Muscle length adaptations to high-velocity training in young adults with Cerebral Palsy

Gallinger, Tessa Leigh

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Muscle length adaptations to high-velocity training in young adults with Cerebral Palsy

by

Tessa Leigh Gallinger

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Abstract

**Purpose:** To investigate high-velocity training as an effective strategy to increase muscle fascicle length and improve peak power outputs in young adults with Cerebral Palsy (CP). Muscle moment arm lengths were compared to typically developing peers.

**Methods:** Triceps surae muscle force-power-velocity and force-length relationships were quantified using ultrasound and dynamometry before and following a 10-week high-velocity training program involving sprints, agility and plyometrics. Triceps surae moment arm was assessed with the visual method and tendon travel method.

**Results:** After training, muscle fascicle lengths increased by ~9%, but no significant changes in power output and peak velocity were seen. Muscle force and moment arm were significantly smaller in CP compared to typically developing.

**Conclusion:** High-velocity training can increase muscle fascicle length in CP, but additional training may be needed to enhance power output. A shorter moment arm and decreased muscle force contribute to the reduced plantar flexion function observed in CP.
Preface

Reduced muscle fascicle lengths and/or serial sarcomere number have been consistently observed in research on Cerebral Palsy. This thesis provides an investigation into a method for increasing muscle fascicle length and peak power output using sport-specific exercises at high angular velocities. Manuscripts based on Chapter 3 and 4 will be submitted for publication, therefore there may be repeated information in these chapters.
Acknowledgements

I am incredibly grateful to the support and expertise I received over the last 4 years which has contributed to the formation of this thesis; thank you to Brian and Jared for being absolutely marvelous, understanding, and always available (even when you should be on vacation). I would also like to give many thanks to the MacIntosh lab group and my HPL peers and friends for the critical feedback provided, and support and encouragement around the lab making this the best Masters experience I could have. Lastly, many thanks also to the Sport Science Association of Alberta for the funding support for this research.
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<tr>
<td>CP</td>
<td>Cerebral palsy</td>
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<td>TD</td>
<td>Typically developing</td>
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<td>GMFCS</td>
<td>Gross Motor Function Classification Scale</td>
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<td>MG</td>
<td>Medial gastrocnemius</td>
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<td>LG</td>
<td>Lateral gastrocnemius</td>
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<td>TA</td>
<td>Tibialis Anterior</td>
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<td>MTU</td>
<td>Muscle-tendon unit</td>
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<td>EMG</td>
<td>Electromyography</td>
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<td>RMS</td>
<td>Root mean square</td>
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<td>ROM</td>
<td>Range of motion</td>
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<tr>
<td>CSA</td>
<td>Cross-sectional area</td>
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<tr>
<td>PCSA</td>
<td>Physiological cross-sectional area</td>
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<td>HVT</td>
<td>High velocity training</td>
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<td>MA</td>
<td>Moment arm</td>
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<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
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<tr>
<td>MVC</td>
<td>Maximal voluntary contraction</td>
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<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>dL</td>
<td>Tendon displacement</td>
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<tr>
<td>( \theta, d\theta )</td>
<td>Angle, change in ankle angle</td>
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<tr>
<td>( \text{rad, rad}\cdot\text{s}^{-1} )</td>
<td>Radians, radians per second</td>
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<td>( \text{deg, deg}\cdot\text{s}^{-1} )</td>
<td>Degrees, degrees per second</td>
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<td>Velocity</td>
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<td>Vopt</td>
<td>Optimal Velocity</td>
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<td>m</td>
<td>Slope</td>
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<tr>
<td>ANOVA</td>
<td>Analysis of variance</td>
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<td>ANCOVA</td>
<td>Analysis of covariance</td>
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Chapter One: Introduction

1.1 Overview

This thesis presents the results of the muscle architectural assessments of 8 young adults with Cerebral Palsy (CP) following a 10-week high velocity training (HVT) intervention. The cross-over repeated measures design of this study allowed comparisons of the changes occurring to a muscle’s force-power-velocity and force-length relationship with and without training. The training was intended to stimulate muscle growth and improve muscle power output, specifically, through an increase in number of sarcomeres in series within the medial gastrocnemius (MG) muscle. As longer muscle fascicle lengths (more sarcomeres in series) are correlated with faster sprint times, and the number of sarcomeres in series and parallel are linearly related to muscle power, we expect the HVT to increase fascicle length with a corresponding improvement in the peak power output.

There are two manuscripts prepared for publication within this thesis. The co-authors for these papers have given permission to include these manuscripts in the thesis.

1. Mechanisms of reduced plantarflexor function in Cerebral Palsy; smaller triceps surae moment arm and reduced muscle force.

2. Muscle length adaptations to sport specific velocity training in young adults with cerebral palsy.

1.2 Background

1.2.1 Cerebral Palsy defined; altered muscle activity

CP is a group of permanent movement disorders resulting from an upper motor neuron lesion in the brain occurring before, during or after birth (Rosenbaum et al., 2006). Although the
brain damage is not progressive, secondary adaptations to muscle structure, function and composition occur causing abnormal muscle activity and coordination. This movement disorder varies amongst individuals but can be assessed by the Gross Motor Function Classification System (GMFCS, Appendix A), where the increasing levels I-V are used to class individuals based on a decrease in functional ability and quality of movement (Palisano et al., 2007). Of the most noticeable of changes to impact ability, is a reduced muscle volume when individuals with CP are compared to their typically developing (TD) peers (Barrett and Lichtwark, 2010). Less noticeable but highly pronounced, is the stiffer muscle-tendon unit (MTU) in paretic limbs (Geertsen et al., 2015; Kalkman et al., 2018), usually defined as an “increased resistance to stretch”. When considering the MTU stiffness in CP, an uncertainty exists in the clinical differentiation of reflex-mediated (active) from passive (inactive) contributions to muscle stiffness (Sinkjaer and Magnussen, 1994). It is essential that passive and active contributions to increased joint stiffness are distinguished to improve therapeutic targets and reduce the risk of a child with CP receiving an unnecessary anti-spastic treatment.

Commonly, in clinical settings and research literature, the term spasticity is used as an umbrella term to encompass both the active and passive properties of muscle stiffness during a stretch (Lorentzen et al., 2010). This is in part due to lack of appropriate or validated assessments of spasticity available to clinicians (Kohan 2010), where routine clinical examinations include, simply, a clinician mobilizing and passively rotating the ankle joint to lengthen the MTU until end range (Barber et al., 2011). With the addition of electromyography (EMG) of the muscle, it would be possible to determine whether a true reflex activation and/or co-activation of antagonist and agonist muscles is present. Sinkjaer and Magnussen (1994) used this methodology to show that although total stiffness in limbs affected by CP is higher, it was
the passive stiffness that accounted for almost all of the joint stiffness measured. Lack of evidence for best practice in assessing spasticity is likely due to no adequate definition of spasticity. A majority of the literature defines spasticity as an increase in muscle tone (Lance, 1980), where muscle tone is also poorly defined. Muscle tone seems to refer to increased involuntary tension or resistance to an externally imposed moment. For the remainder of this manuscript, we will use our adapted version of the definition proposed by the SPASM consortium, or A European Thematic Network to Develop Standardized Measures of Spasticity. The term spasticity will refer to an active property of muscle and can include signs of increased active muscle tone, increased excitability of the stretch reflex, and all intermittent or sustained involuntary hyperactivity of skeletal muscle associated with an upper motor neuron lesion (Lance, 1980; Pandyan et al., 2005).

To date, limited data support the assumption that reduced muscle belly length, also referred to as contractures, may be caused by spasticity. Recent research attributes a majority of the differences seen in CP muscle to the changes in the passive muscle properties (Willerslev-Olsen et al., 2013). Contractures are considered the main cause of functional disability in children and adults with CP, but treatments like physical therapy, surgery and neurotoxin injections have not prevented contracture formation (Tilton, 2006). It is thought that contractures are associated with reduced muscle belly length and longer tendon lengths due to muscle tissue growth, addition of sarcomeres, failing to keep up with bone growth, and/or a thickening of the tertiary perimysium, a connective tissue that encircles bundles of muscle fibres (De Bruin et al., 2014; Hösl et al., 2015; Willerslev-Olsen et al., 2013). For the remainder of the manuscript, the term contracture will be used to refer to an inactive property of muscle, including
signs of reduced muscle length, increased passive stiffness, and a reduced range of motion (ROM) due to mechanical changes to the passive properties of muscle.

1.2.2 Cerebral Palsy – Skeletal muscle properties

Studies on CP are typically based on small samples of children, making it difficult to determine the time course of normal adult muscle development. Alterations in the passive properties of CP muscle have been identified as early as the age of 3 years old (Mathewson and Lieber, 2015; Willerslev-Olsen et al., 2013), contributing to the muscle weakness, limited ROM and increased passive stiffness observed in even highly functioning individuals with CP (Kruse et al., 2017). Also apparent in the literature are changes in sarcomere length (Lieber and Fridén, 2002; Smith et al., 2011), fascicle stiffness (Barber et al., 2011), extracellular connective tissue stiffening with increased collagen (Lieber et al., 2004; Smith et al., 2011), and variation in muscle fibre size and structure (Lieber et al., 2004). This variation in fibre structure in CP muscle includes an increased number of “rounded” fibres, instead of a polygon shape, resulting in decreased force per muscle cross-sectional area (CSA) with increased extracellular (unused) space between fibres (Foran et al. 2005).

Willerslev-Olsen et al. (2018) recently found that the medial gastrocnemius muscle in children with CP had a normal growth pattern until 12 months of age, when the muscle then showed a significantly slower rate of growth when compared to TD children. A muscles force-generating capacity is related to its cross-sectional area, where the term physiological cross-sectional area (PCSA) may provide more practical relevance in pennate muscle, as it is the total area of the cross-sections perpendicular to the muscle fascicles. A reduced muscle volume, as seen in CP, could result from alterations in fascicle length (Smith et al. 2011), pennation angle
and/or muscle thickness (Lieber and Fridén, 2000). In CP muscle, these are common alterations to the mechanical properties of skeletal muscle tissue found in the literature, all of which affect the range through which they can develop force and power output capabilities (Barrett and Lichtwark, 2010; Pal, 2014).

1.2.3 The sarcomere – Effects of sarcomere length, serial sarcomere number, and sarcomeres in parallel

Within a muscle fascicle is a bundle of myofibres each of which consists of bundles of myofibrils. The myofibrils are composed of myofilaments, including actin, myosin and titin, which repeat along the length of the myofibril to make up the basic force-producing units within muscle, the sarcomere. Sarcomere length and the number of sarcomeres in parallel across the muscle will dictate a muscle’s force producing capability, and the number of sarcomeres in series will dictate a muscle’s shortening velocity. The passive and active tension developed by a muscle is length dependent, where active tension generated is a function of the number of cross-bridges formed per half-sarcomere across the full cross-sectional area (PCSA), i.e. proportional to the overlap between myofilaments, actin and myosin (Huxley and Niedergerke, 1954). In CP, as in TD, maintenance of a muscle in a shortened position can result in shortening of muscle fascicles as a result of loss of sarcomeres in series. This is due to serial sarcomere number changing in response to functional demands, where reduced limb activity, induced by immobilization can result in muscle atrophy, a reduction in sarcomeres in series and/or in parallel (Williams and Goldspink, 1978).

Alternatively, healthy muscle (unaffected by a neurological disorder) has been shown to add sarcomeres in series in response to postnatal development, chronic stretch, regeneration
following injury, or muscle growth (Williams and Goldspink, 1978). This response in healthy muscle is presumably to maintain an optimal functioning sarcomere length, i.e. the sarcomere length where the most cross-bridges can form and (muscular) active force can be produced. In humans, the theoretical maximal active force plateaus at sarcomere lengths 2.64μm to 2.81μm (Walker and Schrodt, 1974). The sarcomere length-tension relationship demonstrates the effect of sarcomere length on force (See Figure 1-1), where the ascending limb represents increasing muscle force as sarcomere lengths approach optimal, and the descending limb represents decreasing levels of force due to decreasing overlap between the myofilaments (Rassier et al., 1999). This relationship is specifically dependent on joint angle, muscle tendon length and number of sarcomeres within the muscle fascicle.

![Figure 1-1. Theoretical sarcomere length-tension relationship](image)

*The ascending limb of the sarcomere length-tension relationship increases from approximately 1.27μm to 2.64μm, with the max force occurring on the plateau (2.64μm to 2.81μm), and the*
descending limb decreasing to a theoretical maximum sarcomere length of 4.24\(\mu\)m (Rassier et al., 1999).

It is incredibly difficult to analyze whether a muscle has more or less sarcomeres in series, with observations of sarcomeres (and their lengths) within intact whole muscle or isolated myofibres being done primarily on cadaveric subjects until the 1990s (Wickiewicz et al., 1983). Recent advancements have allowed direct visualization of sarcomeres in living muscles, where these in vivo approaches still involve a high degree of invasiveness as a muscle fascicle will either be locally dissociated from a whole muscle, or a micrometer-sized endoscope can be inserted within a muscle (Moo et al., 2016). In addition, sarcomere lengths are non-uniform in vivo, meaning a mean sarcomere length from one location may not be representative of the distribution of sarcomere lengths across the whole muscle (Moo and Herzog, 2018). A less invasive method is to use various 2D and 3D medical imaging techniques of the muscle, where researchers will typically infer number of sarcomeres in series based on an assumed resting sarcomere length, and a direct measurement of muscle fascicle length in vivo.

Due to a lack of methodology in measuring fascicle length and sarcomere lengths at the same time in CP, inconclusive results exist in identifying differences between CP and TD (Lieber and Fridén, 2002; Malaiya et al., 2007; Mohagheghi et al., 2008, 2007; Moreau et al., 2013, 2009; Shortland et al., 2002). Some research has found CP presents similar fascicle lengths but with fewer sarcomeres and therefore longer sarcomere lengths, whereas alternative research has observed shorter fascicle lengths (clearly fewer sarcomeres in series). Regardless, both scenarios indicate sarcomeres in series are likely reduced in individuals affected by CP, which will result in a disadvantage in force production due to the reduced myofilament overlap (Figure 1-1).
1.2.4 Muscle fascicle force-length and force-velocity relationship

The *in vivo* force-length relationship of a muscle has a dome-shaped relationship, with force and length increasing on the ascending limb, a plateau where maximal force output will theoretically occur at “optimal” fascicle lengths, and a descending limb corresponding to decreasing force with increased length (Rassier et al., 1999). The interaction and overlap of the myofilaments, actin and myosin, within a sarcomere (sarcomere length) can determine a muscle’s maximal isometric force at different fascicle lengths (or joint angles) to quantify a fascicle force-length relationship. The force-velocity relationship demonstrates a muscle’s ability to produce force at a range of angular velocities, where at velocity equal to zero, force is at its highest (disregarding muscle lengthening contractions). With increasing muscle shortening velocities, force generation will decrease due to reduced force per cross-bridge and reduced number of cross-bridges engaged within a sarcomere. Muscle length is the primary determinant of a muscle’s shortening velocity and excursion, as a longer muscle fascicle would contain more sarcomeres in series (Moreau et al., 2013). With more sarcomeres in series, it would be expected that, at a given sarcomere length, the shortening speed of each sarcomere would be slower for a given speed of fascicle shortening, allowing for a right and upward shift of the curve and improving force output at similar or higher velocities.

