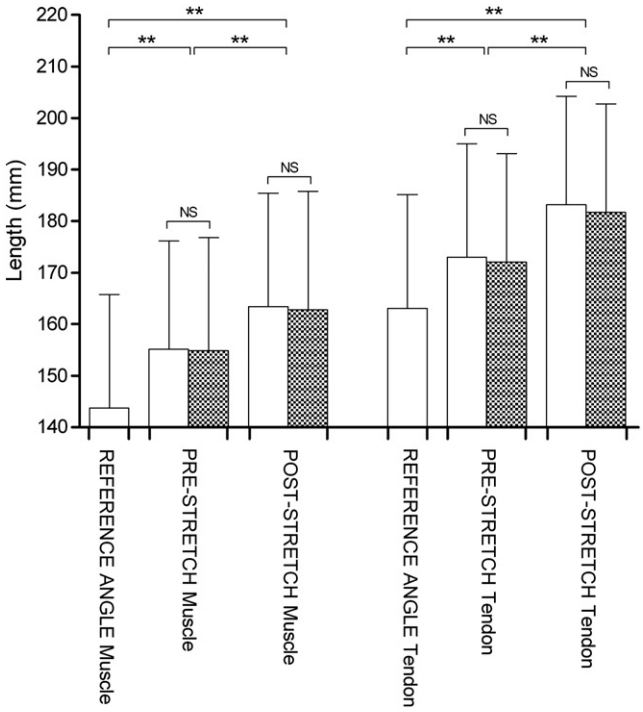

Fig. 5. Comparison of muscle and tendon length at each testing condition for the PT-stretch technique (empty columns) and the self-stretch technique (hashed columns). Values are expressed as mean (SD). Asterisks represent significance (**$P < 0.001$).

4. Discussion

The first purpose of the study was to examine whether stretch-induced increases in ankle RoM in children with CP would be due to alterations in medial gastrocnemius muscle length and/or Achilles tendon length. Following five stretches, ankle dorsiflexion angle increased approximately 10°, which is in agreement with previous findings (Miedaner and Renander, 1988). This increase was accompanied by relatively equal amounts of Achilles tendon elongation (1 cm), and medial gastrocnemius elongation (0.8 cm). Muscle fascicles lengthened by 0.6 cm pre- to post-stretch. This result was not necessarily expected, as children with CP have an abnormally large amount of connective tissue in their muscles (Smith et al., 2011), which could decrease the muscle's extensibility. However, in our participants, this accumulation of connective tissue did seem not to affect the extensibility of muscle fascicles. A possible explanation for this result is a lack of organisation and integrity of the connective tissue in spastic muscles, which may impair the tissue's tensile strength (Lamontage et al., 1997). A consequence could be that this connective tissue is therefore weak and compliant, which would, in turn, allow the muscle fascicle to stretch.

The stretch-induced increases in muscle and fascicle lengths were not necessarily expected for two reasons. First, increased neural activation in the spastic muscle could increase its resistance to stretch (Calota et al., 2008). An increased gain or lower threshold of the tonic stretch reflex could cause the muscle to become active during periods of stretching, preventing elongation of the muscle. Second, in children with CP, rest and slack lengths of the Achilles tendon are longer, whilst gastrocnemius muscle length has been reported to be shorter than in typically developing children (Wren et al., 2010). This was also the case in our participant population (see Fig. 3). Longer tendons have previously been shown to be more compliant (Zhao et al., 2009), and so one might have expected the tendon to take up more of the stretch than the muscle. However, our results show that despite muscle spasticity and tendon length differences, muscles and tendons elongate relatively equally in response to stretching, with smaller length changes from the fascicles.

