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Article

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Topic: **Rapid force development**.

Abstract

Children develop lower levels of muscle force, and at slower rates, than adults. Whilst strength training in children is expected to reduce this differential, a synchronous adaptation in the tendon must be achieved to ensure forces continue to be transmitted to the skeleton with efficiency whilst minimizing the risk of strain-related tendon injury. We hypothesized that resistance training (RT) would alter tendon mechanical properties in children concomitantly with changes in force production characteristics. Twenty prepubertal children (8.9 ± 0.3 years) were equally divided into control (non-training) and experimental (training) groups. The training group completed a 10-week RT intervention consisting of 2-3 sets of 8-15 plantar flexion contractions performed twice-weekly on a recumbent calf raise machine. Achilles tendon properties (cross-sectional area, elongation, stress, strain, stiffness and Young's modulus), electromechanical delay (EMD; time between the onset of muscle activity and force), rate of force development (RFD; slope of the force-time curve) and rate of EMG increase (REI; slope of the EMG-time curve) were measured before and after RT. Tendon stiffness and Young's modulus increased significantly after RT in the experimental group only (~29% and ~25%, respectively); all other tendon properties were not significantly altered, although there were mean decreases in both peak tendon strain and strain at a given force level (14% and 24%, respectively (n.s)) which may have implications for tendon injury risk and muscle fiber mechanics. A ~13% decrease in EMD was found after RT for the experimental group which paralleled the increase in tendon stiffness (r = -0.59), however RFD and REI were unchanged. The present data show that the Achilles tendon adapts to RT in prepubertal children and is paralleled by a change in EMD, although the magnitude of this change did not appear to be sufficient to influence RFD. These findings are of potential importance within the context of the efficiency and execution of movement.

Review

Huge differences in performance have been observed between adults and children during explosive muscle strength tasks, i.e., at high movement speeds and when evaluating rate of force development (RFD) (e.g. 2, 5). This has implications for movement and performance, specifically in dynamic actions such a jumping (e.g., 4) or to stabilise joints and recover a safe stance following a

postural perturbation (6, 10). Achieving a high RFD is clearly dependent on the individual's ability to rapidly increase muscle activation (1) to develop large muscle forces. However, numerous studies have shown that RFD is associated with the stiffness of the tendon, which is in-series between the force-producing muscle and the body segment at which the rate of external force is measured (e.g., 3). Stiffness of the Achilles (11) and patellar (9) tendons is known to increase with maturation, and thus it seems likely that the greater RFD achieved by adults can partially be attributed to improved tendon properties. This appeared to be confirmed in a cross-sectional study that developed regression models predicting RFD across maturation and into adulthood ($R^2 \approx 70\%$) using rate of electromyographic activity rise (to indicate muscle activation rate), age and tendon stiffness (12).

Subsequently, the same group undertook a progressive resistance training study in children (n=20, 8.9 ± 0.3 years old), to determine whether resistance training can alter the mechanical properties of the Achilles tendon (13). Indeed, tendon stiffness increased post-training (~29%), due primarily to increased material stiffness rather than tendon cross-section. The increase in stiffness is approximately half that observed in adults, but the mechanism by which it is increased is consistent with that in adults (e.g. 8). Interestingly, however, RFD did not improve significantly post-intervention (group x time interaction: F= 1.944-0.062, p = 0.185–0.807); two possible explanations exist for this finding. First, contractions during training were slow but the RFD requires fast contractions, so there may be a lack of training specificity. Such an effect was apparent in the strength gains; training load more than doubled during slow concentric contractions, but strength during rapid isometric contractions did not improve compared to the control group. Similarly, the rate of electromyographic activity rise did not improve post-training, and this is the primary determinant of RFD. Second (and the primary reason this paper has been highlighted in this review of the year), the magnitude of change in tendon stiffness may not have been sufficient to elicit meaningful changes in RFD. Although it is possible this study was under-powered, according to those authors' previous regression models (12), the magnitude of change in tendon stiffness following 10-weeks of PRT was only adequate to elicit small changes in RFD, which are of comparable magnitude to those actually observed. The lack of change in muscle activation rate means such an attempt to isolate the impact of tendon stiffness on RFD is less likely to be confounded by their overlapping effects.