Sport performance is largely determined by muscle power output, requiring maximal force generation at high shortening velocities, as well as an ability to maintain maximal force over a period of high intensity efforts. The power producing capability of a muscle can be analyzed through force-velocity relationships and muscle fascicle length, where a muscle with longer muscle fascicles (more sarcomeres in series) will generate a greater force at the same
absolute velocity (Moreau et al. 2013; Nasirzade et al. 2014). In addition, increased sarcomere number in parallel, or an increased muscle CSA, would be directly proportional to an increase in force production. Because of this, a majority of resistance training programs revolve around improving muscle architecture to maximize an athlete’s potential.

In the case for individuals affected by CP, both CSA (Barber et al., 2011; Barrett and Lichtwark, 2010; Malaiya et al., 2007; Mathewson and Lieber, 2015) and sarcomeres in series (Mohagheghi et al., 2007; Moreau et al., 2009; Smith et al., 2011) appear to be limited. In addition, sarcomeres operating at longer lengths would result in an active force-length relationship operating on the descending limb. Reduced serial sarcomere number and/or longer sarcomere lengths could then limit force at any muscle length or shortening velocity, contributing to reduced power production and therefore, sport performance. The literature has well documented the benefits of high-intensity strength training to improve muscle CSA and force production in CP, but studies confirming increases in muscle fascicle length or sarcomeres in series in CP muscle are lacking.

1.3 Purpose:

The purpose of this study is to determine whether muscle architecture in CP adapts with sport specific HVT, similar to those who are typically developing (TD) and unaffected by a neurological disorder. Muscle architectural differences will be compared across active CP, sedentary CP and TD individuals. The primary measure of this study was to measure resting fascicle length, with secondary measures of triceps surae muscle peak power, peak force and peak velocity.
A second purpose of this study is to identify the difficulties and potential challenges when measuring muscle moment arms, and therefore quantifying muscle force, in a population that shows large passive plantar flexion and dorsiflexion moments at the ankle. As limited research exists in this area, this knowledge would allow confirmation of the mechanism responsible for the weak moment we see in these individuals (weak muscle or short moment arm). We will compare two non-invasive methods to estimate moment arm of the triceps surae muscle in CP; the tendon travel method at different ROM, and the visual method.

1.4 Hypothesis:

It is hypothesized that:

1. High velocity training will increase resting muscle fascicle length in CP after 10 weeks.

2. An increase in fascicle length will also result in an increase in peak shortening velocity and muscle power output,

3. Muscle moment arms will be shorter in individuals with CP, contributing to a weak moment and reduced plantarflexion function.

1.5 Outline:

In the following chapter, a review of the literature will be presented. This review will focus on changes in the architecture and function of muscle of individuals with CP in comparison with TD individuals. This will be followed by two chapters presenting the research results; one focused on moment arm determination and one presenting the results related to fascicle length and power production. A final chapter will summarize the results, discuss the limitations of the study and present future directions.
Chapter Two: Critical Review of Current Literature

2.1 Purpose:

This literature review will critically evaluate published research conducted on muscle architectural differences, and the effects of training adaptations to muscle in those affected by Cerebral Palsy (CP). A majority of muscle research in CP is focused on improving muscle tissue growth, structure and function as these areas appear to be limited in those with a neurological impairment. We still do not fully understand the causes of muscle weakness and dysfunction, but consensus amongst researchers is growing resulting in improvements in the methodology to assess and treat people with CP.

2.2 Muscle weakness in CP

Weakness, as defined by Edwards (1978), is a failure or inability to generate or maintain an expected level of force. Muscle weakness as a clinical characteristic of CP has been long documented in research, where the term “Cerebral Palsy” means weakness originating at the brain. It is likely that muscle weakness would be accentuated by sedentary behavior (disuse or immobilization), and because of this, recent research in CP has been highly focused on improving activity and increasing muscle strength, with reports of improved functional abilities and changes to muscle architecture with training interventions (Dodd et al., 2002; Moreau et al., 2013; Reid et al., 2010; Willerslev-Olsen et al., 2014). Understanding the causes of muscle weakness in CP are important to appropriately design training/rehabilitation programs or clinically prescribe treatments targeting improvements in locomotion and daily function. Muscle growth appears to be reduced in CP as early as 12 months after birth (Willerslev-Olsen et al., 2018), and is likely affected by a multitude of factors including
neuronal innervation, mechanical demands (immobilization or prolonged stretch), endocrine factors and nutrition, all of which can influence protein synthesis and degradation (Gough and Shortland, 2012). Figure 2-1 illustrates the complexity of the different factors involved in regulating muscle tissue growth, contractile properties of muscle, and the stiffness of the tissue (Pingel et al., 2017).

Figure 2-1. Tissue homeostasis in the neuromuscular-tendon-connective tissue complex.

Normal and healthy tissue is regulated through homeostasis of a multitude of factors. Alterations to the homeostasis may result in adaptation to the muscle, tendon or connective tissue. In Cerebral Palsy, the formation of contracture is likely a result of alterations to one or many of the pathways shown above. (AMPK, 5-AMP-activated protein kinase; FAK, focal adhesion kinase; mn, motor neuron; NFAT, nuclear factor of activated T-cells.) Pingel et al. (2017) with permission.
For simplicity of this thesis, we will highlight the secondary effects of these changes to the muscle-tendon-connective tissue complex. Specifically, the decreased voluntary force production, proposed to be a result of muscle architectural differences (including a decreased muscle CSA) (Barber et al., 2011), and/or altered muscle activity (Wiley and Damiano, 1998). Lastly, weakness may also be a result of the reduced mechanical advantage to produce force at the joint, as a result of smaller muscle moment arm in CP (Kalkman et al., 2018).

2.2.1 Muscle architectural differences

Muscle architecture is defined as the physical arrangement of muscle fibres, motor units, and connective tissue elements that can determine a muscle’s mechanical function and performance capacity. In relation to the mechanical function, a muscle’s CSA is directly related to its maximal force output, and muscle length is the primary determinant of a muscle’s shortening velocity and excursion (Moreau et al., 2013). The literature is consistent in the findings for reduced muscle cross-sectional area (CSA) present in individuals with CP when compared to typically developing (TD) children (Barrett and Lichtwark, 2010), with medial gastrocnemius (MG) muscle volumes found to be decreased by nearly 50% (Malaiya et al., 2007; Mathewson and Lieber, 2015). A reduced muscle volume reflects not only decreases in cross-sectional area, but may also result from alterations to muscle fascicle length, i.e. sarcomeres working in series (Lieber and Fridén, 2000). Combined, a reduced muscle fascicle length and CSA would result in a reduced number of sarcomeres working in series and parallel, causing a detrimental effect on a muscle’s capability to produce high forces, particularly at high shortening speeds.
In CP, muscle architecture can also be affected through increased collagen content, titin stiffening and increased sarcomere lengths, which have been proposed mechanisms to the increased passive resistance to stretch or development of contracture (Figure 2-2) (De Bruin et al., 2014; Pingel et al., 2017). Findings of 51% higher mean values of ankle stiffness (Barber et al., 2011) would require greater amounts of force to cause deformation to muscle, contributing further to the observed muscle weakness in CP (Pal, 2014). Research surrounding the titin isoform has found titin unable to account for the increased passive stiffness of the muscle as a whole in CP, showing a more compliant isoform (Leonard et al., 2019; Smith et al., 2011) possibly due to findings of reduced titin content in CP (Leonard et al., 2019). It is also possible that sarcomeres within the muscle fascicle operate at longer lengths than in TD muscle (Leonard et al., 2019; Lieber and Fridén, 2002; Smith et al., 2011) and/or with fewer sarcomeres in series (Mohagheghi et al. 2008; Moreau et al. 2009). According to data on the sarcomere length-tension relationship of spastic flexor carpi ulnaris muscle in CP, the highly stretched sarcomeres identified in wrist flexion (3.54 ±0.3 \( \mu m \) vs. 2.3 \( \mu m \), p<0.001) would be predicted to generate only 40% of their maximum force and this magnitude would only decrease with extension (Lieber and Fridén, 2002).
2.2.2 Altered muscle activity; Spasticity, co-contraction, reduced muscle activation

In addition to reduced muscle CSA contributing to muscle weakness in CP, muscle specific tension (moment/unit area) is reduced by approximately 40% in the plantar flexors when compared to TD (Elder et al., 2003). Muscle weakness is also a result of reduced voluntary muscle activation in CP (Elder et al., 2003; Rose and McGill, 1998), with findings of prolonged muscle activation and relaxation times affecting forces at high speeds (Downing et al., 2009). The net muscle moment at a joint is a product of all muscle forces acting around the joint,
therefore co-contraction of the agonist and antagonist muscle groups, along with increased reflex excitability/spasticity can be further debilitating to muscle function in CP.

Historically, it was believed that excessive co-contraction and spasticity must be reduced prior to introducing any type of movement training. This was based on the notion that the altered muscle activity is the result of the observed motor deficits in CP (Bobath B., 1990). Strength training individuals with CP was not recommended, instead, the emphasis was placed on treatment for alleviating spasticity as it was believed to be exacerbated with excessive effort (Bobath B., 1990). It is now well understood in the literature and practiced by clinicians, that maximal effort strength training can improve muscle strength in CP without adverse effect/spasticity (Clark et al., 2006; Fowler et al., 2001; Mockford and Caulton, 2008; Patten et al., 2013). The literature should be reviewed with caution, as there are discrepancies in studies measuring clinical signs of spasticity and relating it to the reduction in movement function (Dietz and Sinkjaer 2007).

The reflex-excitability/spasticity present in CP was also believed to contribute to the reduced muscle volume and/or development of muscle contracture (Hägglund and Wagner, 2011). Spasticity’s effect on muscle growth has recently been challenged by authors, where impaired muscle growth and increased muscle passive stiffness have been found to precede observed increases in reflex-excitability in CP muscle (De Bruin et al., 2014; Gough and Shortland, 2012; Willerslev-Olsen et al., 2018). Signs of impaired triceps surae muscle growth and passive stiffness are apparent without reflex-mediated stiffness present, at as early an age as 12 and 27 months, respectively (Willerslev-Olsen et al., 2018). This coincides with interventions aiming to reduce spasticity, where anti-spastic medications had no effect on the development of contractures (Tedroff et al., 2011). It is also apparent that stretching, splinting, and casting may
have a small immediate effect on reducing spasticity or increasing joint mobility, but little to no long-term effects (Katalinic et al., 2011). Alternatively, exercise is often used to treat and prevent muscle contractures and is generally recommended for children and adults with CP (Geertsen et al., 2015; Verschuren et al., 2016).

2.2.3 Shorter moment arm in CP

Moment arm (MA) is defined as the perpendicular distance from the joint centre of rotation to the line of action of the muscle, and is needed to measure joint forces (Pandy, 1999). A shorter MA would result in a reduced mechanical advantage to produce a moment at the joint, and in the case for CP, a shorter triceps surae muscle MA would further impair plantar flexion function. This is important, as the muscle weakness observed in CP could then be attributed to either (or both) smaller plantar flexor muscle CSA or a shorter triceps surae MA compared to TD (Kalkman et al., 2017). Correct triceps surae MA calculations would also have important clinical implications for determining appropriate surgical procedures targeting equinus (toe-walking) gait, to improve muscle forces during walking. In the next section, methods commonly used for estimating moment arm will be introduced, along with the limitations surrounding each.

2.3 Moment Arm

To directly measure muscle force in vivo would be highly invasive and time consuming, where measuring force at the ankle joint would involve implanting a buckle-type transducer on the Achilles tendon (Fukashiro et al., 1993). Therefore, researchers will typically resort to estimating actual muscle forces from measured joint moments, which can be done with knowledge of the muscle MA (An et al. 1984). MA can be estimated using a number of non-
invasive methods, but there is no true gold standard in the measurement of joint MA due to limitations to these common methods, which are described in detail below. Limited research exists on MA in CP (Alexander et al., 2019; Kalkman et al., 2017), with disagreement between the two studies on whether MA in CP is longer or shorter than TD. Furthermore, these studies are limited to children with CP.

2.3.1 Magnetic Resonance Imaging

Magnetic Resonance Imaging (MRI) can offer a high-quality imaging of boney structures under static conditions with either two or three-dimensional (2D or 3D) methods, but both are costly and time-consuming. At the ankle, MRI methods can directly image the tarsal bones from which the joint centre of rotation at the talocrural joint can then be estimated. MA can then be determined by measuring the perpendicular distance from the assumed joint centre to the line of action of the MTU; this is usually taken as the Achilles tendon. Both 2D and 3D methods require a knowledge of anatomy to accurately estimate the joint centre of rotation, where 2D imaging has been found to overestimate Achilles tendon MA in comparison to 3D methods (Hashizume et al., 2012). This overestimation can be attributed to an inaccurate estimation of the joint centre of rotation, due to the image not captured and orientated orthogonal to the talocrural joint axis. The overestimation may also be a result of the difference in the anteroposterior position of the lateral and medial malleoli, as they are not perfectly aligned.

2.3.2 The visual method

The visual method of estimating MA (Scholz et al., 2008) is less costly and time consuming than MRI methods, involving the use of standardized photographs to calculate the
Achilles tendon MA from visually measuring the perpendicular distance from an assumed apparent centre of rotation to the assumed line of action of the muscle group (Figure 2-3). Typically, the visual method will use images of the ankle at different angles to find the “functional axis” (Wade et al. 2019), and the Achilles tendon moment arm is taken to be the shortest distance from the assumed centre of rotation to the midline of the tendon. An alternative would be to estimate the transmalleolar midpoint, the midline between the medial and lateral malleoli, or the transmalleolar axis which would run on a diagonal directly through the medial and lateral malleoli. The visual method has similar limitations as 2D MR imaging, but without the talocrural joint in view, the joint centre can only be estimated with the medial (or lateral) malleolus in view.

![Figure 2-3. Moment arm estimation using the visual method.](image)

*The dotted line represents the triceps surae muscle line of action, and perpendicular distance from the centre of rotation (closed circle) to the Achilles tendon midpoint (open circle). Here only the medial malleolus is in view, with the transmalleolar midpoint estimated as the centre of rotation.*
Recent research from Wade et al. (2019) with 2D imaging and reflective markers, found that using the functional axis, over the transmalleolar axis or midpoint, resulted in larger overall values and changing MA with plantar flexion and loading. The use of the anatomical landmarks, such as the transmalleolar axis or midpoint, did not show variation in MA with increasing joint angle or under load (Wade et al., 2019). Typically, experimental studies have reported an increase in moment arm with increasing plantar flexion angles (Kalkman et al., 2017; Maganaris et al., 2000), but there have also been reports that Achilles tendon moment arm remains nearly constant across angles both by 3D imaging using MRI (Hashizume et al., 2012), and the tendon travel method, when accounting for passive moments acting to stretch the tendon (Fletcher and MacIntosh, 2018).