From a clinical perspective, these results are of immense importance. Previously, inferences about a spastic muscle's extensibility have been made based on the assumption that changes in RoM would be reflective of muscle length changes. This assumption is not intuitive, as increases in RoM can be due to the extensibility of tendons and other passive structures. Our results demonstrate that in a group of children with spastic diplegic CP classified as GMFCS levels 1 or 2, passive stretching at the ankle joint resulted in a significant and acutely sustained elongation of the muscle. They thereby confirm the clinical assumption that acute stretching might increase muscle length in children with CP. Therefore, the present study is a useful and necessary prerequisite to examine the effectiveness of long-term stretching as a clinical intervention to achieve a sustained increase in muscle length in this group of children.

The acute and transient changes in muscle length in response stretching raise the question about the underlying mechanisms. Acute changes in muscle length in response to stretch have been previously reported in healthy adults (Magnusson et al., 1996b) and are thought to result from two main mechanisms. First, the golgi tendon organs dampen the effect on the motor neuronal discharges, thereby causing relaxation of the muscle–tendon unit, which would in turn reset its resting length. Second, Pacinian corpuscles serve as pressure sensors to

regulate pain tolerance. Both mechanisms lead to a change in the muscle's tolerance to stretch. Repeated stretching has also been shown to reduce passive tension and to allow greater elongation through small changes in the viscoelastic properties of the muscle–tendon unit (Ryan et al., 2008). These mechanisms cause only acute changes in maximal muscle and tendon lengths, which dissipates shortly after stretching has stopped. However, they may provide the necessary stimulus for the muscle's adaptive process. Whilst the exact mechanism for sarcomere addition and longitudinal growth in the muscle is unknown, previous research has indicated that muscle stretching is a very powerful stimulant (Williams, 1990). Future research is needed to confirm the speculation that an appropriately designed stretching regime would result in long term longitudinal muscle growth, reductions in spasticity and potential improvements in function in children with CP. Within this context, our results may provide a possible avenue to induce muscle elongation in an ethical and ecologically valid fashion. A limitation to this recommendation is that it is not clear whether the magnitude or duration of this stimulus would be sufficient to bring about long term adaptations in the muscle. A logical question arising from these results is whether such mechanical changes in muscle structure can be achieved with an ecologically valid and clinically applicable long term stretching protocol. Our results provide a first step towards answering this question.

The second purpose of the study was to determine whether any change in muscle length was dependent on stretch technique. The results demonstrate that changes in ankle RoM, maximal muscle and tendon lengths were independent of stretch technique. It was hypothesised that there may be a reduction in neural activation associated with standing (Ali and Sabbahi, 2000), through inhibition of the H-reflex, which could have resulted in a greater maximal stretch. This may be because the self-stretch position required less modulation of the vestibular system as participants were asked to place their hands on the wall in front of them and were not unstable in this position. Therefore the magnitude of inhibition may not have been different between techniques. This finding has direct clinical implications, by providing parents and clinicians reassurance that as the child takes steps towards self management, and transitions from physiotherapist led stretching to self stretching, both techniques are equally effective. It needs to be noted that in our study, the self-stretch technique was performed under the supervision of a physiotherapist who gave verbal feedback where necessary. Thus, our findings do not rule out the possibility that the self-stretch would be less effective if performed without professional supervision.

It is important to recognise the limitations of this method for calculating muscle and tendon lengths. We modelled muscle and tendon paths as two straight lines. This approach does not take the curvature of muscle and tendon into consideration. At maximal ankle dorsiflexion this systematic error may have been minimal since any slack from muscle and tendon would have been taken up in this position. At rest, muscle and tendon lengths may have been underestimated by assuming two straight lines due to muscle, and in particular, tendon slack. Another limitation of our approach is that our estimate of "muscle length" included the length of the proximal tendon of the medial gastrocnemius. However, its role within the context of muscle/tendon dynamics is considered to be negligible (Maganaris and Paul, 2002; Morse, 2011), and therefore, we believe that this assumption does not affect our results.

In summary, we found that acute stretching causes transient increases in both muscle and tendon length in children with CP independent of stretch technique. The results thereby suggest that stretching in children with CP is a suitable treatment to gain short-term increases in muscle length, which may lead to long term adaptations if repeated over a period of weeks or months. These findings constitute a first step towards a more refined understanding of the relationship between stretching and changes in the mechanical structure of muscles and tendons. They thereby have direct implications for clinicians treating children with CP.

