USING THE LATENCY CORRECTION METHOD TO CALCULATE THE TONIC STRETCH REFLEX IN SPASTIC CEREBRAL PALSY PRE AND POST EXERCISE INTERVENTION: A CASE STUDY

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ABSTRACT
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ABSTRACT

Cerebral palsy (CP) is the most common childhood disability, caused by brain lesions during early development, often leading to motor-related symptoms and spasticity. Exercise interventions focusing on strength, gait, flexibility, and balance are valuable for managing CP. Tailoring exercise programs to individual needs may maximise outcomes. This case study was a smaller project related to the EXECP project. The case study aimed to evaluate changes in the tonic stretch reflex threshold (TSRT) following a 12-week exercise intervention for stretch hyper-reflexia in spastic cerebral palsy (SCP), aiming to provide an insight into the potential benefits of exercise on the TSRT in SCP. One participant with spastic cerebral palsy was selected (N=1) for the case study and performed passive muscle stretching of the soleus and medial gastrocnemius using a motor-driven dynamometer (Neuromuscular Research Center, University of Jyväskylä, Finland). The dynamometer induced passive ankle dorsiflexion from 20° of plantarflexion to 0° at four angular velocities (55, 110, 210, and 291°/s) as part of the EXECP study. The EMG data was then provided for this case study, allowing the TSRT to be calculated and assessed at four time points (PRE1, PRE2, POST1, POST2) and four velocities (55°/s, 110°/s, 200°/s, 291°/s) for the medial gastrocnemius and soleus. This case study used the EMG burst activity data from the EXECP intervention, and TSRTcorrected was calculated in Spike2 from Cambridge Electronics Design, Cambridge, UK . An individualized Hoffman-reflex (H-reflex) latency correction method was used, based on suggestions from Valadão et al. (2022) The h-reflex EMG data also came from the EXECP intervention. The case study analysed a portion of the h-reflex EMG data using MATLAB (R2023a, The MathWorks Inc, Natick, United States). The primary research questions are 1) is the TSRT corrected velocity dependent in this case study subject for the soleus and medial gastrocnemius? 2) how does TSRT corrected for the soleus and medial gastrocnemius muscles vary across different stretch velocities (55, 110, 200, and 291°/s) and 3) pre vs post exercise intervention. The soleus showed higher variability and sensitivity to velocity changes compared to the medial gastrocnemius, suggesting its role in adaptive postural control. After exercise, reflex thresholds in the soleus decreased and variability was reduced, indicating modulation of reflex activation. In contrast, the medial gastrocnemius showed stable, higher reflex thresholds, indicating its role in high-speed dynamic movements. The study reveals the distinct behaviours of the soleus and medial gastrocnemius, highlighting their specialised functional roles and neuromechanical properties. These findings suggest that the soleus is specialized for postural stability, while the medial gastrocnemius is suited for dynamic movements, informing targeted rehabilitation approaches. The medial gastrocnemius shows stable reflex thresholds, suited for dynamic, high-speed tasks, while the soleus exhibits more variability, reflecting its role in postural stability and controlled movements. These findings highlight the distinct roles of these muscles and suggest targeted interventions could enhance motor control in individuals with cerebral palsy, especially those with spasticity. The results must be interpreted cautiously, and no generalisations should be made as this was a case study.

Key words: cerebral palsy, hyper-resistance, stretch reflex threshold, stretch hyper-reflexia, assessment, upper motor neuron syndrome, neuromuscular function.