2.3.3 The tendon travel method

The tendon travel method does not require knowledge of a joint centre of rotation which may otherwise introduce error into the estimation. The tendon travel method has also been found to be in line with MA estimations using the 3D MRI method, but is more cost and time effective for researchers. This method calculates MA from the change in tendon displacement (dL) relative to change in ankle angle (dθ radians), where the ratio dL/dθ is the estimated MA. To monitor tendon displacement, ultrasound imaging is used to track a clear fascicle-aponeurosis intersection, with the displacement of the fascicle-aponeurosis junction representing the tendon displacement (Figure 2-4) (Ito et al., 2000; Maganaris et al., 2000). This method is based on the principle of virtual work, which assumes a constant force is applied to the tendon during passive rotation (An et al., 1984). Given that tendons are compliant and stretch when a force is applied, and passive moments are present during dorsiflexion (Fletcher and MacIntosh, 2018), the
principle of virtual work is violated, and so the measured tendon elongation observed during dorsiflexion may be limited to a restricted joint range of motion.

**Figure 2-4. Ultrasound image; Sample tracking of fascicle-aponeurosis intersection.**

*Left image shows tracked (red arrow) fascicle-aponeurosis intersection at rest. Right image shows fascicle-aponeurosis displacement (dashed line from original red arrow position to final position) during plantar flexion of a passive trial.*

During ankle dorsiflexion, the plantar flexor muscle group contributes a passive plantar flexion moment as a result of resistance to stretch from the muscle (Barber et al., 2011; Fletcher and MacIntosh, 2018; Kalkman et al., 2018), causing an additional lengthening of the Achilles tendon over and above the tendon displacement resulting from passive joint rotation. Depending on the material properties of the tendon (i.e. compliance), even a small passive moment can result in substantial tendon elongation, thus affecting MA estimates. Fletcher and MacIntosh (2018) found that at ankle angles greater than 90° (plantarflexion) in TD subjects, the tendon travel method should prove valid as no appreciable passive plantar flexion moment is present. They also present data that MA should not change as a function of ankle angle, although it has been demonstrated that MA will increase in length with plantar flexion due to the posterior
translation of the calcaneus (Leardini et al., 1999; Maganaris et al., 2000). It is important to note that the calcaneus also rotates as it translates, possibly resulting in a constant MA throughout the ankle ROM as suggested by Fletcher and MacIntosh (2018). Therefore, if all moments acting on the tendon are quantified, the tendon travel method should prove to be a valid method in estimating MA length.

2.3.4 Research and limitations in measuring moment arm in CP

All three of these methods’ present dilemmas in estimating triceps surae MA accurately in CP. These include bony deformities found in CP (Stevenson et al., 1995; Theologis, 2013) which may affect the orientation and location of the centre of rotation, and high and variable passive muscle forces which may affect the measured tendon displacement. The tendon travel method assumes a constant force is applied to the tendon during passive rotation (An et al., 1984), but given that tendons are compliant and stretch when a force is applied, the increasing passive resistive forces present during dorsiflexion in CP would violate the principle of virtual work.

Only two previous studies have estimated MA in CP children, one of which used tendon displacement (Kalkman et al., 2017) and the other, MRI imaging (Alexander et al., 2019). Our results (see Chapter 3) reveal a similar average MA in CP as Kalkman et al. (2017) found at 20° of plantar flexion. It is important to note Kalkman et al. (2017) estimated tendon displacement by monitoring calcaneus displacement. This was likely done to avoid the need to correct for changing passive moment on the tendon, by monitoring the position of Achilles tendon insertion at the calcaneus with clusters of markers on the skin over the bone. Specifically, the change in tendon displacement is estimated in relation to an external marker placed on the skin at the
assumed muscle-tendon junction. This method also assumes that any change in external marker
displacement is equal to the tendon-aponeurosis displacement at the muscle-tendon junction.
Maganaris (2005) supported the assumption that calcaneus displacement is equal to the
myotendinous junction displacement, because the skin marker displacement gives minimal error
(0.2 ± 0.4mm) during a passive rotation of the ankle. This method also assumes passive net joint
moments are low and the muscles are inactive, as any change in ankle angle due to heel lift or
compression of the footpad on the footplate would result in error. Although Kalkman et al.
(2017) confirms passive net moment was low (1-2 Nm), it is difficult to explain the linear
moment arm–angle relationship, with absolute MA lengths ranging from as low as 5mm to as
high as 80mm across both CP and TD children.

Alexander et al. (2018) found larger absolute mean triceps surae muscle MA lengths at
20° of plantarflexion in CP children (47.1 ± 3.5 mm) in comparison to TD children (41.8 ± 5.85
mm). The authors also compared absolute mean MA lengths for TD adults (range 22-48 years,
52.8 ± 5.62mm) from an existing dataset (Clarke et al., 2015). MA at 20° of plantar flexion was
estimated by extrapolating the data acquired from in vivo MRI scans to an Opensim lower limb
model. When CP children were compared to TD adults, no significant differences were found
between the TD adults and CP children (p=0.082). Considering children likely have smaller foot
bones and a smaller triceps surae muscle CSA compared to fully grown adults, these MA values
seem extremely large for the CP children. A comparable study by Hashizume et al. (2012), also
using MRI, found much lower mean MA estimates of 40.7 ± 5.3mm in their TD adults. Using
the tendon travel method, Fletcher and Macintosh (2018) found mean MA measurements in adult
distance runners were approximately 33 ± 7 mm. In addition, it has been demonstrated that MA
remains nearly constant across ankle angles both by 3D imaging using MRI (Hashizume et al.,
2012), and the tendon travel method, when accounting for passive moments acting to stretch the tendon (Fletcher and MacIntosh, 2018). This is not the case in other studies using 2D (Kalkman et al. 2017) or 3D (Alexander et al. 2018) estimates of MA.

2.4 Methods for measuring muscle fascicle length

Our understanding of human muscle from the sarcomere to whole muscle had relied primarily on human cadaveric data up until the 1990s. Fixed tissues of cadavers would not accurately represent the architecture of actively contracting muscles. Skeletal muscle architecture is now reliably assessed in vivo microscopically utilizing laser diffraction methods or macroscopically with various medical imaging techniques including conventional B-mode ultrasonography and MRI.

2.4.1 Intraoperative laser diffraction

The laser diffraction technique involves directly measuring sarcomere lengths by dissecting a single fibre segment, securing the ends to custom made clamp, and then transilluminating the sample with a laser (Lieber et al., 1984). This technique is commonly used by Lieber and colleagues, where he also (using laser diffraction) completed the first noninvasive measurements of muscle sarcomere length in a swimming glass catfish (Lieber et al., 1992), as well as in intact human wrist extensor muscles of those undergoing surgery for tennis elbow (Lieber et al., 1994). Noninvasive methods used by Lieber intraoperatively, include sliding a laser device beneath a small fibre bundle in vivo.

According to Moo et al. 2016, sarcomere lengths are typically measured at a single spot (mid-belly) and assumed to represent the sarcomere lengths at other locations within the muscle
(i.e. Sarcomere lengths are uniform). Moo et al. (2016) found non-uniform in vivo sarcomere lengths across a mouse tibialis anterior muscle when stretched passively, and later found that mean sarcomere lengths obtained from a single location are not necessarily representative of the distribution of sarcomere lengths in active muscle (Moo and Herzog, 2018). Therefore, studies inferring fascicle lengths or applying these assumptions of sarcomere length at a single spot to the force-length relationships of that muscle need to be analyzed with caution.

2.4.2 2D and 3D Imaging: B-mode ultrasonography and MRI

Although MRI may be considered the “gold standard” muscle imaging modality, access or affordability to MRI is limited. Alternatively, conventional B-mode ultrasonography is more cost-effective, and provides a more feasible option to visualize muscles in vivo. Ultrasound can be used to directly measure muscle fascicle length, with the potential to also detect structural alterations, i.e. muscle atrophy, non-contractile tissue (Pitcher et al., 2015). With the analysis of a muscle length, it is possible to infer the number of sarcomeres in series (Fukunaga, 1997; Rutherford and Jones, 1992; Smith et al., 2011). The medial gastrocnemius provides a reliable choice, as the muscle fascicles can be viewed entirely within an image without the need to extrapolate outside the field of view. For those muscles that extend outside the image, alternative methods are available allowing segments of the images to be strewn together.

Ultrasound misalignment is likely the most important error source for fascicle length measurements, where orienting the transducer perpendicular to the surface of the leg results in the smallest absolute error in fascicle length (0.4mm per degree of misalignment) (Bolsterlee et al., 2016a, 2016b). In an earlier study by Bolsterlee et al. 2015, they found that skin compression from the ultrasound technique does not bias fascicle lengths or deep pennation.
angles but does cause a systematic underestimation of superficial pennation angles. Therefore, by ensuring a perpendicular placement of the ultrasound probe and alignment of the superficial and deep aponeuroses, technician measurement error can be reduced significantly.

It has been proposed that the sarcomeres may be operating at longer lengths in CP, with data supporting these findings in the semitendinosus, adductor longus, gracilis, soleus, and flexor carpi ulnaris muscles (Leonard et al., 2019; Lieber and Fridén, 2002; Mathewson et al., 2014; Smith et al., 2011). Resting sarcomere lengths for the soleus muscle at maximum dorsiflexion in CP were found to be 4.07\(\mu m\) in comparison to results found for TD (2.67\(\mu m\))(Mathewson et al., 2014). Without directly measuring sarcomere lengths, it may be possible to analyze a muscles force-length relationship (Gordon et al., 1966), where sarcomeres operating at longer lengths would result in a force-length relationship operating on the descending limb (see Figure 2-5).
Figure 2-5. Theoretical sarcomere force-length relationship for TD and CP subjects

Sarcomere lengths for CP (O symbol) appear to be longer than TD (Δ). Sarcomere resting lengths for MG muscle in CP are not available, but the sarcomere resting length for TD subjects of the MG in anatomical position is 2.733μm (Cutts, 1988). Resting sarcomere length for the soleus in maximum dorsiflexion in TD (2.67μm) are shorter than CP (4.07μm) (Mathewson et al., 2014). Data for resting sarcomere lengths in other CP muscles: gracilis (3.54μm) and semitendinosus at 90 degrees of flexion (3.62μm) (Smith et al., 2011).

2.5 Fascicle and sarcomere lengths in CP

Due to the lack of methodology in measuring muscle fascicle length and sarcomere lengths in CP at the same time, inconclusive results exist in identifying differences between CP and TD (Lieber and Fridén, 2002; Malaiya et al., 2007; Mohagheghi et al., 2008, 2007; Moreau et al., 2013, 2009; Shortland et al., 2002). Variations in study conclusions could also be in part
due to differences in GMFCS level in CP participants, inadequate sample sizes, or age and sex may also play a role in differences in overall muscle adaptation and function. We are aware of one publication involving the author Lieber, that was the first to use both ultrasound and intraoperative laser diffraction techniques on the same segments of muscle removed from individuals with CP and control (cadaver) TD subjects (Mathewson et al. 2014). Their results support that fascicle length may not be different in CP, but sarcomere length was dramatically longer in children in CP (4.07 ± 0.45 μm vs. TD = 2.17 ± 0.24 μm; p<0.0001). Overall, it appears that number of sarcomeres in series is reduced, by either a reduced serial number resulting in a shorter fascicle length, or by increased sarcomere length resulting in fascicle lengths similar to TD.

In 2002, Lieber and colleagues assessed the wrist extensor muscles in 6 CP subjects with wrist flexion contractures using intraoperative laser diffraction, and found overstretched sarcomeres of 3.54 ± 0.30 μm (Lieber and Fridén, 2002). Findings of overstretched sarcomeres in CP have been consistent in the research completed by Lieber, including in the TD subjects with tennis elbow pain (Mathewson et al., 2014; Ponten et al., 2007; Smith et al., 2011). Although Lieber states that care was taken when removing segments of muscle and/or measuring sarcomere lengths intraoperatively, it is possible that this process could have resulted in an inaccurate elongation of sarcomere length. Lieber’s results are supported in recent literature using isolated fascicles and laser diffraction, where the mean in vivo sarcomere lengths for CP children (approximately 3.56 μm) are on the descending limb of the force-length relationship (Leonard et al., 2019).

Of the first to measure muscle fascicle length in CP was Shortland et al. (2002). Shortland et al. (2002) used ultrasound-based measures of MG fascicle lengths in 7 CP children
(7 to 11 years, 31.8 ±9.8mm), and found the fascicle lengths in CP to be similar to 5 TD adults (24 to 36 years, 37.4 ± 10.1mm). A larger sample size may have highlighted a significant difference between groups, but even then, the authors have chosen to compare children to adults which will have confounded their results with different stages of growth. Mohagheghi et al. (2008) and Moreau et al. (2009) both found shorter muscle fascicles in CP, where their reliability using ultrasound measures of muscle fascicle length were proven excellent (ICC 0.91-0.97) and included appropriate sample sizes (N= 18 and 16, respectively). A study by Frisk et al. (2019) analyzed MG muscle fascicle length range in TD adults (36 years) and ambulatory adults with CP (36 years). They reported reduced resting fascicle lengths from dorsiflexion to plantarflexion of 47.8mm to 26.6mm in the CP group, compared to 73.6 to 45.4 mm in TD. Resting and active MG muscle fascicle lengths were analyzed in TD and CP young adults (18± 2years, 59±3 kg) by Barber et al. (2012), finding significant reductions (34%) in resting MG fascicle lengths in the CP group. The active MG muscle fascicle lengths were not significantly different (-17%) at maximal dorsiflexion in CP compared to TD, which is likely attributable to the 55% less MG fascicle shortening during maximal stimulation in the CP group.

