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Prolonged stretching of the ankle plantarflexors elicits muscle-tendon adaptations relevant to ankle gait kinetics in children with spastic cerebral palsy

Teresa Martín Lorenzo[a,b,*], Eduardo Rocon[c], Ignacio Martínez Caballero[a], Ana Ramírez Barragán[a], Sergio Lerma Lara[d]

[a] Laboratorio de Análisis del Movimiento, Hospital Infantil Universitario Niño Jesús, Madrid, Spain
[b] Facultad de Ciencias de la Salud, Universidad Rey Juan Carlos, Madrid, Spain
[c] Centro de Automática y Robótica, Consejo Superior de Investigaciones Científicas, Arganda del Rey, Madrid, Spain
[d] Facultad de Ciencias de la Salud, Centro Superior de Estudios Universitarios La Salle - Universidad Autónoma de Madrid, Madrid, Spain

ABSTRACT

Tissue related ankle hyper-resistance has been reported to contribute to equinus gait in children with spastic cerebral palsy. Hence, ankle plantarflexor stretching programs have been developed in order to restore passive ankle dorsiflexion. Despite high quality evidence on the limited effects of stretching on passive joint mobility, further muscle-tendon adaptations have been reported which may impact gait performance. As such, children with spastic cerebral palsy subject to long-term manual static stretching achieved dorsiflexion gains through the reduction of muscle and fascicle strain whilst preserving tendon strain, and prolonged use of ankle-foot orthoses achieved similar dorsiflexion gains through increased tendon strain whilst preserving muscle and fascicle strain. The latter concurred with normalization of early stance plantarflexor moment yet reductions in push-off plantarflexor moment given the increase in tendon compliance. Therefore, similar limited gains in passive ankle joint mobility in response to stretching may be achieved either by preserving/restoring optimal muscle-tendon function, or at the expense of muscle-tendon function and thus contributing gait impairments. The largest increase in ankle passive joint mobility in children with SCP has been obtained through prolonged plantarflexor stretching through ankle casting combined with botulinum neurotoxin type A. However, to our knowledge, there are no published studies on muscle-tendinous adaptations to ankle casting combined with botulinum toxin type A and its effect on ankle joint gait kinetics. Therefore, we hypothesized that ankle casting elicits muscle-tendon adaptations which concur with altered ankle joint kinetics during the stance phase of gait in children with SCP. More information is needed about the relationships between muscle structure and function, and the effect of specific interventions designed to alter muscle properties and associated functional outcomes in children with spastic cerebral palsy.

Introduction

Cerebral palsy (CP) has been defined as “a group of permanent disorders affecting the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain” [1]. The most common type of CP is spastic (SCP) and results in impaired sensory-motor control, muscle weakness, and muscle hyper-resistance [2]. Typical gait abnormalities observed in SCP are the consequence of these interrelated neuromuscular deficits [2].

Muscle hyper-resistance has been recently defined as the pathophysiological neuromuscular response to passive muscle stretch of neural and non-neural origin [3]. The latter refers to tissue related contributors to muscle hyper-resistance and comprise muscle shortening, elasticity, and viscosity [3]. Accordingly, previous studies have observed that children with SCP have reduced medial gastrocnemius (MG) belly length [4] and elasticity compared to typically developing children (TD) [5,6], which contribute to increased ankle stiffness and limited ankle maximum dorsiflexion (MDF) [6,7] and may result in gait equinus [2].

In order to prevent or treat gait equinus in children with SCP, stretching programs have been developed to gain muscle length and

* Corresponding author at: Laboratorio de Análisis del Movimiento, Hospital Infantil Universitario Niño Jesús, Avenida Menéndez Pelayo 65, 28009 Madrid, Spain.
E-mail address: martin.teresa.83@gmail.com (T. Martín Lorenzo).

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elasticity, and therefore improve ankle joint mobility [8]. However, while the MG muscle-tendon has been reported to respond and adapt to stretching [9–11], a recent systematic review reported high-quality evidence indicating that stretching does not have clinically relevant effects on joint mobility [8]. Regardless of this, reported muscle-tendon adaptations [9,10] would ultimately determine muscle function and how forces are generated for body support and propulsion during gait [12–14]. To our knowledge, only one study has reported concurrent muscle-tendon adaptations to stretching and effects on gait kinetics in SCP [10].

Hypothesis

There is a lack of scientific literature regarding muscle-tendon adaptations to stretching in children with SCP [9–11], and how these adaptations affect force generation for body support and propulsion during gait [10]. In summary, one study observed that MG muscle and fascicles, and the Achilles tendon, lengthened in response to one session of passive manual stretching in children with SCP [11]. Furthermore, another study observed that long-term exposure to the stretching stimulus through a systematic manual static stretching program reduced MG stiffness and increased muscle and fascicle strain, whilst preserving tendon structure and mechanics in children with SCP [9]. Conversely, another study observed that a continuous and prolonged exposure to the stretching stimulus through ankle-foot orthoses (AFO) resulted in shorter MG fascicles and more compliant Achilles tendons in children with SCP which concurred with normalization of early stance plantarflexion moment, loss of push-off plantarflexion moment, and conservation of peak power generation at push-off [10]. Therefore, despite limited effects of stretching on joint mobility, muscle-tendinous adaptations have been reported which may concur with beneficial or detrimental effects on gait kinetics that clinicians should be aware of.