ABBREVIATIONS

CP- cerebral palsy

MDD- motor development disorder

SCP- spastic cerebral palsy

SR- stretch reflex

SRT- stretch reflex threshold

TSRT - tonic stretch reflex threshold

TSRTcorrected - latency corrected tonic stretch reflex threshold

MTL- motor tract lesion

CNS- central nervous system

PNS- peripheral nervous system

MS- multiple sclerosis

iSCI- incomplete spinal cord injury

SCI- spinal cord injury

EMG- electromyography

RMS- root mean square

LMNs- lower motor neurons

UMNS- upper motor neuron syndrome

UMN- upper motor neurons

GMFCS- gross motor function classification system

H-reflex- Hoffman reflex

TD- typically developing individuals

PCSA- physiological cross-sectional area

MMS- Montreal spasticity measurement

SOL- soleus

MG- medial gastrocnemius

CONTENTS

1 INTRODUCTION

Cerebral palsy (CP) is the most common childhood motor development disorder (MDD), caused by abnormal development or lasting non-progressive injury to the developing infant brain. It is a permanent, non-progressive condition affecting movement and posture (Graham et al., 2016a). Although classified as non-progressive, some symptoms may appear to worsen over time. CP arises during the prenatal, perinatal, or postnatal periods, typically resulting from a brain or motor lesion most often affecting the motor cortex, basal ganglia, or cerebellum (Bar-On et al., 2018). Globally, CP occurs at rates ranging from 1.5 to 3 per 1,000 live births (Graham et al., 2016a; Johnson, 2002). Increased survival rates among premature infants may have slightly elevated CP prevalence, especially in developed nations. Preterm birth remains the most significant risk factor for CP, with risk decreasing as gestational age increases. The likelihood of CP rises moderately at 38 weeks, while infants born before 28 weeks face up to 50 times the risk of those born at full term (Kuban et al., 2009). Another major risk factor is white matter damage, which can be detected via ultrasonography or magnetic resonance imaging (MRI) (Graham et al., 2016b; Kuban et al., 2009).

Features of CP is its clinical and functional presentation in early development, often involving persistent movement disorders. Motor impairments primarily result from damage to upper motor neurons, disrupting motor planning, execution, and feedback loop functions (Graham et al., 2016b). This frequently manifests as upper motor neuron syndrome (UMNS), with spasticity or stretch hyper-reflexia as key features. Spasticity is characterized by a velocity-dependent increase in muscle tone due to hyperactive stretch reflexes, caused by imbalances in excitatory and inhibitory signals within the spinal cord and brain. These abnormalities exaggerate muscle responses to stretch, significantly impairing voluntary movement and functional abilities. Beyond motor impairments, CP is often associated with challenges in cognition, communication, sensation, perception, epilepsy, and musculoskeletal health (Graham et al., 2016a). Key clinical goals for individuals with CP include maximizing functional activities and participation, while minimizing the impact of associated conditions such as epilepsy, feeding difficulties, hip dislocation, and scoliosis (Graham et al., 2016b). Table 1 below gives a summary of cerebral palsy and its main pathologies and developmental changes.

CP encompasses a wide range of complex aetiologies and pathophysiologies. Despite advances from epidemiologic, neuroimaging, animal model, and post-mortem studies, the mechanisms, features, and phenotypic variations of CP remain only partly understood. This limits comprehensive prevention strategies. Early clinical descriptions linked CP to premature birth and neonatal asphyxia, which were once thought to be definitive causes. However, recent research suggests these factors are reflective of broader developmental influences (Graham et al., 2016b). This case study aimed to examine the calculation of the stretch reflex threshold in an individual with spastic CP (SCP). It employed the stretch reflex threshold corrected method (TSRTcorrected) to calculate the stretch reflex threshold, as recently proposed by Valadão et al. (2022). The investigation focused on the TSRTcorrected in the soleus and gastrocnemius muscles of the triceps surae, which are critical for postural control and gait.

Table 1: A summary of cerebral palsy (Lundy-Ekman & Weyer, 2022).

Condition	Cerebral palsy
Pathology	Developmental abnormalities
Aetiology	Abnormal development in the pre-, peri or post-natal period, metabolic anomalies, immune system syndromes, coagulation syndromes, infection, trauma, hypoxia, central nervous system damage before second birthday
Speed of onset	Unknown
Signs and symptoms	
Consciousness	Normal
Cognition, language, and memory	Often associated with intellectual disability, language challenges, although not all cases. Some individuals have above average IQ.
Sensory	75% of cases present with pain, or impairments in somatosensation.
Autonomic	25% of individuals experience incontinence.
Motor	33% unable to walk, hypotonic individuals show low muscle tone, impaired movement capacity. Spastic type, paresis, muscle