Muscle atrophy, or reduction in the number of sarcomeres in series and parallel, are important causes of the reduced force generation and velocity of muscle shortening observed in CP. It has been shown that youth with CP have reduced torque at higher velocities of movement (Damiano et al., 2000) and reduced rapid force generation (Moreau et al., 2012), resulting in detrimental reductions in muscle power output. Reduced MG muscle fascicle lengths would reduce the speed and endurance capability during walking in this population, where increases in muscle fascicle length may improve walking or running speeds.
2.6 Methods for increasing muscle fascicle length

Where training that increases muscle CSA can result in improved force output, training that increasing muscle length can result in greater shortening velocities (Moreau et al., 2009). A muscle with longer muscle fibres will generate a greater force at the same absolute velocity, as the shortening speed of each sarcomere in a fascicle would be relatively slower for a given speed of whole fibre shortening (Moreau et al. 2013; Nasirzade et al. 2014). To improve muscular force and power, sport training uses different methods to promote these muscle architectural changes. Considering architectural adaptations are training/contraction specific, sport science professionals can choose the appropriate training methods for a specific result. Hypertrophy training is a proposed mechanism for increasing muscle CSA and fascicle length, as it consists of lengthening and/or shortening contractions to promote large amounts of stress on the muscle through a full range of motion (Lynn and Morgan, 1994; Proske and Morgan, 2001). Repeated bouts of heavy resistance training can result in increased muscle fascicle length, CSA and increased motor unit recruitment (Coffey and Hawley, 2007), where an increase in muscle CSA due to heavy resistance training, may not be associated with an increase in muscle fascicle length (Fukutani et al., 2015). Performing these muscle shortening or lengthening actions at high velocities could promote even larger changes to muscle fascicle length, and result in increased active muscle range of the muscle’s force-length relationship (Moreau et al., 2013). Possible interventions proposed for increasing fascicle length in unaffected muscle include static stretching, bracing, loaded muscle lengthening (eccentric) training, and high velocity training. Whether these interventions hold true in CP is still relatively unknown. These approaches could be ineffective, considering muscle in CP does not appear to keep up with the stimulus of normal bone growth.
2.6.1 Surgery

Tendon transfer, intramuscular lengthening, and distal femoral extension osteotomy surgeries exist as clinical procedures used for individuals with CP to correct equinus gait deformities and improve muscle function (Dreher et al., 2012; Salami et al., 2018; Svehlik et al., 2012). Operations on the Achilles tendon and/or triceps surae muscle complex are often used to correct contracture of the gastrocnemii and soleus, to restore the ROM of the ankle by decreasing excessive passive moment (Svehlik et al., 2012). Muscle lengthening is more commonly used over tendon-lengthening surgeries in the shank due to a reduced risk of overcorrection (Dreher et al., 2012). After surgery it’s possible a muscle may become stronger or weaker depending on the change in the sarcomere length-tension relationship from its starting condition. For example, if a sarcomere is longer than optimal pre-surgery, increasing its length during surgery will further decrease its force production. In contrast, Svehlik et al. (2012) found improved dorsiflexion angles at the ankle and enhanced power generation at push-off following surgery, indicating that the sarcomere length-tension relationship may have improved following intramuscular lengthening. For tendon lengthening surgeries, there is a risk for over lengthening the Achilles tendon, which has been shown to result in calcaneal deformity and crouch gait (Shore et al. 2010). Although ROM may improve after tendon lengthening, research has shown that a longer tendon and shorter muscle belly is already present in CP (Gao et al., 2011), increasing the tendon length could have further consequences on moment-generating capacity of the triceps surae in comparison. Although many options for surgeries exist, to reduce the possibility of negative functional consequences, both sarcomere and muscle fascicle lengths need to be defined prior to surgery.
2.6.2 Chronic stretching and bracing

Methods utilizing chronic stretching or bracing have consistently found increases in end-range joint angles (muscle extensibility), but without the observation of a shift in the passive moment/angle curve (no increase in sarcomeres in series) (Weppler and Magnusson, 2010). Concerns of long-term bracing include atrophy of the immobilized muscle (reduced muscle CSA) and/or lengthening at the tendon, resulting in a shortened muscle (reduction in sarcomeres in series and fascicle length) (Hösl et al., 2015; Simard et al., 1982; Williams and Goldspink, 1978). This shows the importance of including evaluations of moment and CSA of muscle into studies to reflect actual fascicle length change, not just measures of extensibility (Blazevich et al., 2014; Magnusson et al., 1997; Weppler and Magnusson, 2010). Training protocols that involve exercises that preserve or improve muscle fascicle length and/or CSA would be more effective interventions, especially for those affected by CP.

2.6.3 Loaded lengthening contractions

Loaded lengthening contractions place greater stress and cause more damage to muscle than shortening contractions. Studies have consistently shown this type of training to result in increased fascicle length in healthy subjects (Coffey and Hawley, 2007; Franchi et al., 2014; Lynn and Morgan, 1994; Proske and Morgan, 2001). In 2010, Reid et al. determined the neuromuscular outcomes of a loaded muscle lengthening strength program for children and adolescents with CP. After a 6-week program of 3 sessions per week of upper limb lengthening contractions, children with CP had improved peak moment when normalized to body mass and EMG activity which decreased to similar levels after training as TD children (decreased co-contraction). The authors found no change in muscle fascicle length at the peak of the elbow moment-angle curve, which would imply this program did not increase the number of
sarcomeres in series. Although muscle fascicle length at peak torque may not have changed, there was a significant change in the mean moment-angle relationship width to a more similar width as the TD group (p=0.015). An increase in the width of the moment-angle relationship would result in a greater muscle length range for force production, and greater torque production at all elbow angles.

2.6.4 High velocity contractions

Incorporating strength training with high velocity contractions is another proposed mechanism for increasing fascicle length with a correspondent increase in muscular power. Research in TD (Blazevich et al., 2003), older adults (McKinnon et al., 2017) and stroke patients (Patten et al., 2013) have shown improvements in muscle power output after resistance training with a focus on high-velocity shortening. Unfortunately, these along with other training interventions for people with CP appear to elicit slow (<30°·s⁻¹) to moderate (120°·s⁻¹) training speeds. Available research for HVT in CP is limited, but some studies have found the newly gained muscle strength after strength training may not transfer to sports and higher level activities unless the strength training velocity is higher (Moreau et al., 2012; Park and Kim, 2014). At higher velocities of walking or running, it is likely the limited ROM and reduced muscle fascicle length observed in CP may limit their maximal joint angular velocity and/or muscle shortening speed. This would also affect the ability for these individuals to run and jump at sport-specific velocities, or those high-velocity movements required during sport.

Blazevich et al. (2003) were of the first to show fascicle length increases in response to an athletic training intervention. They utilized a sprint/jump protocol over 5 weeks and attributed the improvements in sprint and jump performance of their subjects to muscle
architectural changes, specifically a decrease in fascicle angle and increased vastus lateralis muscle fascicle length. Sharifnezhad et al. (2014) completed a randomized control trial (RCT) utilizing training groups completing vastus lateralis muscle lengthening training at different magnitudes (low-65% and high-100% MVC) at different training velocities (slow-90°·s⁻¹ and high-240°·s⁻¹). They found an importance of the magnitude of the fascicle lengthening, but more importantly the addition of the high velocity muscle fascicle shortening in increasing sarcomeres in series. The angular velocity of 240°·s⁻¹ at 100%MVC was a more effective training velocity, considering the average knee joint angular velocities during a whole movement of jumping (70 to 225°·s⁻¹), running (190 to 230°·s⁻¹) and sprinting (~320°·s⁻¹)(Sharifnezhad et al., 2014).

There are two studies we are aware of that emphasized training at moderate-high velocities in CP (Kirk et al., 2016; Moreau et al., 2013). Kirk et al. (2016) developed a 12-week heavy resistance training protocol (36 session) which focused on “explosive” execution during 4 isolated lower body exercises on machines (toe raise, calf raises, leg press, and hamstring curls). They hypothesized that the training would positively impact the neural activation and mechanical properties of muscle and connective tissue. This group did not measure muscle volume, muscle fascicle length, nor reveal velocity of movement or change in neural activation. They did find rate of force development (0-30 and 0-50ms) and active ROM in the dorsiflexors improved, but the authors were unable to show any improvements in plantar flexion push-off velocity during walking (180 to 220°·s⁻¹), passive or reflex-mediated stiffness, or in any of the walking speed and endurance tests (6-minute walk test, Timed Up and Go).

Moreau et al (2013) developed an 8-week (24 session) training protocol using a Biodex dynamometer where subjects completed 6 sets of 5 knee extensions, each at an increasing velocity (30 to 120deg/s). For the 9 CP subjects, the authors found a statistically significant
increase in rectus femoris fascicle length (p=0.012, d=1.25), angular velocity (p< 0.001), peak power (p=0.023), and faster walking speeds (p=0.03). This study only trained at slow-moderate speeds, with subjects reaching 90 to 120 deg/s for their last two sets by (on average) their 12th and 16th session, respectively. This joint angular velocity is quite slow, considering the push-off velocity during walking found by Kirk et al. (2016) exceeds the maximal training velocities from the Moreau et al. (2013) study. Ankle maximum plantar flexion and dorsiflexion velocities between TD and CP don’t appear to be significantly different during walking (Granata et al., 2000), but no data (that we are aware of) exists on ankle joint velocities in CP during running, jumping or sprinting. To compare to individuals without a neurological disorder, ankle joint velocities of 400-600°•s\(^{-1}\) have been found during running (Heidenfelder et al., 2008). 800°•s\(^{-1}\) found during the push-off phase of a jump (Bobbert et al., 1987a, 1987b), and approximately 1500°•s\(^{-1}\) during a sprint (Bezodis et al., 2008).

These ankle angular velocities during running, jumping and sprinting in TD far exceed those used in any training intervention in CP, or that can be completed on a commercial dynamometer. To achieve enhancements in movements at high-velocities, these individuals will likely need to train specific movements at high-velocities.
Chapter Three: **Mechanisms of reduced plantar flexor function in Cerebral Palsy; smaller triceps surae moment arm and reduced muscle force**

### 3.1 Abstract

Cerebral Palsy (CP) is a movement disorder with muscle mechanics involvement, including reduced moment. To accurately measure muscle forces, knowledge of muscle moment arm (MA) is required. The tendon travel method of estimating MA requires passive forces to be constant, but those with CP have large, changing passive forces with a limited range of motion. 12 young adults with CP (Gross Motor Function Classification System I or II, 15-32 years) and 10 typically developing (TD) peers (17-26 years) had their triceps surae muscle MA quantified by tendon travel and by visually measuring the perpendicular distance from an assumed centre of rotation to Achilles tendon midpoint. MA was calculated as the ratio of tendon displacement to joint rotation during passive rotation (0.17rad·s⁻¹), at 90° and at a reference angle (~106°) where net passive moment was zero. Similar average MA in CP subjects was found at the reference angle for tendon travel (28.8 ± 5.6mm) and visual method (29.1 ± 5.5mm), but absolute within subject difference between these methods was large (2.9 ± 1.9mm). TD subjects had significantly larger triceps surae muscle MA than CP subjects, with MA at the reference angle for tendon travel (35.4 ± 4.1 mm) and visual method (35.4 ± 3.6 mm) being 6.3 to 6.5mm larger, respectively. The results of this study show the challenges in estimating MA in CP, where large passive forces are present and regardless of method, both shorter triceps surae MA and decreased muscle force contribute to the reduced plantar flexion function observed in CP.
3.2 Introduction

Cerebral Palsy (CP) is a permanent movement disorder with non-progressive muscle mechanics and neurological involvement as a result of a lesion in the brain stem occurring before, during or just after birth (Palisano et al., 2008). Individuals with CP commonly have a significantly smaller muscle cross-sectional area (CSA), with corresponding muscle weakness, as well as reduced joint range of motion (ROM) (Barber et al., 2012). The limited ROM has been attributed to high muscle-tendon unit (MTU) stiffness limiting muscle excursion, likely due to passive (not reflex-mediated) muscle stiffness (Willerslev-Olsen et al., 2013). Muscles in CP may have increased sarcomere lengths (Lieber and Friden 2002), increased fascicle stiffness (Barber et al., 2011), and/or increased collagen content /stiffening within the extracellular matrix (De Bruin et al., 2014; Lieber et al., 2004; Smith et al., 2011). The plantarflexors are commonly affected by the movement disorder, where limited plantar flexion moment, ROM, and rate of force development have been found to impair walking and running speed and endurance (Vulpen et al., 2017). A shorter triceps surae moment arm (MA) would further compromise plantar flexion function by reducing the maximal moment at the joint. The muscle weakness observed in CP has been attributed to either (or both) smaller plantar flexor muscle CSA or a shorter triceps surae MA compared to typically developing (TD) controls (Kalkman et al., 2017).

Correct triceps surae MA calculations would have important clinical implications for determining appropriate surgical procedures targeting equinus (toe-walking) gait, to improve muscle function during walking. Researchers will typically estimate muscle forces from measured joint moments and knowledge of the muscle/tendon moment arm (An et al. 1984), because direct in vivo measurements of force are highly invasive (Fukashiro et al., 1993). Moment arm, defined as the perpendicular distance from the joint centre of rotation to the line of
action of the muscle, can be estimated using a number of non-invasive methods (Pandy, 1999). Magnetic Resonance Imaging (MRI), although costly and time consuming, can offer a high-quality image of the boney structures at the ankle, allowing estimation of the axis of rotation at the talocrural joint. The visual method of estimating MA (Scholz et al., 2008) is less costly and time consuming, involving the use of photographs to calculate the surae MA from measuring the perpendicular distance from an apparent centre of rotation, to the assumed line of action of the triceps surae muscle. The tendon travel method is also a cost and time effective option. This approach does not require knowledge of a joint centre of rotation, which may otherwise introduce error into the estimation. The tendon travel method allows calculation of the MA from the change in tendon displacement (dL) relative to change in ankle angle (dθ, in radians). To monitor tendon displacement, ultrasound imaging is used to track a clear fascicle-aponeurosis intersection, with the displacement of the fascicle-aponeurosis junction representing the tendon displacement (Ito et al., 2000; Maganaris et al., 2000).

Currently, there is no gold standard in the measurement of MA. Further, all three methods present dilemmas in estimating triceps surae MA accurately in CP. These include boney deformities (Stevenson et al., 1995; Theologis, 2013) which may affect the orientation and location of the centre of rotation, and high and variable passive muscle forces which may affect the measured tendon displacement. The tendon travel method is based on the principle of virtual work, which assumes a constant force is applied to the tendon during passive rotation (An et al., 1984). Given that tendons are compliant and stretch when a force is applied, the increasing passive resistive forces present during dorsiflexion in CP will violate the principle of virtual work.
To our knowledge, there are only two studies in which measurement was made of triceps surae muscle MA in CP; both included high functioning CP children (7-13 years) with a Gross Motor Classification (GMFCS) I and II. There is disagreement as to whether MA in children with CP is larger (Alexander et al. 2018) or smaller (Alexander et al., 2019; Kalkman et al., 2017) than in TD children.

Given these inconsistencies in the estimation of MA in CP, the purpose of this study was to compare two common non-invasive methods to estimate the MA of the triceps surae muscles in individuals with and without CP. In order to not violate the principle of virtual work, we aimed to measure MA using the tendon travel method at an ankle angle where net moment acting on the tendon is zero and compared these measures to the visual method. Our intent was to determine the triceps surae MA length in CP and TD young adults. It was hypothesized that both a smaller MA and decreased muscle force could be contributing factors to the reduced plantar flexion function observed in CP.