The unexpected conclusion of Waugh et al. (13) that the magnitude of change in children's tendon stiffness following PRT is not adequate to improve RFD has subsequently been supported by a recent cross-sectional study in adults reporting that RFD was not independently associated with tendon stiffness, when maximum voluntary force was controlled for (7). Although muscle force is highly correlated with tendon stiffness, meaning co-linearity may have confounded their approach, the agreement between these separate studies is hard to dismiss. Consequently, further work is required to fully understand the direct effect of tendon properties on rapid force development and performance of dynamic exercises. Direct interventions in which muscle strength or tendon stiffness are altered independently, whilst also not affecting activation rate, are likely difficult, but should be sought to confirm these observations. Pharmaceutical options may be possible, where justified or convenient. Alternatively, an elaborate matching study of individuals with similar muscle strengths

but different tendon stiffness, or vice versa, may be possible with significant recruitment efforts. Those with an interest in muscle and tendon mechanics face a challenge to untangle the independent effects of strength and tendon stiffness on RFD. This likely has particular importance for paediatric exercise since the changes in tendon stiffness throughout maturation may be up to an order of magnitude larger than those achieved by training adults.

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Topic: **Measuring and understanding spasticity in children with cerebral palsy**.

Key <u>papers</u> of interest:

Bar-On L, Aertbeliën E, Molenaers G, Desloovere K (2014). **Muscle activation patterns when passively stretching spastic lower limb muscles of children with cerebral palsy**. PLoS One. 20;9(3):e91759.

<u>Abstract</u>

The definition of spasticity as a velocity-dependent activation of the tonic stretch reflex during a stretch to a passive muscle is the most widely accepted. However, other mechanisms are also thought to contribute to pathological muscle activity and, in patients post-stroke and spinal cord injury can result in different activation patterns. In the lower-limbs of children with spastic cerebral palsy (CP) these distinct activation patterns have not yet been thoroughly explored. The aim of the study was to apply an instrumented assessment to quantify different muscle activation patterns in four lower-limb muscles of children with CP. Fifty-four children with CP were included (males/females n = 35/19; 10.8±3.8 yrs; bilateral/unilateral involvement n = 32/22; Gross Motor Functional Classification Score I–IV) of whom ten were retested to evaluate intra-rater reliability. With the subject relaxed, single-joint, sagittal-plane movements of the hip, knee, and ankle were performed to stretch the lower-limb muscles at three increasing velocities. Muscle activity and joint motion were synchronously recorded using inertial sensors and electromyography (EMG) from the adductors, medial hamstrings, rectus femoris, and gastrocnemius. Muscles were visually categorised into activation patterns using average, normalized root mean square EMG (RMS-EMG) compared across increasing position zones and velocities. Based on the visual categorisation, quantitative parameters were defined using stretch-reflex thresholds and normalized RMS-EMG. These parameters were compared between muscles with different activation patterns. All patterns were dominated by high velocity-dependent muscle activation, but in more than half, low velocity-dependent activation was also observed. Muscle activation patterns were found to be both muscle- and subject-specific (p<0.01). The intra-rater reliability of all quantitative parameters was moderate to good. Comparing RMS-EMG between incremental position zones during low velocity stretches was found to be the most sensitive in categorizing muscles into activation patterns (p<0.01). Future studies should investigate whether muscles with different patterns react differently to treatment.

Bar-On L, Desloovere K, Molenaers G, Harlaar J, Kindt T, Aertbelien E. **Identification of the neural component of torque during manually applied spasticity assessments in children with cerebral palsy**. Gait Posture. 2014;40(3):346-51.