Conflict of interest

None of the authors has any commercial or other interests that create a conflict of interest for the work presented here.

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References

Ali, A.A., Sabbahi, M.A., 2000. H-reflex changes under spinal loading and unloading conditions in normal subjects. Clin. Neurophysiol. 111 (4), 664–670.
Alrowayeh, H.N., Sabbahi, M.A., Etnyre, B., 2005. Soleus and vastus medialis H-reflexes: similarities and differences while standing or lying during varied knee flexion angles. J. Neurosci. Methods 144 (2), 215–225.
Bandy, W.D., Sanders, B., 2001. Therapeutic Exercise: Techniques for Intervention. Lippincott, Williams and Wilkins, Baltimore, Maryland 24–50.
Barber, L., Barrett, R., Lichtwark, G., 2011. Passive mechanical properties of the medial gastrocnemius muscle in young adults with cerebral palsy. J. Biomech. 44 (13), 2496–2500.
Barber, L., Barrett, R., Lichtwark, G., 2012. Medial gastrocnemius muscle fascicle active torque-length and Achilles tendon properties in young adults with spastic cerebral palsy. J. Biomech. 45 (15), 2526–2530.
Blazevich, A.J., Cannavan, D., Waugh, C., Fath, F., Miller, S., Kay, A.D., 2012. Neuromuscular factors influencing the maximum stretch limit of the human plantar flexors. J. Appl. Physiol. 113 (9), 1446–1455.
Calota, A., Feldman, A.G., Levin, M.F., 2008. Spasticity measurement based on tonic stretch reflex threshold in stroke using a portable device. Clin. Neurophysiol. 119 (10), 2329–2337.
Coutinho, E.L., Gomes, A.R.S., Franca, C.N., Oishi, J., Salvini, T.F., 2004. Effect of passive stretching on the immobilised soleus muscle fiber morphology. Braz. J. Med. Biol. Res. 37 (12), 1853–1861.
Elder, G.C., Kirk, J., Stewart, G., Cook, K., Weir, D., Marshall, A., et al., 2003. Contributing factors to muscle weakness in children with cerebral palsy. Dev. Med. Child Neurol. 45 (8), 542–550.
Guissard, N., Duchateau, J., 2006. Neural aspects of muscle stretching. Exerc. Sport Sci. Rev. 34 (4), 154–158.
Herbert, R., 2004. Adaptations of muscle and connective tissue. In: Refshauge, K.M., Gass, E.M. (Eds.), Musculoskeletal Physiotherapy: Clinical Science and Evidence Based Practice. Butterworth-Heinemann, Oxford, p. 43.
Herbert, R., Moseley, A.M., Butler, J.E., Gandevia, S.C., 2002. Change in length of relaxed muscle fascicles and tendons with knee and ankle movements in humans. J. Physiol. 539 (2), 637–645.
Hummelsheim, H., Munch, B., Butefisch, C., Neumann, S., 1994. Influence of sustained stretch on late muscular response to magnetic brain stimulation in patients with upper motor neuron lesions. Scand. J. Rehabil. Med. 26 (1), 3–9.
Kubo, K., Kanehisa, H., Fukunaga, T., 2002. Effects of resistance and stretching training programmes on the viscoelastic properties of human tendon structures in vivo. J. Physiol. 538 (1), 219–226.
Lamontage, A., Malouin, F., Richards, C., Dumas, F., 1997. Impaired viscoelastic behaviour of spastic plantarflexors during passive stretch at different velocities. Clin. Biomech. 12 (7/8), 508–515.
Lichtwark, G.A., Bougoulias, K., Wilson, A.M., 2007. Muscle fascicle and series elastic element length changes along the length of the human gastrocnemius during walking and running. J. Biomech. 40 (1), 157–164.
Maganaris, C.N., Paul, J.P., 2002. Tensile properties of the in vivo human gastrocnemius tendon. J. Biomech. 35 (12), 1639–1646.
Magnusson, S.P., Simonsen, E.B., Aagaard, P., Kjaer, M., 1996a. Biomechanical responses to repeated stretches in human hamstring muscle in vivo. Am. J. Sports Med. 24 (5), 622–628.
Magnusson, S.P., Simonsen, E.B., Aagaard, P., Dyhre-Poulsen, P., McHugh, M.P., Kjaer, M., 1996b. Mechanical and physiological responses to stretching with and without preisometric contraction in human skeletal muscle. Arch. Phys. Med. Rehabil. 77 (4), 373–378.
Malaiya, R., McNee, A.E., Fry, N.R., Eve, L.C., Gough, M., Shortland, A.P., 2007. The morphology of the medial gastrocnemius in typically developing children and children with spastic hemiplegic cerebral palsy. J. Electromyogr. Kinesiol. 17 (6), 657–663.
McNair, P.J., Stanley, S.N., 1996. Effect of passive stretching and jogging on the series elastic muscle stiffness and range of motion of the ankle joint. Br. J. Sports Med. 30 (4), 313–317.