3.3 Methods

3.3.1 Subjects

12 Young adults with CP (GMFCS I or II) and 10 young TD adults without a neurological disorder volunteered for this study. Subjects gave their informed written consent to the experimental procedures prior to data collection. The University of Calgary Conjoint Health Research Ethics Board approved the study (#REB15-2026). Subject characteristics are presented in Table 3-1. All subjects had no injury, lower limb surgery or botulinum neurotoxin A (Botox) injections within the previous 6 months.
Table 3-1. TD and CP subject characteristics

<table>
<thead>
<tr>
<th>Group</th>
<th>Gender</th>
<th>N</th>
<th>Age (years)</th>
<th>Height (m)</th>
<th>Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TD</td>
<td>Male</td>
<td>4</td>
<td>23.5 ± 1.3</td>
<td>1.74 ± 0.08</td>
<td>70.0 ± 6.0</td>
</tr>
<tr>
<td>TD</td>
<td>Female</td>
<td>6</td>
<td>21.8 ± 3.5</td>
<td>1.63 ± 0.07</td>
<td>66.3 ± 10.5</td>
</tr>
<tr>
<td>CP</td>
<td>Male</td>
<td>8</td>
<td>22.3 ± 6.3</td>
<td>1.69 ± 0.09</td>
<td>64.3 ± 14.4</td>
</tr>
<tr>
<td>CP</td>
<td>Female</td>
<td>4</td>
<td>23.8 ± 6.2</td>
<td>1.57 ± 0.09</td>
<td>57.0 ± 8.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD

3.3.2 EMG and moment

To measure passive moment, the subject laid on their side with their right or more affected foot affixed to a footplate of a Dynamometer (Biodex System 3, Shirley NY). Pairs of surface bipolar electromyography (EMG) electrodes (Norotrode 20 bipolar Ag-AgCl electrodes, Myotronics Inc, Kent, WA, USA, interelectrode distance: 22±1 mm) were placed over the tibialis anterior (TA) and lateral gastrocnemius (LG) muscles to monitor muscle activation during maximal voluntary contractions (MVC) and passive stretch. An electrode over the medial malleolus served as ground. The ultrasound probe prevented monitoring EMG of the MG, so the LG was chosen as a surrogate (Fiebert et al., 2018). Performing the measurement in the horizontal plane removed any moments resulting from gravity; the weight of the footplate and weight of the foot (Fletcher and MacIntosh, 2018). The subject’s ankle was passively rotated by the dynamometer at 0.17rad·s⁻¹ through the full ROM, measuring the passive moment and EMG generated during the trials. This slow velocity was chosen to avoid initiating a stretch reflex (Lorentzen et al., 2010). Antagonist and agonist activity were monitored for co-activation. Any
EMG root mean square (RMS) above 3 standard deviations (SD) from baseline noise, measured prior to the trial, would have been considered muscle activation. In no case did tibialis anterior (TA) activation exceed 3 SD above zero during the passive trials.

To measure peak active moment, the subject was transitioned to a prone position with the same foot affixed to a footplate of the dynamometer. The subject completed a randomized trial of isometric MVCs of the plantarflexors at a fixed relative percentage (0-20-40-60-80-100%) of their ankle ROM. The highest peak moment of 3 repetitions at each of the 6 ankle angles were kept for analysis. Absolute ankle angles (deg) were recorded at each relative ankle angle, and active moment was corrected for any extra joint rotation and gravitational forces during the MVC. Muscle force was calculated as the ratio of the moment measured during the trial and the estimated muscle triceps surae moment arm. Active moment values were calculated by subtracting the passive moment measured at the corresponding fascicle length, to correct for muscle length changes during contraction (MacIntosh and MacNaughton, 2005).

3.3.3 Tendon travel method

To calculate MA using the tendon travel method, ultrasound (12.5 MHz linear array, Philips Envisor, Eindhoven, Netherlands) of the MG muscle belly was used to acquire images of the deep and superficial aponeuroses and MG fascicles to a depth of 3 cm and frequency of image collection was 49 Hz (Figure 3-1). A clear fascicle-aponeurosis intersection was tracked at 6 time points through the full ROM using publicly-available image analysis software (Image J, Baltimore, MD).
The change in displacement of the fascicle-aponeurosis intersection was assumed to be equal to the tendon translation (dL) as a result of ankle rotation (dθ) (Figure 3-2). Passive moment change would have caused the tendon to change length (Fletcher and MacIntosh, 2018). Tendon travel (dL) with respect to ankle angle (θ) for each subject were fitted to a 3rd-order polynomial equation across the measured range of motion where a, b, c and d are constants:

$$dL = a\theta^3 + b\theta^2 + c\theta + d.$$  [Eq. 1]

For both CP and TD subjects, the MA was calculated from the 1st derivative of Eq.1, at 90° and at an ankle angle (referred to as reference angle) where net passive moment was zero. A subgroup of CP subjects (n=9) had the protocol repeated 10 weeks later, to quantify the test-retest reliability and the within-subject coefficient of variation between measurements.
3.3.4 Visual method

To calculate MA with the visual method, the subject laid prone with their foot affixed to a footplate of a Dynamometer (Biodex System 3, Shirley NY) at or near 90° (Figure 3-3). The same foot was used for the visual method as for the tendon travel method. A ruler was secured to the edge of the foot plate, to serve as a reference scale. A mark was made at the apparent centre of rotation of the ankle (i.e. talocrural joint), specifically slightly posterior and inferior to the medial malleolus to account for potential diagonal alignment of the true joint axis of rotation relative to the centre of the malleoli (Lundberg et al., 1989). The diagonal alignment of the malleoli would be referred to as the transmalleolar axis, where the transmalleolar midpoint would be the midline between the medial and lateral malleoli (Wade et al., 2019). The medial
aspect of the foot and leg were then photographed (Apple iPad, 9.3.5) perpendicular with the plane of ankle rotation. The perpendicular distance from the assumed centre of rotation to the Achilles tendon midpoint, representing the triceps surae muscle line of action, representing the MA, was measured using ImageJ.

Figure 3-3. Moment arm estimation using the visual method.
Note: The dotted line represents the triceps surae muscle line of action, and perpendicular distance from the centre of rotation (closed circle) to the Achilles tendon midpoint (open circle).

3.3.5 Statistics

Values are presented as mean ± standard deviation. A two-way repeated measures analysis of variance (ANOVA) was used to assess differences in MA between CP and TD (group) comparing the visual and tendon travel methods at reference angle. Paired t-tests were used to compare CP and TD for total ROM, height and peak active moment. The agreement
between repeated measures of MA in the CP group was assessed according to Bland and Altman 1999. Statistical significance was considered \( p \leq 0.05 \).

### 3.4 Results

#### 3.4.1 Range of motion

Maximum dorsiflexion and plantar flexion angles, total ROM for TD and CP are reported in Table 3-2. ROM was significantly reduced in CP compared to TD.

<table>
<thead>
<tr>
<th>Range (deg)</th>
<th>TD</th>
<th>CP</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max DF</td>
<td>73.7 ± 4.9</td>
<td>82.3 ± 9.8</td>
<td>p&lt; 0.02</td>
</tr>
<tr>
<td>Max PF</td>
<td>143.5 ± 6.5</td>
<td>128 ± 9.1</td>
<td>p&lt; 0.001</td>
</tr>
<tr>
<td>Total ROM</td>
<td>69.8 ± 6.6</td>
<td>45.7 ± 12.3</td>
<td>p&lt; 0.001</td>
</tr>
</tbody>
</table>

Values are mean ± SD

#### 3.4.2 Moment and EMG

Peak active moment during the MVC was significantly lower in the CP group (53.9 ± 17.4 Nm) when compared to the TD group (147.0 ± 50.2 Nm). There was a smaller average MA in CP, and peak active force (N) was significantly lower in the CP group (1983.8 ± 887.0 N) compared to TD (4104.9 ± 1154.9 N, \( p < 0.001 \)). The passive moment-angle relationship for the CP group had higher passive moments present for any given ankle angle (Figure 3-4), where the steeper moment-angle relationship below zero is associated with a higher dorsiflexion moment
for a given ankle angle. An independent t-test revealed significantly higher passive moment at a similar ankle angle in the CP group (7.4 ±4.4 Nm, 100% ROM: 82.3 ±9.8°) than TD (4.2 ±1.32 Nm, 82.3°, p=0.037). During the MVCs, the CP group had a significantly lower EMG RMS for LG (0.111±0.113 V) compared to the TD group (0.347±0.201 V, p<0.01).

Figure 3-4. Passive-moment angle relationship for representative CP & TD subjects.

Passive moment-angle relationships for a representative TD participant (open squares) and CP participant (closed diamonds). The TD subject displays a sigmoidal relationship over a larger ROM, where minimal passive moment (± 2Nm) occurs at ankle positions highlighted in light gray. The CP subject displays a less obvious sigmoidal relationship, where minimal passive moment occurs at the ankle positions highlighted in dark grey. Zero represents the ankle angle for both subjects where passive moment crossed zero (reference angle). Positive positions are in dorsiflexion with an associated positive (plantarflexion) moment from the triceps surae muscle group. Negative positions are in plantarflexion, with an associated negative (dorsiflexion) moment presumably from the tibialis anterior muscle.
3.4.3 Moment arm and method of measurement

A two-way repeated measures ANOVA revealed that MA was not significantly different when comparing the tendon travel method at the reference angle to the visual method across all subjects (p=0.935; see Table 3-3). There was a significant main effect of group (p=0.002); CP MA was significantly shorter compared to TD, regardless of method. The reference angle was similar for both CP (106.5 ± 8.0°) and TD (106.4 ± 7.6°).

3.4.4 Height and moment arm

MA at the reference angle had a very strong correlation with height in the TD group for both the tendon travel (r=0.861, p=0.001) and visual methods (r = 0.907, p<0.001) and. In the CP group, height was moderately correlated to MA when using the tendon travel method (r=0.577, p=0.05), with a weak correlation to MA when using the visual method (r=0.215, p=0.502) (see Figure 3-5).
Figure 3-5. Moment arm-height relationships; tendon travel vs. visual method

A: TD; Tendon travel method at reference angle.

B: TD; Visual method at 90°.

C: CP; Tendon travel method at reference angle.

D: CP; Visual method at 90°.

* Correlation is significant at the p<0.05 level.

** Correlation is significant at the p<0.001 level
Table 3-3. Mean moment arm lengths.

<table>
<thead>
<tr>
<th>Method</th>
<th>Measurement position</th>
<th>TD (mm)</th>
<th>CP (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tendon travel</td>
<td>90°</td>
<td>30.1 ± 5.6</td>
<td>23.6 ± 13.9</td>
</tr>
<tr>
<td>Tendon travel</td>
<td>Reference angle</td>
<td>35.4 ± 4.1</td>
<td>28.8 ± 5.6 *</td>
</tr>
<tr>
<td>Visual</td>
<td>90°</td>
<td>35.4 ± 3.6</td>
<td>29.1 ± 5.5 *</td>
</tr>
</tbody>
</table>

Values are mean ± SD
*Significantly different at the p<0.01 level (2-tailed) when compared to TD.

3.4.5 Test-retest reliability and variation between methods

A Bland & Altman plot (Bland and Altman, 1986) was used to compare MA estimates between the first and second measures (n=9) within methods (Figure 3-6). Both methods showed a weak correlation, but the visual method showed a smaller mean bias (0.8 mm) between test/retest compared to the tendon travel method (6.2 mm).
Figure 3-6. Bland & Altman plots comparing first and second moment arm estimates.

A: Visual Method at 90°. B: Tendon Travel method at reference angle. Limits of agreement are represented by a dotted line.

3.5 Discussion

Our results confirm that moment arm is significantly smaller in young adults with CP (~6.5 mm smaller than TD), contributing to a significantly reduced peak moment (~63%). As calculated peak force was also reduced (~52%), it is apparent that a reduced triceps surae muscle CSA and reduced muscle activation, contribute to the reduced plantar flexion function in CP. As expected, we observed that MA was reliably estimated for the TD group, with similar mean MA estimates for both the visual and tendon travel methods (Table 3-3). The CP group also showed similar mean MA estimates between methods but had substantial within-subject absolute differences in MA, reducing our confidence in these estimates. In spite of the large variability, we demonstrate a significant difference in MA between TD and CP. This study provides a good
first step in identifying methodological challenges and barriers researchers face when estimating MA and muscle forces in CP.

3.5.1 ROM and passive muscle properties of muscle in CP

Reduced muscle growth in CP has been identified as early as the age of 12 months (Willerslev-Olsen et al., 2018), contributing to the muscle weakness, limited ROM and increased passive stiffness observed in even high-functioning individuals with CP (Kruse et al., 2017; Mathewson and Lieber, 2015; Willerslev-Olsen et al., 2013). Our data confirmed ROM in CP is reduced by nearly 35% when compared to TD, with reduced maximum plantar flexion and dorsiflexion angles. The significantly reduced ROM and reduced active muscle forces observed in CP is related to 51% higher values for ankle stiffness (Barber et al., 2011). It is likely that progressive extracellular matrix stiffening due to increasing collagen content is involved in the increased passive resistance to stretch in CP (De Bruin et al., 2014; Pingel et al., 2017). Another proposed mechanism for increased passive forces in CP has been that sarcomeres operate at longer lengths than in TD muscle (Leonard et al., 2019; Lieber and Fridén, 2002; Smith et al., 2011). The passive force contributed by titin, would be proportionally higher at longer sarcomere lengths.

Our data revealed a passive moment-angle relationship with a narrow range of ankle positions where passive moment was minimal (below 2Nm). The triceps surae would account for most of the resistance to stretch during dorsiflexion, while the TA muscle will resist dorsiflexion in the passive moment-angle relationship (see Figure 3-4). We assumed a dorsiflexion moment would not have an effect on tendon lengthening in plantar flexion angles. However, it is possible that the net passive moment was close to zero with some dorsiflexion and
plantar flexion moment present. Coexisting moments would be more likely in CP and this may have contributed to the variability in the tendon travel method of measuring MA. The fact that EMG of the dorsiflexors and plantarflexors was at rest levels during these passive moment measurements would reduce the likely contribution from these muscles.

3.5.2 Tendon travel method

Moment arm is difficult to validate across studies, as literature values tend to vary based on subject stature and/or methodology used. It appears our MA estimates (35.4 ± 4.1) using the tendon travel method in the TD group is similar to previous studies (averaging 35.1mm) analyzed at a similar ankle angle (90° to 100°) (Fath et al., 2010; Lee and Piazza, 2009; Manal et al., 2013). If all moments acting on the tendon were quantified, the tendon travel method at the reference angle should have proven valid in estimating MA length for both the CP and TD groups. This assumption is difficult to confirm.

Within the CP group, the results revealed that limits of agreement between the first and second measurements of MA using the tendon travel method was large (-16.0 to 11.3 mm) (Figure 3-6). In an attempt to minimize error in our measurement, the protocol was done in the side lying position to remove the need to correct for gravity, and the speed chosen appears to not have elicited an active stretch-reflex response or activation of the TA (confirmed from the measurements of EMG). Therefore, the variability between methods within the CP group could at least be partially attributed to the large plantar flexion moments present through their limited ROM.
3.5.3 Visual method

The visual method showed minimal variation between the first and second measurements of moment arm in the CP group, where any differences can be attributed to within-tester measurement error. When considering CP subjects, bony deformities may be present altering the foot mechanics and/or alignment of the malleoli. These boney deformities are likely a result of bone adapting to altered loading patterns and structural alterations to tissue (Theologis, 2013), but it is likely that the physical appearance of the axis of rotation for each subject will not vary between repetitive measurements unless the subject has undergone surgery. The visual method appears to provide researchers with a greater reliability between measurements in comparison to the tendon travel method. Overall, it is important the researcher standardize the measurements across subjects to improve the reliability in using the visual method.