Continuous stretching through casting has been frequently used in the management of equinus gait in children with SCP, and has often been combined with the previously mentioned manual static stretching and AFO stretching [15,16]. Further combined with botulinum neurotoxin type A (BoNT-A), casting has elicited the greatest passive ankle joint mobility improvements in children with SCP [15]. Nevertheless, given its similarities to stretching through the use of AFOs [10], and reports on detrimental structural changes in response to BoNT-A in children with SCP [17], casting may further elicit muscle-tendinous adaptations that gait kinetics. Unfortunately, to our knowledge, there are no published studies on MG muscle-tendon adaptations to ankle casting and its effect on ankle joint gait kinetics in children with SCP.

Therefore, we hypothesize that ankle casting elicits muscle-tendon adaptations which concur with altered ankle joint kinetics during the stance phase of gait in children with SCP.

Evaluation of the hypothesis

Tissue related hyper-resistance in children with SCP has been associated to muscle shortening and reduced elasticity [3], properties which have been reported to further compromise muscle strength in children with CP [18]. Accordingly, muscle shortening and reduced elasticity have been reported to contribute to gait abnormalities in children with SCP [2] including altered ankle joint gait kinetics [19]. However, underlying mechanisms are still not well understood [2].

Regarding muscle shortening, the scientific literature has consistently reported that children with SCP have shorter MG muscle bellies than TD children [4]. However, reports on fascicle length differences have been inconclusive [4]. Conversely, children with SCP have shown overstretched sarcomeres comprising MG fascicles of similar length than those of TD children [20]. This has been attributed to impaired muscle adaptation to longitudinal growth [21]. With growth, the MG muscle belly and fascicles of TD children have been observed to lengthen proportionally to longitudinal growth of the tibia [22]. Moreover, in response to this chronic stretch, fascicles have been reported to adapt through in-series sarcomere addition in order to regain initial sarcomere length and maintain force production capacity [23]. This does not appear to occur in children with SCP which accordingly present impaired force production capacity [18]. Alongside, the Achilles tendon of children with SCP has been reported to be longer than that of TD children [5,24] and may further affect force production capacity [25].

Regarding muscle elasticity, children with SCP have shown greater MG belly stiffness and fascicle stiffness than TD children [5,6]. The latter increase in fascicle stiffness has been reported to be accompanied by reduced passive MG fascicle strain with respect to TD subjects [6]. Hence, authors suggested that the increased resistance to stretch during passive ankle dorsiflexion in individuals with SCP could have partially been attributed to the inability of muscle fascicles to elongate with added force, possibly due to fewer over-stretched sarcomeres [6]. Furthermore, alongside increased MG belly and fascicle stiffness of children with SCP, tendon elasticity has been reported to be similar than that of TD children, thus, muscle to tendon stiffness in children with SCP has been reported to be significantly greater than in TD children [5]. These muscle alterations may have functional implications affecting gait performance in children with SCP [5,12,26].

Stretching regimens have been traditionally used in order to obtain muscle length and elasticity gains [8,15]. Despite high-quality evidence on the limited effects of stretching on joint mobility [8], MG muscle-tendinous adaptations have been reported to occur in children with SCP [9,10] and appear relevant to gait function [10].

Manual static stretching of the ankle plantarflexors in children with SCP has been shown to elicit acute lengthening of the MG belly, fascicles, and Achilles tendon (+5.8%, +14.3%, +6.3% respectively) in one session, resulting in moderate gains of MDF (+10°) [11]. Moreover, long-term manual static stretching has been reported to reduce ankle joint stiffness in children with SCP which, given the preservation of Achilles tendon mechanical properties, could have been attributed to reductions in muscle stiffness and increased muscle and fascicle strain [9]. Therefore, despite the poor effects on ankle MDF (+3°), long-term manual static stretching provided the normalization of MG mechanical properties in children with SCP [9]. Conversely, continuous and prolonged exposure to stretching through AFOs (16 ± 4 weeks) has been reported to elicit inverted adaptations as those reported for long-term manual static stretching [9,10]. Thus, while long-term manual static stretching reduced muscle and fascicle strain whilst preserving tendon strain [9], prolonged use of AFOs increased tendon strain whilst preserving muscle and fascicle strain [10]. The latter concurred with normalization of early stance plantarflexor moment yet reductions in push-off plantarflexor moment given the increase in tendon compliance [10]. Moreover, preservation of plantarflexor power generation at push-off could be attributed to the lack of muscle and fascicle level adaptations, which appear to provide for energy absorption and generation in children with SCP [12].

In line with programs providing continuous exposure to stretching, ankle casting has been frequently used in the management of equinus gait in children with SCP [15]. Combined with BoNT-A, casting has elicited the greatest ankle joint mobility improvements in children with SCP [15]. However, as observed in response to the use of AFOs, two unpublished studies have suggested that passive joint mobility gains after ankle casting in children with SCP had been achieved through structural adaptations of the Achilles tendon [15]. Moreover, BoNT-A has been reported to further elicit structural adaptations of the muscle such as fascicle lengthening, atrophy, and changes in elastic properties [17]. Therefore, despite large passive joint mobility gains, underlying muscle-tendinous adaptations should be taken into account as they may determine how forces are generated for body support and propulsion during gait [12,13]. Unfortunately, to our knowledge, there are no published studies on the effects of casting on MG muscle-tendon structure and mechanics and ankle joint gait kinetics.