	architectural changes, and increased resistance in muscle. Dyskinetic type may show slow, writhing, jerky movements, or sustained involuntary postures. Ataxic type, shows incoordination, shaky during voluntary activities.
Cranial nerves	Indirectly affected, due to abnormal neural input, causing impairment in output of cranial nerves.
Vision	Eye movement and vision often impaired.
Associated disorders	Seizure affects approximately 25% of individuals.
Region affected	Brain, and some spinal cord inclusion.
Demographics	Developing nervous system altered due to motor lesion on cerebral hemisphere.
Prevalence	2-3 per 1000 live births
Prognosis	Neural abnormality is constant, but functional limits may alter as individual grows and over the life span.

2 LITERATURE REVIEW

2.1 Lesion mechanisms and pathophysiology of cerebral palsy

Brain lesions are a primary cause of cerebral palsy (CP), with up to 90% of cases resulting from the destruction of healthy brain tissue rather than abnormal brain development (Graham et al., 2016a). These lesions are most associated with hypoxic or ischemic injury affecting the cerebral cortex, white matter, basal ganglia, or cerebellum. Such injuries stem from inadequate oxygenation or blood flow to the brain, triggering cellular energy depletion, excitotoxicity, oxidative stress, and chronic inflammation (Mukherjee & Chakravarty, 2010). The timing of the injury during brain maturation plays a critical role in determining the lesion's type, location, and severity (Pagnozzi et al., 2017)

Early in brain development, when blood vessels have limited capacity for dilation, ischemic injury can be particularly severe. Ischemic events during the second or third trimester often result in diffuse brain damage, characterized by liquefaction necrosis. This process transforms affected tissue into a viscous liquid mass, leading to the formation of porencephalic cysts (Raybaud, 1983). Imaging studies have highlighted variability in lesion sites, which can include the cerebral cortex, white matter, basal ganglia, or cerebellum (Bax et al., 2006). Clinical presentations of CP are influenced by lesion timing, location, severity, and the body's systemic response to the injury (Bax et al., 2006; Kuban et al., 2009). Most cases, however, involve hypoxic or ischemic destruction of the cerebral cortex (Graham et al., 2016a). Studies by Pagnozzi et al. (2017) have identified various types of injury contributing to unilateral CP, as illustrated in Figure 1. A) Cortical malformations with white matter atrophy and ventricular expansion, b) Lesions near the lateral ventricles due to white matter death, c) White matter loss with secondary ventricular enlargement from infarction, d) Excess gyri formation, e)Scar tissue in the posterior limb of the internal capsule, f) Periventricular cystic lesions causing minor ventricular expansion. Despite differences in injury type, all forms of CP involve atrophy of the cerebral cortex alongside damage to subcortical structures and axons near the lateral ventricles.