3.5.4 Moment arm research in CP

Only two previous studies have estimated MA in CP children, one of which uses tendon travel/displacement (Kalkman et al., 2017) and the other, MRI imaging (Alexander et al., 2019). Our results reveal a similar average MA in CP as Kalkman et al. (2017) found at 20° of plantar flexion. It is important to note Kalkman et al. (2017) estimated tendon displacement by monitoring calcaneus displacement. This was likely done to avoid length change of the tendon due to changing passive moment. This method assumes that any change in external marker displacement is equal to the tendon-aponeurosis displacement at the muscle-tendon junction. This method could be corrupted by any change in ankle angle due to heel lift or compression of the footpad on the footplate. Although Kalkman et al. (2017) confirm passive net moment was
low (1-2 Nm), it is difficult to explain the linear moment arm–angle relationship, with absolute MA lengths ranging from as low as 5mm to as high as 80mm across both CP and TD children.

Alexander et al. (2018) found larger absolute mean triceps surae muscle MA lengths at 20° of plantar flexion in CP children (47.1 ± 3.5 mm) in comparison to TD children (41.8 ± 5.9 mm). The values were model derived from the in-vivo MA measurement from MRI scans, and the authors also compared absolute mean MA lengths for TD adults (22-48 years, 52.8 ± 5.6 mm) from an existing dataset (Clarke et al., 2015). When CP children were compared to TD adults, no significant differences were found (p=0.082). Considering children likely have smaller foot bones and a smaller triceps surae muscle CSA compared to fully grown adults, these MA values seem extremely large for the CP children.

A comparable study by Hashizume et al. (2012), also using MRI, found much lower mean MA estimates of 40.7 ± 5.3mm in their TD adults. In addition, it has been demonstrated that MA remains nearly constant across ankle angles both by 3D imaging using MRI (Hashizume et al., 2012), and the tendon travel method, when accounting for passive moments acting to stretch the tendon (Fletcher and MacIntosh, 2018). A study by Wade et al. (2019) used reflective markers and 2D imaging and found that MA did not change as a function of increasing plantar flexion angle or loading if the transmalleolar axis or midpoint were used over the functional axis of rotation. This is not the case in other studies using 2D (Kalkman et al. 2017) or 3D (Alexander et al.2018) estimates of MA.

3.6 Conclusion

Inaccurate estimations of MA could result in incorrect calculation of muscle forces and/or ineffective clinical treatment. By identifying the challenges in estimating MA in CP as well as
limitation to previous literature, it will be possible to improve methods used to determine muscle forces in these individuals. Our data confirm that passive forces at the ankle are higher in CP, likely contributing to variability in MA estimates while using the tendon travel method. Therefore, in estimating triceps surae muscle MA non-invasively in CP, we suggest that the visual method is preferred because of the high reliability.

It has been well documented in the literature that individuals affected by CP have reduced plantar flexor strength, and our data support this finding showing reduced plantar flexor strength (~52%) along with significantly smaller moment arm lengths (~18%) in CP young adults. A smaller moment arm length reduces the mechanical advantage for moment generation at the joint, and can have detrimental effects on walking performance in CP. Both plantarflexor muscle weakness and a shorter triceps surae MA contribute to the reduced plantar flexion function.
Chapter Four: MUSCLE LENGTH ADAPTATIONS TO HIGH-VELOCITY TRAINING IN YOUNG ADULTS WITH CEREBRAL PALSY

4.1 Abstract

Fascicle length is an important determinant of a muscle’s shortening velocity and excursion. Changes in fascicle length have been shown in typically developing adults following a period of high-velocity training (HVT); however, studies confirming this adaptation in Cerebral Palsy (CP) muscle are lacking. Improvements in fascicle length may allow these individuals to improve their muscular power output and therefore enhance functional movements including those used in sport performance. The purpose of this study was to evaluate high-velocity training as an intervention strategy to increase medial gastrocnemius (MG) muscle fascicle length and improve peak power outputs in CP. 12 young adults with CP (GMFCS I or II, 15-30 years) were randomly allocated to no training (CP-NT)(n=8), or training (CP-T) (n=8) for 10 weeks. 10 typically developing (TD) recreationally trained young adults (17-26 years) were also recruited for the study. The training consisted of 1-hour sessions biweekly for 10 weeks (20 sessions), with exercises involving sprints, plyometrics and agility tasks. Triceps surae muscle force-power-velocity and force-length relationships were quantified pre and post training using dynamometry and ultrasound. Triceps surae muscle moment arm was significantly different between TD (35.4 ± 3.6) and CP (29.1 ± 5.5mm, p=0.002). The CP-T group had a significant increase in resting fascicle length (+4.1 mm, p<0.002) at a common ankle angle of 105°, but no significant change in power output and peak velocity. High velocity training was able to increase fascicle length, but additional training may be needed to enhance power output.
4.2 Introduction

Cerebral Palsy (CP) is a permanent movement disorder resulting from an upper motor neuron lesion in the brain occurring before or during birth. Although the brain damage is not progressive, secondary adaptations to muscle structure, function and composition occur causing abnormal muscle activity and difficulty with coordination. Consistent evidence in the literature identifies a reduced muscle cross-sectional area (CSA) present in individuals with CP when compared to their typically developing (TD) peers (Barrett and Lichtwark, 2010). As a muscle’s function is determined by its architecture, a reduced muscle volume may result in alterations to muscle fascicle length or CSA (Lieber and Fridén, 2000). While a muscles CSA is directly related to its force output, muscle length is an important determinant of a muscles shortening velocity and excursion (Moreau et al., 2013). Combined, a reduced muscle fascicle length and CSA would result in a reduced number of sarcomeres working in series and in parallel, causing a detrimental effect on a muscle’s power output. Findings of reduced muscle fascicle length in CP compared to TD are inconsistent in the literature, but it does appear that serial sarcomere number is reduced (Mathewson et al., 2014; Smith et al., 2011). This results in an average long sarcomere length. These inconsistent results are likely due to the difficulty in measuring fascicle and sarcomere lengths at the same time, or variation in the use of different normalization techniques. Regardless, these alterations to muscle can contribute to muscle weakness, limited range of motion (ROM) and increased passive stiffness in even high functioning individuals with CP (Kruse et al., 2017).

With increasing speed of muscle shortening, force production of a muscle will decrease as a result of decreased force per cross-bridge and fewer simultaneous cross-bridges, between actin and myosin, engaged. This forms the force-velocity relationship, demonstrating a muscles
ability to produce force at multiple velocities, where at velocity equal to zero, force is at its highest. Where velocity is zero, this type of contraction can be referred to as a maximal voluntary isometric contraction, and commonly used to assess maximal strength of a muscle group at a particular joint angle. Force can also be quantified during maximal voluntary contractions (MVCs) at increasing isokinetic speeds, where CSA and fascicle length can determine a muscle's capability to produce maximal force at high shortening velocities. It is possible, that a muscle can produce more force for a given speed of shortening, by increasing the number of sarcomeres in series within a muscle fascicle. This increase in muscular force at a particular velocity can occur as a result of a slower shortening speed of each sarcomere to develop force, for a greater given speed of whole fibre shortening.

Incorporating strength training with high velocity contractions is a proposed mechanism to target improvements in muscular power, by increasing the number of sarcomeres in series (Blazevich et al., 2003). This has been demonstrated in the literature using TD adults, where Blazevich et al. was of the first to find significant changes in vastus lateralis fascicle length with the incorporation of a sprint and jump training regime over 5 weeks. Studies confirming muscle fascicle length change and/or muscle power output increases with training in CP are lacking, and in part due to the “Bobath neurodevelopmental treatment approach” in the 90’s advising high intensity efforts in CP may increase spasticity (Bobath B., 1990). Research has since developed in CP (Fowler et al., 2001), identifying reduced muscle power in CP as a limiting factor during ambulation (Dallmeijer et al., 2011). An increase in fascicle length of the triceps surae could improve the power generation at the ankle joint, providing an effective push-off and improving the reduced step length and walking speeds found in CP (Vulpen et al., 2017). Although several methods have been used to increase muscle strength in CP to enhance the walking endurance, it
appears that the newly gained muscle strength may not transfer unless the strength training velocity is at a higher speed (Moreau et al., 2012; Park and Kim, 2014). Moreau, Holthaus, and Marlow (2013) tested this theory in a group of CP adolescents, finding a fascicle length increase correlating with significant improvements in faster walking speeds after strength training on a Biodex at moderate joint angular speeds of $120^\circ\cdot s^{-1}$.

To improve the ability of an individual with CP to partake in more than just daily activities, and increase their capability to be competitive in sport, true high velocity training (HVT) needs to be considered. This is largely in part that sport performance is determined by muscle power output, requiring both maximal force generation at high shortening velocities and an ability to maintain maximal force over a period of high intensity efforts. In the case for individuals affected by CP, both CSA and fascicle length appear to be limited, therefore limiting power production and likely, sport performance. As joint angular velocities in sport usually exceed speeds greater than $120^\circ\cdot s^{-1}$, no study to date has essentially investigated changes in fascicle length following true HVT ($>120^\circ\cdot s^{-1}$) in CP. Therefore, the purpose of this study is to determine whether muscle architecture in CP adapts with sport specific HVT, and whether an increase in muscle fascicle length results in increased triceps surae muscle power output.

4.3 Methods

4.3.1 Subjects

12 young adults with hemiplegic or diplegic CP in the lower leg muscle(s) and 10 TD recreationally-trained young adults without a neurological disorder were recruited. The CP group consisted of subjects with a Gross Motor Function Classification (GMFCS) level I (n=8) and II (n=4). 8 subjects within the CP group were identified as recreationally active or highly
trained (training 3 or more times per week), and 4 were identified as sedentary (not meeting Canada’s Physical Activity guidelines within the last 6 months). TD subjects included those who were participating in sprint-agility training 3 or more times per week. The CP group were randomly allocated in a cross-over design to no training (CP-NT)(n=6) or training (CP-T)(n=6) for 10 weeks. Within the CP-NT group, 2 subjects dropped out prior to the post control assessment and 2 subjects completed the training after serving as a control. Within the CP-T group, 4 subjects served as a control after completing the training. This resulted in 8 subjects participating in both the training and no-training groups. Subject characteristics can be found in Table 4-1. All subjects had no previous injury, lower limb surgery or Botox injection within the previous 6 months. Procedures for this study were approved by the University of Calgary Conjoint Health Research Ethics Board (#REB15-2026). Subjects gave their informed written consent for all experimental procedures.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sex</th>
<th>N</th>
<th>Age</th>
<th>Height</th>
<th>Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>TD</td>
<td>Male</td>
<td>4</td>
<td>23.5 ± 1.3</td>
<td>1.74 ± 0.08</td>
<td>70.0 ± 6.0</td>
</tr>
<tr>
<td>TD</td>
<td>Female</td>
<td>6</td>
<td>21.8 ± 3.5</td>
<td>1.63 ± 0.07</td>
<td>66.3 ± 10.5</td>
</tr>
<tr>
<td>CP</td>
<td>Male</td>
<td>8</td>
<td>22.3 ± 6.3</td>
<td>1.69 ± 0.09</td>
<td>64.3 ± 14.4</td>
</tr>
<tr>
<td>CP</td>
<td>Female</td>
<td>4</td>
<td>23.8 ± 6.2</td>
<td>1.57 ± 0.09</td>
<td>57.0 ± 8.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD
4.3.2 Moment arm estimation

Subjects had their triceps surae muscle MA quantified using both the tendon travel method (An et al., 1984), and the visual method (Fukunaga, 1997; Maganaris, 2003). The tendon travel method was found to be less reliable in comparison to the visual method (see Chapter 3) in the CP group, therefore only the visual method was used.

Refer to Chapter 3; page 42, 3.3.4 Visual method

4.3.3 EMG and moment

Refer to Chapter 3; page 37, 3.3.2 EMG and moment. Co-activation of the LG and TA using a 100ms window of the RMS EMG was monitored throughout the isometric and isokinetic MVCs for the TD and post assessment for the CP subjects.

4.3.4 Moment-angle and force-length relationships

The force-length relationship required the measurement of both passive and active moment (Nm) generated at each ankle angle, estimation of MA, and fascicle length measurement. Ultrasound (12.5 MHz linear array, Philips Envisor, Eindhoven, Netherlands) of the MG muscle belly was used to acquire images of the deep and superficial aponeuroses and MG fascicles to a depth of 3 cm and frequency of image collection was 49 Hz. Fascicle lengths were measured using publicly-available image analysis software (Image J, Baltimore, MD) done by the same examiner. Total moment is defined as the ankle plantar flexor strength generated during the active and passive trials, and muscle force was calculated as the ratio of the moment
generated during the trial and the estimated triceps surae muscle MA. Peak active moment values were calculated by subtracting the passive moment from the measured total moment at the corresponding fascicle length, to correct for muscle length changes during contraction. Peak passive and active moment values were gravity corrected; weight of the foot and foot plate. MG active fascicle length at peak isometric moment was recorded.

Muscle fascicle shortening (%) was calculated as:

\[
\frac{(\text{resting fascicle length} - \text{active fascicle length})}{\text{resting fascicle length}} \times 100
\]

Resting fascicle length (mm), pennation angle (deg) and muscle thickness (mm) were recorded at the ankle angle where peak isometric moment occurred. Resting fascicle length at a common ankle angle of 105° was compared across TD and CP subjects.

4.3.5 Moment-velocity and power-velocity relationships

The moment-velocity relationship was determined by completing MVCs on a Biodex dynamometer at six pre-determined and randomized isokinetic speeds (30, 60, 120, 180, 300, 500°•s⁻¹). Fascicle length at peak isokinetic moment was measured in order to calculate active moment (i.e. subtract the passive moment from the measured total moment at the corresponding fascicle length), and active moment values were gravity corrected. A linear regression equation was fitted to the active moment-angular velocity data, using the measured peak isometric moment as the y-intercept \(M_o\). Moment at any angular velocity \(v\) can then be determined using the calculated slope \(m\) and y-intercept \(M_o\) according to the equation:

\[
\text{Moment} = mv + M_o
\]

Peak angular velocity (rad•s⁻¹) or the x-intercept, can be calculated where moment = 0,

\[
v = - \frac{M_o}{m}
\]
Peak angular velocity and peak moment (Nm) at peak angular velocity were recorded for all subjects. Optimal velocity ($v_{opt}$) was determined as 50% of the estimated peak angular velocity. Where, moment = $mv + M_o$, power can be calculated at any velocity as power = moment * angular velocity, or:

$$\text{Power} = (mv + M_o) \times v = mv^2 + vM_o$$

Peak power was calculated as the moment generated at $v_{opt}$. This point also corresponds to where slope of the power regression relationship was equal to zero or the greatest regression value. Regression values were used instead of observed/measured values for the product of peak moment and the corresponding velocity.

$$\text{Peak Power} = mv_{opt}^2 + v_{opt}M_o$$

Average peak power for CP-T, CP-NT and TD groups were calculated by averaging the slope and y-intercepts ($m$ and $M_o$), and calculating peak angular velocity.