<u>Abstract</u>

Clinical assessment of spasticity is compromised by the difficulty to distinguish neural from non-neural components of increased joint torque. Quantifying the contributions of each of these

components is crucial to optimize the selection of anti-spasticity treatments such as Botulinum Toxin (BTX). The aim of this study was to compare different biomechanical parameters that quantify the neural contribution to ankle joint torque measured during manually-applied passive stretches to the gastrocsoleus in children with spastic cerebral palsy (CP). The gastrocsoleus of 53 children with CP (10.9 ± 3.7yrs; females n=14; bilateral/unilateral involvement n=28/25; Gross Motor Functional Classification Score I-IV) and 10 age-matched typically developing (TD) children were assessed using a manually-applied, instrumented spasticity assessment. Joint angle characteristics, root mean square electromyography and joint torque were simultaneously recorded during passive stretches at increasing velocities. From the CP cohort, 10 muscles were re-assessed for intra-rater reliability and 19 muscles were re-assessed 6 weeks post-BTX. A parameter related to mechanical work, containing both neural and non-neural components, was compared to newly developed parameters that were based on the modeling of passive stiffness and viscosity. The difference between modeled and measured response provided a quantification of the neural component. Both types of parameters were reliable (ICC>0.95) and distinguished TD from spastic muscles (p<0.001). However, only the newly developed parameters significantly decreased post-BTX (p=0.012). Identifying the neural and non-neural contributions to increased joint torque allows for the development of individually tailored tone management.

Bar-On L, Molenaers G, Aertbeliën E, Monari D, Feys H, Desloovere K (2014). **The relation between spasticity and muscle behavior during the swing phase of gait in children with cerebral palsy**. Res Dev Disabil. 35(12):3354-64.

Abstract

There is much debate about how spasticity contributes to the movement abnormalities seen in children with spastic cerebral palsy (CP). This study explored the relation between stretch reflex characteristics in passive muscles and markers of spasticity during gait. Twenty-four children with CP underwent 3D gait analysis at three walking velocity conditions (self- selected, faster and fastest). The gastrocnemius (GAS) and medial hamstrings (MEHs) were assessed at rest using an instrumented spasticity assessment that determined the stretch- reflex threshold, expressed in terms of muscle lengthening velocity. Muscle activation was quantified with root mean square electromyography (RMS-EMG) during passive muscle stretch and during the muscle lengthening periods in the swing phase of gait. Parameters from passive stretch were compared to those from gait analysis. In about half the children, GAS peak muscle lengthening velocity during the swing phase of gait did not exceed its stretch reflex threshold. In contrast, in the MEHs the threshold was always exceeded. In the GAS, stretch reflex thresholds were positively correlated to peak muscle lengthening velocity during the swing phase of gait at the faster (r = 0.46) and fastest (r = 0.54) walking conditions. In the MEHs, a similar relation was found, but only at the faster walking condition (r = 0.43). RMS-EMG during passive stretch showed moderate correlations to RMS-EMG during the swing phase of gait in the GAS (r = 0.46–0.56) and good correlations in the MEHs (r = 0.69–0.77) at all walking conditions. RMS-EMG during passive stretch showed no correlations to peak muscle lengthening velocity during gait. We conclude that a reduced stretch reflex threshold in the GAS and MEHs constrains peak muscle lengthening velocity during gait in children with CP. With

increasing walking velocity, this constraint is more marked in the GAS, but not in the MEHs. Hyper-activation of stretch reflexes during passive stretch is related to muscle activation during the swing phase of gait, but has a limited contribution to reduced muscle lengthening velocity during swing. Larger studies are required to confirm these results, and to investigate the contribution of other impairments such as passive stiffness and weakness to reduced muscle lengthening velocity during the swing phase of gait.

Review

Cerebral Palsy (CP) is the most common cause of physical disability in children. It is characterised by neurological impairment and reflex hyperactivity, which is most apparent during high velocity muscle lengthening, and presents as spasticity. Common sequelae to spasticity include muscle contracture, which further increases muscle-tendon-joint stiffness, and bony deformations, both exacerbate the impaired movement function. Consequently, much effort has gone into defining characteristics of spasticity and evaluating its effects on movement.