Miedaner, J.A., Renander, J., 1988. The effectiveness of classroom passive stretching programs for increasing or maintaining passive range of motion in non-ambulatory children. Phys. Occup. Ther. Pediatr. 7 (3), 35–43.
Morse, C.I., 2011. Gender differences in the passive stiffness of the human gastrocnemius muscle during stretch. Eur. J. Appl. Physiol. 111 (9), 2149–2154.
Morse, C.I., Degens, H., Seynnes, O.R., Maganaris, C.N., Jones, D.A., 2008. The acute effect of stretching on the passive stiffness of the human gastrocnemius muscle tendon unit. J. Physiol. 586 (1), 97–106.
National Institute for Health and Clinical Excellence (NICE), 2012. Spasticity in children and young people with non-progressive brain disorders: management of spasticity and co-existing motor disorders and their early musculoskeletal complications. CG145. (London, UK).
Odeen, I., 1981. Reduction of muscular hypertonus by long-term muscle stretch. Scand. J. Rehabil. Med. 13 (2–3), 93–99.
Palisano, R., Rosenbaum, P., Walter, S., Russell, D., Wood, E., Galuppi, B., 1997. Development and reliability of a system to classify gross motor function in children with cerebral palsy. Dev. Med. Child Neurol. 39 (4), 214–223.
Rose, J., McGill, K.C., 2005. Neuromuscular activation and motor-unit firing characteristics in cerebral palsy. Dev. Med. Child Neurol. 47 (5), 329–336.
Ryan, E.D., et al., 2008. The time course of musculotendinous stiffness responses following different durations of passive stretching. J. Orthop. Sports Phys. Ther. 38 (10), 632–639.
Smith, L.R., Lee, K.S., Ward, S.R., Chambers, H.G., Lieber, R.L., 2011. Hamstring contractures in children with spastic cerebral palsy result from a stiffer extracellular matrix and increased *in vivo* sarcomere length. J. Physiol. 589 (10), 2625–2639.
Williams, P.E., 1990. Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. Ann. Rheum. Dis. 49 (5), 316–317.
Winter, D.A., 1990. Biomechanics and Motor Control of Human Movement, 2nd ed. Wiley, New Jersey 41–45.
Wren, T.A.L., Cheatwood, A.P., Rethlefsen, S.A., Hara, R., Perez, F.J., Kay, R.M., 2010. Achilles tendon length and medial gastrocnemius architecture in children with cerebral palsy and equinus gait. J. Pediatr. Orthop. 30 (5), 479–484.
Zhao, H., Ren, Y., Wu, Y., Liu, S.Q., Zhang, L., 2009. Ultrasonic evaluations of Achilles tendon mechanical properties post-stroke. J. Appl. Phys. 106 (3), 843–849.