4.3.6 High velocity training program

A cross-over repeated measures design was implemented due to lack of subjects recruited, where 8 subjects participated in both the training (CP-T) and no-training (CP-NT) groups for 10 weeks each. 4 of the 12 subjects recruited chose to drop out and/or not partake in the training intervention. The training program was intended to target sport specific training velocities, or maximal ankle plantar flexion velocities. Movements included sprint, agility and plyometric drills, expected to reach maximal joint angular velocities greater than 400°•s$^{-1}$ (Figure
All subjects were required to complete a minimum of 80% attendance, or 16 of 20 sessions. The control group was instructed to continue their regular activities.

Figure 4-1. High velocity training program

HVT program for weeks 1-2, 3-6, and 7-10 are presented. Training days included a 10-minute warmup, followed by sprint drills (completed first on Day 1 and second on Day 2 of each training week) and agility/jump drills. Rest included 1 minute between sprint repetitions, and 2 minutes between sets. Sprints were based on distance, but if this distance was too long, a sprint time was also included to account for individual abilities. Pacing speeds were identified as easy, medium and fast to help subjects gauge their speed, where easy was also described as 50% max speed, Medium = 75%, Fast = 100% or as fast as possible. (Accel: accelerations = progressive increase in speed, Decel: decelerations = progressive decrease in speed, CMJ: countermovement jump, stride = hold given pace speed)
4.3.7 Statistics

A one-way ANOVA (and post-hoc analysis) was performed using SPSS V.20 (SPSS, Chicago, Illinois, USA) to determine the active force-power-velocity and force-length relationships between TD, CP-NT, and CP-T groups (part 1) at baseline. An ANCOVA was used in part 2 with pretest measures as covariate, to compare the post results for CP-T and CP-NT groups. An independent t-test was performed to determine differences between CP-T and TD groups for ROM, peak force, % fascicle shortening at peak force, EMG RMS for the LG and TA at peak force, resting fascicle length at 105° of ankle ROM, active fascicle length at peak force, pennation angle and muscle thickness.

4.4 Results

4.4.1 Range of motion and passive moment

Pre and post HVT results for maximum dorsiflexion and plantar flexion angles, and total ROM for CP-T and TD are reported in Table 4-2. ROM was significantly reduced in CP compared to TD (p<0.001), with no change in ROM for the CP-T group (p=0.525). Passive moment observed from the passive moment-angle relationships at maximal dorsiflexion was not significantly different before and after training CP-T group (p=0.243). An independent t-test revealed significantly higher passive moment at a similar ankle angle in the CP group (7.4±4.4 Nm, 100% ROM: 82.3 ±9.8º) than TD (4.2 ±1.3 Nm, 82.3º, p=0.037).
Table 4-2. Subject range of motion for TD and CP-T

<table>
<thead>
<tr>
<th>Range (deg)</th>
<th>CP</th>
<th>CP-T</th>
<th>TD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max dorsiflexion</td>
<td>82.8 ± 1.6</td>
<td>85.0 ± 1.6</td>
<td>73.7 ± 4.9*</td>
</tr>
<tr>
<td>Max plantar flexion</td>
<td>129.4 ± 2.7</td>
<td>132.2 ± 2.7</td>
<td>143.5 ± 6.5**</td>
</tr>
<tr>
<td>Total ROM</td>
<td>47.5 ± 1.9</td>
<td>48.0 ± 1.9</td>
<td>69.8 ± 6.6**</td>
</tr>
</tbody>
</table>

Values are mean ± SD

CP values are adjusted for covariate

*TD values are significantly different than the CP training group at the p<0.02

**TD values are significantly different than the CP training group at the p<0.001

Figure 4-2. Mean resting fascicle length-angle relationship for TD and CP subjects pre and post training

Maximum plantar flexion=0%, maximum dorsiflexion=100%
4.4.2 Differences in muscle architecture

Mean primary outcomes for CP-NT and CP-T groups are found in Table 4-3. As the groups were different at baseline, pretest measures were used as covariate. An ANCOVA revealed an overall statistically significant increase in measured resting fascicle length at 105° (Figure 4-2) \( p=0.002, \ d = 0.537 \) after training. No significant difference in post-measurements between CP-NT and CP-T groups were found for muscle thickness, pennation angle, and active fascicle length.

Table 4-3. Mean primary outcomes for CP, CP-NT, and CP-T

<table>
<thead>
<tr>
<th></th>
<th>CP</th>
<th>CP-NT</th>
<th>CP-T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle thickness (mm)</td>
<td>13.6 ± 0.5</td>
<td>14.0</td>
<td>14.2</td>
</tr>
<tr>
<td>Pennation angle (deg)</td>
<td>19.7 ± 1.8</td>
<td>19.3</td>
<td>20.3</td>
</tr>
<tr>
<td>Active fascicle length (mm)</td>
<td>30.4 ± 2.1</td>
<td>30.3</td>
<td>31.9</td>
</tr>
<tr>
<td>Resting fascicle length (mm)</td>
<td>42.0 ± 1.1</td>
<td>40.0</td>
<td>46.1*</td>
</tr>
<tr>
<td>Fascicle shortening (%)</td>
<td>33.4 ± 2.7</td>
<td>33.5</td>
<td>34.1</td>
</tr>
<tr>
<td>Peak power (W)</td>
<td>80.7 ± 8.8</td>
<td>83.4</td>
<td>85.63</td>
</tr>
<tr>
<td>Peak velocity (rad·s(^{-1}))</td>
<td>5.6 ± 0.5</td>
<td>5.5</td>
<td>6.14</td>
</tr>
<tr>
<td>Peak Moment (Nm)</td>
<td>55.6 ± 5.1</td>
<td>61.6</td>
<td>52.16</td>
</tr>
<tr>
<td>Peak force (N)</td>
<td>1965.4 ± 171.6</td>
<td>2171.6</td>
<td>1812.8</td>
</tr>
</tbody>
</table>

Values are mean ± SD

CP values are mean adjusted for covariate

*Measure was significantly different than pre-test measure at the p<0.002 level

TD and CP-T group differences can be found in Table 4-4. Significant differences muscle thickness \( p<0.001 \), active fascicle length at peak moment \( p=0.05 \), and resting fascicle
length at 105° (p<0.001) were also present. No differences in pennation angle were found between TD and CP groups.

Table 4-4. Mean primary outcomes for TD and CP-T

<table>
<thead>
<tr>
<th>Variable</th>
<th>TD</th>
<th>CP-T</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle thickness (mm)</td>
<td>18.1 ± 2.0</td>
<td>14.0 ± 2.0*</td>
<td>0.001</td>
</tr>
<tr>
<td>Pennation angle (deg)</td>
<td>16.2 ± 2.8</td>
<td>20.9 ± 6.3</td>
<td>0.134</td>
</tr>
<tr>
<td>Active fascicle length (mm)</td>
<td>47.8 ± 11.4</td>
<td>32.6 ± 9.0*</td>
<td>0.025</td>
</tr>
<tr>
<td>Resting fascicle length (mm)</td>
<td>61.2 ± 9.7</td>
<td>42.5 ± 11.8*</td>
<td>0.002</td>
</tr>
<tr>
<td>Fascicle shortening (%)</td>
<td>48.1 ± 8.5</td>
<td>33.9 ± 11.9*</td>
<td>0.022</td>
</tr>
<tr>
<td>Peak power (W)</td>
<td>439.1 ± 171.5</td>
<td>75.0 ± 42.5*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak velocity (rad·s⁻¹)</td>
<td>11.8 ± 1.9</td>
<td>5.7 ± 1.7*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak Moment (Nm)</td>
<td>146.8 ± 49.9</td>
<td>50.3 ± 17.4*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peak force (N)</td>
<td>4631.3 ± 2021.5</td>
<td>1670.0 ± 572.4*</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Values are mean ± SD

4.4.3 Moment-angle and force-length relationships

There were no significant differences in post-measurements between CP-T and CP-NT groups for peak moment, peak force, percentage fascicle shortening at peak force (Table 4-3) or EMG RMS of the LG or TA. An independent t-test revealed significantly lower peak isometric moment in the CP-T group (50.3 ± 17.4 Nm) compared to TD (146.8 ± 49.9 Nm, p<0.001). MA was significantly lower across all CP participants (29.1 ± 5.5mm) compared to TD (35.4 ± 3.6mm, +6.3mm, p<0.01). Compared to TD, the CP-T group also had a significantly lower percentage fascicle shortening (33.9 ± 11.9%, 48.1 ± 8.5%, p=0.022) and lower peak isometric force (1670.0 ± 572.4, 4631.3 ± 2021.5, p<0.001) (Table 4-4). Mean active force-length values for both TD and CP-T can be observed in Figure 4-3. During the MVCs, the CP group had a
significantly lower EMG RMS for LG (0.111±0.113 V) compared to the TD group (0.347±0.201 V, p<0.01). Co-activation of the TA and LG was apparent during both the isokinetic and isometric trials for the CP participants (measured during the post-assessment for all CP-T and CP-NT subjects). For the CP group, TA moment accounted for 50.7±81.3 % (6.5±5.9 Nm) of the isometric moment produced by the triceps surae across 6 different ankle angles. During the isokinetic trials, TA moment accounted for 21.4±27.9 % of the peak moment produced by the triceps surae in the CP group, and 11.3±12.6 % in the TD group.

![Figure 4-3. Mean force-length relationships for TD and CP-T](image)

Mean and SD for force-length relationships for CP-T and TD groups. CP-T fascicle length and active force are still substantially reduced in comparison to TD. Values for CP-NT or pretest
values are not shown, as the averaging of force-length relationships did not accurately represent the individual results. The relationship appeared linear as some subjects’ force-length relationships were operating on the descending limb, and others on the ascending limb.

4.4.4 Moment-power-velocity relationships

No significant difference between CP-T and CP-NT groups were found for peak power or peak velocity (Table 4-3, Figure 4-4). There was a significant difference in peak power and peak velocity between CP-T and TD groups (p<0.001; Table 4-4).

The slope of the force-velocity relationship was determined by a linear regression of the force-velocity values. The measured peak isometric moment from the force-length MVC data were used to calculate the linear regression relationship, versus using an estimated peak isometric moment value. For CP-T, the average difference between measured peak isometric moment (51.2 ± 17.8 Nm) and estimated peak isometric moment (46.1 ± 13.6 Nm) were statistically different (p=0.001). The average fit (r²= 0.822 ± 0.12, 0.835 ± 0.12) and slopes (-11.18 ± 5.03, -11.71 ± 5.52) of the force-length data were not statistically different, therefore using the measured peak isometric moment to calculate the slope of the force-velocity relationship was preferred.
Figure 4-4. Moment-velocity and power-velocity relationships for CP and TD groups

No significant increase in peak isometric moment, peak power or peak velocity after training in the CP-T group (—). *TD (·-) values are significantly different at a p<0.001.
4.5 Discussion

This study investigated the effects of a HVT intervention on muscle power output and muscle architectural changes in CP. Our main finding was that resting muscle fascicle length can increase after 10 weeks in response to a high-intensity training program involving sprinting and jumping activities that involve high velocity ankle rotation. We originally hypothesized that an increase in fascicle length would also result in an increase in peak muscle shortening velocity and muscle power output, but differences in power output were not observed. It is possible the newly acquired muscle length was not tested in a manner related to the training (i.e. task specificity), and/or the neuromuscular coordination had not developed yet to take advantage of the longer fascicles. All participants completed the training safely; no injuries were reported.

The inclusion of a recreationally trained group of TD young adults served as a reference point to the “potential” a muscle has in young adults without a neurological disorder. Our results highlight the compromised isometric and velocity dependent force generation capability for young adults with CP, with peak isometric force approximately 36% in CP relative to TD. These findings are supported by significant reduction in triceps surae muscle moment arm (-6.5mm), which would result in a decreased mechanical advantage of the triceps surae in producing force during walking/running, as well as reduced muscle excursion or muscle fascicle shortening for a given ankle joint rotation. A reduced MG percentage fascicle shortening was observed in CP (-34%) compared to TD (48%), likely affecting where the plantar flexor muscle operates on the muscle force-length and force-velocity curves. The reduced muscle activity of the LG (-68% EMG RMS) in comparison to TD, along with the presence of co-activation / exaggerated antagonist activity from the dorsiflexor muscle group, likely contributes to the observed muscle
weakness. It is apparent that the substantial differences between TD and CP still exist even after 10 weeks of HVT training in young adults with CP.

4.5.1 Force-length relationships

Force-length relationships have been used in the literature as an indicator for longitudinal muscle growth (Butterfield and Herzog, 2005; Proske and Morgan, 2001). A rightward shift in peak force to a greater fascicle length, is indicative of an increase in muscle fascicle length. A leftward shift would be in the direction of shorter muscle lengths, thereby allowing the muscle to produce greater force at shorter lengths. It would be expected that with an increase in fascicle length, there would be an addition of sarcomeres in series. Following the 10-week HVT intervention, resting fascicle length increased by ~9% at a common ankle angle of 105° in the CP-T group, but it doesn’t appear that the active fascicle force-length relationship had an optimal shift in fascicle length upward and to the right (Figure 4-3). The relationship does appear to have shifted upward and to the left, resulting in an increased force at shorter fascicle lengths.

Considering whole muscle-length tension relationships are accurately modelled as scaled sarcomeres, it’s possible to relate this relationship to the sarcomere-length tension relationship (Gregory et al., 2007). Sarcomeres that operate on the ascending and plateau region of the relationship during contraction are considered to be at optimal length, as force capability is near maximal or maximal. Sarcomeres operating on the descending limb are likely a result of sarcomeres operating at longer lengths, resulting in reduced muscle fascicle shortening and force output. This non-optimal relationship was apparent in 3 of the CP subjects prior to training, who were interestingly, of the 4 subjects who were identified as sedentary in the pre-assessment questionnaires. 2 of the 3 subjects participated in the training, and at the end of training, all 8
participants were able to produce 83 to 100% of peak active force at the longest fascicle lengths. This would indicate a less optimal relationship prior to training (reduced muscle force due to long sarcomere lengths), to an optimal shift in their relationship indicative of an increase in serial sarcomere number. This shift can happen without a true increase in fascicle length, resulting in more individual sarcomeres within a given fascicle to operate at more optimal lengths to produce force.

It’s also possible that the training adaptations may have resulted in other adaptations in the whole MTU (not just at the sarcomere or fascicle level). Because the tendon is in series with the muscle, after training, an increase in fascicle length may be compensated by a shorter tendon (or tendon/aponeurosis) (Proske and Morgan, 2001). Considering active fascicle length did not change in the CP-T group, the longer tendon must have stretched more during contraction, resulting from a change in Achilles tendon compliance (Sharifnezhad et al., 2014) and/or more force generated during contraction.