In clinical settings spasticity is assessed using the Modified Ashworth Scale (MAS) (5) or Modified Tardieu Scale (MTS) (6). However, these techniques rely on the subjective assessment of the performing clinician and as a result can have poor reliability (8, 9). Several groups have attempted to develop more objective assessments of spasticity, but for various reasons these have not been widely adopted (for a summary of these see the introduction of 1). Recently, Bar-On et al. (1) developed an instrumented tool for objectively assessing spasticity in the commonly affected lower limb muscles of children with CP. Their integrated biomechanical and neurophysiological approach combines joint position and resistance to stretch with electromyographyic activity in an assessment reported to have moderately high repeatability. Moreover, the equipment required is small and lightweight, and the protocol requires the physician to perform tasks similar to those already used in the MAS and MTS (i.e., passive joint stretches manually applied by the assessor). Consequently, it appears to offer great potential for wide clinical applicability, and to possibly improve assessment of spasticity and aid clinical decision making.

This integrated spasticity assessment tool also presents important research opportunities as it provides detailed biomechanical and neurophysiological data. This has allowed the group to publish several studies throughout 2014 exploring the nature of spasticity in children with CP and the effects it has on joint and whole-body movement function. Here, I highlight three of these papers and summarise what I believe has been particularly interesting and informative from each (2, 3, 4).

The first (2), explored the range of activation patterns in response to slow and fast passive stretches of the hip adductors, medial hamstrings, rectus femoris, and gastrocnemius. The classic velocity-dependent stretch-reflex occurred in all cases, but an activation response was also observed during slow stretches in each of the assessed muscles; most commonly in adductors and medial hamstrings.

Thus, in children with CP the contribution of muscle hyper-activity (spasticity) to increased joint stiffness appears dependent not only on velocity but also joint position during stretch. These effects were muscle- and individual-specific. Whilst these observations likely raise more questions for the treatment of spasticity and joint stiffness than are answered, they further show the very complex nature of "spastic" muscle function and challenge the routine use of standard treatment solutions for every child and all muscles.

The second paper (3) developed a model of joint resistance to stretch based on passive stiffness and viscoelastic properties of the muscle-tendon-joint structures. The model fitted measured data from typically developing children well. In children with CP, deviation from the predicted model was large and this was attributed to the neural component of joint resistance. Moreover, as stretch velocity became faster the joint resistance to stretch increased more in children with CP than typically developing children. Following treatment with combined Botulinum Toxin-A and casting, the neural component was significantly reduced but there remained a large increase in resistance to stretch as stretch velocity increased. It was concluded that the common treatment modality of casting following Botulinum Toxin-A injection did not improve the passive and viscoelastic contribution to resistance to stretch in the spastic gastrocnemius muscles, and more work is required to find optimal interventions.

Finally, the third highlighted paper (4), explored how spasticity and reduced reflex threshold relate to the characteristics of gait. Previous work, quantifying spasticity using the Ashworth and Tardieu scores, failed to find any such associations (7). However, Bar-On et al. found that during the swing phase at self-selected walking speed the predicted medial hamstrings velocity exceeded the reflex-threshold velocity quantified using instrumented assessments. In the gastrocnemius, muscle velocity was lower than reflex-threshold velocity at self-selected walking speed but exceeded threshold-velocity at higher walking speeds. This indicates that low reflex thresholds in both muscles limited muscle velocity, and thus walking speed. In the hamstrings this was the case even at self-selected speeds, but in the gastrocnemius the effect was greater at faster speeds.

This programme of work from Bar-On and colleagues presents an evolution in our understanding of how CP affects muscle-joint function and provides a new insight into the effects on movement. Further work is clearly required before their approach to spasticity assessment can become routinely used in clinical practice and before the implications for physical function are fully understood, but the potential on both fronts is much greater with simple, objective methods to quantify and explore the nature of spasticity and joint stiffness. Specifically, we need to understand how the underlying muscle function is associated with the observed length- and/or velocity-dependent spasticity; whether specific characteristics of length- and velocity-dependent spasticity can be used to determine individualised management to optimise clinical outcome and patient satisfaction; why the hamstrings, especially, appear to have such varied activation characteristics and which treatments can best target their relatively low reflex thresholds during walking; and how the passive stiffness of

the gastrocnemius, which does not appear to be improved by casting following Botulinum Toxin-A, can be targeted and whether this allows improved walking performance.

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