4.5.2 Power-relationships; Hill model, linear regressions

Peak power and peak velocity can be determined with estimation by fitting moment-velocity data to the Hill equation (Hill, 1938), or a linear regression analysis. The velocity at which peak power occurs is termed optimal velocity ($v_{opt}$) and has been determined using moment-angular velocity data for both linear regression and fitting to the Hill equation (MacIntosh et al., 1993; Tihanyi et al., 1982). The Hill equation demonstrates a hyperbolic relationship between shortening velocity and load, first used by A.V. Hill over seven decades ago. The Hill equation is still used quite predominantly in the literature, but lacks precision at
high and low velocities, where mid-range of the hyperbola appears to fit force-velocity data well (Piazzesi et al., 2007). This study used a linear regression analysis of the force-velocity relationship to determine peak velocity and peak power because a true indication of the curvature of the force-length relationship was not available for an appropriate data fit with the Hill equation. As only a restricted velocity range of the full range of angular velocities were tested, this study calculated the power-velocity relationship with a linear regression equation of the moment-angular velocity data. A measured peak isometric moment value (where velocity is zero) was measured, therefore the measured value was used over the estimated peak moment to calculate the slope of the relationship and estimate peak velocity.

4.5.3 Fascicle length in CP

Literature is scarce on fascicle length in young adults with CP, with a majority of the research on children. A literature search revealed resting MG fascicle lengths in CP children average approximately 34.5mm (at 10-20° of plantar flexion) across 3 studies (Gao et al., 2011; Kalkman et al., 2018; Kruse et al., 2018). A study by Frisk et al. (2019) analyzed MG muscle fascicle length in ambulant adults with CP (~36 years), finding resting fascicle lengths from dorsiflexion to plantarflexion decreased from 47.8mm to 26.6mm, which, considering the use of a younger CP population, is similar to our study (43.9mm to 27.8 mm). Barber et al. (2011) recruited young adults (18± 2years, 59±3 kg) and found active MG fascicle lengths at maximal dorsiflexion in CP (27 ± 3 mm) to be approximately 17% shorter than TD (~38 ± 2 mm). Our data resulted in a 32% shorter active fascicle length at maximum dorsiflexion for CP (32.6± 9.0 mm) in comparison to TD (47.8 ± 11.4mm). Greater differences observed in active fascicle length between CP and TD may be due to the inclusion of a more active group of TD participants.
than Barber et al. 2011. The reduced percentage shortening likely contributes to the reduced muscle force, and may be a result of increased passive stiffness as a result of longer sarcomere lengths (Leonard et al., 2019; Mathewson et al., 2014; Smith et al., 2011) and/or increased connective tissue and collagen deposition (De Bruin et al., 2014; Smith et al., 2011).

4.5.4 Training adaptations with HVT in TD and CP

Research in TD (Blazevich et al., 2003), older adults (McKinnon et al., 2017) and stroke patients (Patten et al., 2013) have shown improvements in muscle power output after resistance training with a focus on speed of movement. Blazevich et al. (2003) attributed the improvements in sprint and jump performance of their subjects to muscle architectural changes, specifically a decreased fascicle angle and increased vastus lateralis muscle fascicle length. Fascicle length plays a significant role in determining maximum shortening velocity of muscle, and sprinters may have training-specific increases in fascicle length in response to high-velocity training (Abe et al., 2000). Abe et al. (2000) confirmed longer fascicle lengths observed in the leg muscles of sprinters appear to favor shortening velocity as required for shorter sprint times. The authors found elite 100m sprinters had longer MG muscle fascicle lengths (66.4 ± 13.2 mm) measured with ultrasound imaging, than control (56.9 ± 7.5mm) and distance runners (53.6 ± 7.2mm). The fascicle lengths for elite sprinters would be expected be higher (at a similar ankle angle) than the fascicle lengths observed from the TD recreationally active track athletes (61.9 ± 9.4mm) recruited in this study. It is also apparent that the fascicle lengths for the control group in Abe et al. (2000) are much longer than found in literature for CP subjects.

Available research for training protocols emphasizing speed of movement in CP is limited, with only two studies we are aware of that emphasized training with an emphasis on
speed of movement in CP (Kirk et al., 2016; Moreau et al., 2013). Both of these studies included high functioning young adults (GMFCS level I-III) making it comparable to our study, but neither reached sport specific (true high) velocities. Kirk et al. (2016) developed a 12-week heavy resistance training protocol (36 session) which focused on “explosive” execution during 4 isolated lower body exercises on machines (toe raise, calf raises, leg press, and hamstring curls). Loads were determined by a 1-repetition maximum (RM) test in the pre-test, and loads were progressed during the training from a 12-6RM and set-by-set adjustments to load were made to ensure the last repetition was contraction to failure. They hypothesized that the training would positively impact the neural activation and mechanical properties of muscle and connective tissue. This group did not measure muscle volume, muscle fascicle length, nor reveal velocity of movement or change in neural activation. They did find rate of force development (0-30 and 0-50ms) and active dorsiflexion ROM improved, but the authors were unable to show any improvements in plantar flexion push-off velocity during walking (180 to 220°.s⁻¹), passive or reflex-mediated stiffness, or in any of the walking speed and endurance tests (6-minute walk test, Timed Up and Go).

Moreau et al (2013) developed an 8-week (24 session) training protocol using a Biodex dynamometer to complete 6 sets of 5 knee extensions, each at an increasing velocity (30 to 120°.s⁻¹). For the 9 CP subjects, the authors found a statistically significant increase in rectus femoris fascicle length (p=0.012), angular velocity (p< 0.001), peak power (p=0.023), and faster walking speeds (p=0.03). This study only reached 90 to 120°.s⁻¹ for their last two sets by (on average) their 12th and 16th session, respectively. Unfortunately, these studies along with other training interventions for people with CP appear to elicit very slow (<30°.s⁻¹) to slow-moderate
(120°·s\(^{-1}\) to 180°·s\(^{-1}\)) training speeds, where high velocity training would likely be those above 400°·s\(^{-1}\). To train at true high velocity speeds, average knee joint angular velocities need to reach sport or task-specific velocities, like whole movement of jumping (up to 225°·s\(^{-1}\)), running (190 to 230°·s\(^{-1}\)) and sprinting (~320°·s\(^{-1}\))(Sharifnezhad et al., 2014). Peak values of knee angular velocities can reach much higher (500-600°·s\(^{-1}\)) than these values but are only sustained for milliseconds of movement.

4.6 Conclusion

Although substantial differences between TD and CP still existed after training, the results of this study support the use of HVT in CP to obtain improvements to muscle fascicle length and/or increased sarcomeres in series. Our results show that high-functioning individuals with CP can safely complete exercises with maximal exertion at high velocities. Considering angular joint velocities of the ankle during walking can reach up to 220°·s\(^{-1}\) in CP, the angular velocities during the training drills in this study likely exceed those used in any previous training intervention (30-120°·s\(^{-1}\)). There may be additional benefit with testing that includes specific movements of interest to take advantage of neuromuscular coordination improvements in this population.
Chapter Five: Summary, limitations and future direction

5.1 Summary of results

Our main finding was that following a 10-week HVT intervention involving sprinting, jumping, and agility drills, resting fascicle length increased by ~9% at a common ankle angle of 105° across CP participants. This increase in fascicle length was not accompanied by an optimal rightward shift in the active force-length relationships (similar or higher forces at longer fascicle lengths), and no differences in peak power output or peak velocity were observed. Our results highlight the compromised isometric and velocity dependent force generation capability for young adults with CP, with peak isometric force approximately 36% of TD and peak velocity approximately 48% of TD. These findings are supported by significant reduction in triceps surae muscle moment arm (-6.5mm), percentage muscle fascicle shortening during an MVC (34% vs. 48% in TD), and muscle activation of the LG (-68%).

5.2 Limitations

The tendon travel method and visual method MA values for the TD group were not statistically different, but for the CP group there was a substantial difference in methods. This could be attributable to large passive forces and/or boney deformities that are prevalent in CP. Bony deformities may result in altered foot mechanics and/or alignment of the malleoli which may have resulted in inaccurate estimation of the transmalleolar midpoint in our study. Although very little variation was seen between first and second measurements, where any differences can be attributed to with-in tester measurement error, minimal research exist on CP MA to validate our results.
As a true indication of the curvature of the force-velocity relationship is needed for an appropriate data fit with the Hill equation, and only a restricted range of velocities were tested, peak velocity and peak power were determined with a linear regression analysis. The use of linear regression can sometimes overestimate force-velocity values in comparison to the Hill equation, but the Hill equation has also been found to lack precision at high and low velocities. The mean slope using linear regression for the force-velocity data had a good fit ($r^2 = 0.822$), where we feel the mid-range values (where peak power is estimated to occur) are represented well.

It is not possible to assume this training intervention only consisted of high-velocity shortening contractions, as the subjects were instructed to control their jump landings and decelerate after their sprints to minimize the chance of injury. Lengthening contractions under load have also been found to broaden the force-length relationship or the working range of muscle length in CP (Reid et al., 2010). The use of a dynamometer like the Biodex for training would remove this uncertainty and allow direct measurement of joint angular velocities (which were not measured in this study during training). We did not measure, but make the assumption that the joint angular velocities during the running, sprinting and jumping in this training program were truly high velocity, and above the maximal plantar flexion push-off velocities found in CP during walking (180 to 220°·s⁻¹)(Kirk et al., 2016). This is based on the knowledge of individuals without a neurological disorder reaching ankle joint velocities of 400-600°·s⁻¹ during running (Heidenfelder et al., 2008), 800°·s⁻¹ during the push-off phase of a jump (Bobbert et al., 1987a, 1987b), and approximately 1500°·s⁻¹ during a sprint (Bezodis et al., 2008). Using exercises that could be implemented in the daily training environment was a priority for researchers of this study to be able to make realistic inferences on high-velocity training and
changes to muscle architecture. Results were likely confounded due to responders and non-responders to training within the group, as well as different activity level/training experience of those entering the training intervention. Also, as the training intervention was not based in the lab, subjects were not given the opportunity to practice on the Biodex to be able to give a true representation of change in isokinetic performance measures.

5.3 Future direction

Current methods in estimating muscle forces in CP (2D, 3D and tendon travel) should be used with caution, as this population exhibits high passive resistive forces affecting tendon lengthening, as well as altered axis of rotation due to altered foot mechanics and/or alignment of the malleoli. Future research is warranted in differences in MA in CP, as research is limited and inconsistent. Although substantial differences between TD and CP still existed after training, the results of this study support the use of HVT in CP to obtain improvements to muscle fascicle length and/or increased sarcomeres in series. Our results show that high-functioning individuals with CP can safely complete exercises with maximal exertion at high velocities. Future research on HVT training in CP should include testing and training sport-specific movements of interest to have the greatest effect on neuromuscular coordination and assessment in this population.
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Appendix A: GMFCS Predictor

GMFCS E & R between 12\textsuperscript{th} and 18\textsuperscript{th} birthday: Descriptors and illustrations

GMFCS Level I
Youth walk at home, school, outdoors and in the community. Youth are able to climb curbs and stairs without physical assistance or a railing. They perform gross motor skills such as running and jumping but speed, balance and coordination are limited.

GMFCS Level II
Youth walk in most settings but environmental factors and personal choice influence mobility choices. At school or work they may require a hand held mobility device for safety and climb stairs holding onto a railing. Outdoors and in the community youth may use wheeled mobility when traveling long distances.

GMFCS Level III
Youth are capable of walking using a hand-held mobility device. Youth may climb stairs holding onto a railing with supervision or assistance. At school they may self-propel a manual wheelchair or use powered mobility. Outdoors and in the community youth are transported in a wheelchair or use powered mobility.

GMFCS Level IV
Youth use wheeled mobility in most settings. Physical assistance of 1-2 people is required for transfers. Indoors, youth may walk short distances with physical assistance, use wheeled mobility or a body support walker when positioned. They may operate a powered chair, otherwise are transported in a manual wheelchair.

GMFCS Level V
Youth are transported in a manual wheelchair in all settings. Youth are limited in their ability to maintain antigravity head and trunk postures and control leg and arm movements. Self mobility is severely limited, even with the use of assistive technology.
## Appendix B: High velocity training program

<table>
<thead>
<tr>
<th>Warm Up</th>
<th>10min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smin Cycle Ergometer</td>
<td></td>
</tr>
<tr>
<td>Heel Walk / Toe Walk</td>
<td>15&quot; Each x 2</td>
</tr>
<tr>
<td>Hamstring Sweep</td>
<td>5E</td>
</tr>
<tr>
<td>Carioca Walk / Skip</td>
<td>30&quot; Each / 20m</td>
</tr>
<tr>
<td>Lunge &amp; Reach</td>
<td>5E</td>
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<td></td>
<td></td>
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</tbody>
</table>

*Day 2 perform jumps before sprints

### Warm up

<table>
<thead>
<tr>
<th>Week 1-2</th>
<th>Week 3-6</th>
<th>Week 7-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>10minute warm up</td>
<td>10minute warm up</td>
<td>10minute warm up</td>
</tr>
<tr>
<td>3x (Accel 5m - Decel 5m x5)</td>
<td>30-50m Accel to Easy / Medium</td>
<td>30-50m Accel to Easy / Medium</td>
</tr>
<tr>
<td>3x 5m Agility T Drill</td>
<td>30m(6sec) Accel to Fast x 2</td>
<td>30mAccel to Fast x 2</td>
</tr>
<tr>
<td>Circuit x 4</td>
<td>20m (3-4 sec) Stride Easy / Medium / (Fast x3)</td>
<td>20m Stride Easy / Medium / (Fast x5)</td>
</tr>
<tr>
<td>A. Agility Ladder drills</td>
<td>10m Sprint Starts x 4</td>
<td>10m Flying 10m Sprints x 6</td>
</tr>
<tr>
<td>B. CMI x2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. Medball Vertical Throws x3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>If time: Partner Mirror Drill 10 sec x3</td>
<td>If time: 15m sprint races x 3</td>
<td>If time: 15m sprint races x 3</td>
</tr>
<tr>
<td></td>
<td>OR Partner Mirror Drill</td>
<td>OR Partner Mirror Drill</td>
</tr>
</tbody>
</table>

OR Partner Mirror Drill
Appendix C: Permissions

Re: Permission to use Images from Pingel et al. 2017
Re: Permission for manuscripts in thesis

On Jun 12, 2019, at 3:33 PM, Tessa Gallinger <tessa.gallinger@ucalgary.ca> wrote:

Hi Jared and Brian,

As you are co-authors on both of my manuscripts, do you give permission to include these manuscripts in my thesis:

1. Mechanisms of reduced plantarflexor function in Cerebral Palsy; smaller triceps surae moment arm and reduced muscle force.
2. Muscle length adaptations to sport specific velocity training in young adults with cerebral palsy.

Thanks so much!
Cheers,
Tessa

Tessa Gallinger  |  Graduate Student
Human Performance Laboratory
Faculty of Kinesiology, University of Calgary
2500 University Dr. NW

On Jun 12, 2019, at 3:38 PM, Jared Fletcher <jfletcher@mtroyal.ca> wrote:

Yes!

--
Jared R. Fletcher, PhD
Assistant Professor
Department of Health and Physical Education
Mount Royal University
Calgary, AB

On Jun 12, 2019, at 3:55 PM, Brian MacIntosh <bmacinto@ucalgary.ca> wrote:

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Brian