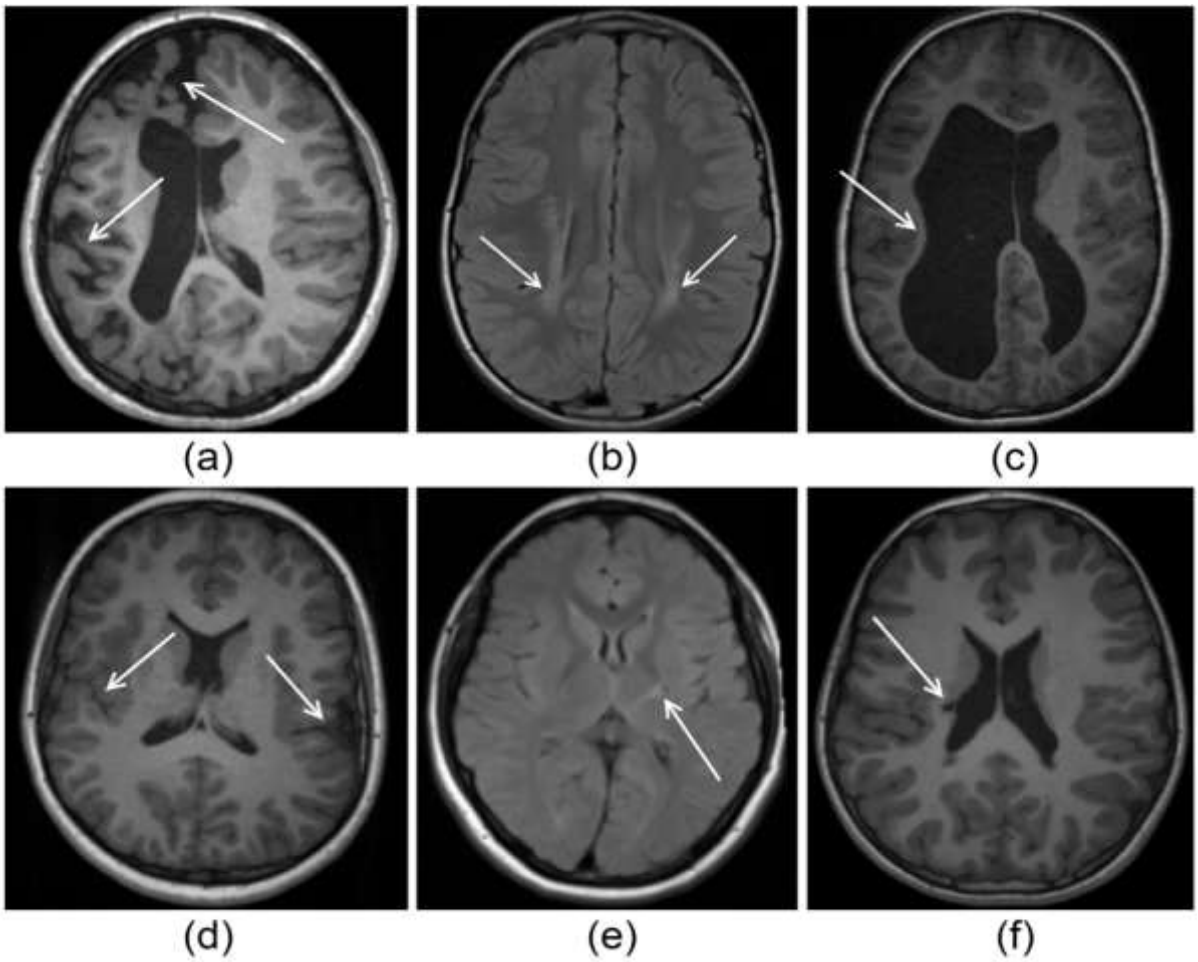

FIGURE 1. Structural magnetic resonance images of different sites and types of brain injury related to cerebral palsy from (Pagnozzi et al., 2017)

These lesions disrupt descending signals from the brain to lower motor neurons, leading to abnormal synaptic connections and irregular spinal motor circuit development (Pagnozzi et al., 2017). As a result, individuals experience difficulties with movement control and coordination (Graham et al., 2016a; Lundy-Ekman & Weyer, 2022). Upper motor neuron lesions, common in spastic diplegia, mixed CP, and hypotonic CP, often involve further abnormalities in the basal ganglia and thalamus (Graham et al., 2016a; Lundy-Ekman & Weyer, 2022).

CP-related injuries have been categorized into three broad groups based on aetiology (Pagnozzi et al., 2017). Cortical abnormalities caused by disruptions during the first or second trimester.

Periventricular white matter injuries in the early third trimester, sometimes with secondary ventricular enlargement due to tissue loss. Cortical and grey matter injuries in the late third trimester, leading to grey matter lesions. These classifications have been used to examine the timing and prevalence of injury in children with CP. However, they remain qualitative and do not account for lesion location or severity, which can be better identified through structural MRI (Pagnozzi et al., 2017). Initially, CP was attributed to premature birth and difficult labours, often resulting in neonatal asphyxia. While these factors are not definitive causes, they reflect broader developmental influences (Graham et al., 2016b). Preterm birth remains the most significant risk factor, with risk decreasing as gestational age increases. Infants born before 28 weeks have a 50-fold higher risk of developing CP compared to those born full-term, while moderate increases in risk are noted at 38 weeks (Kuban et al., 2009). See figure 2 below for the relationship between gestational age and frequency of cerebral palsy (Graham et al., 2016a).

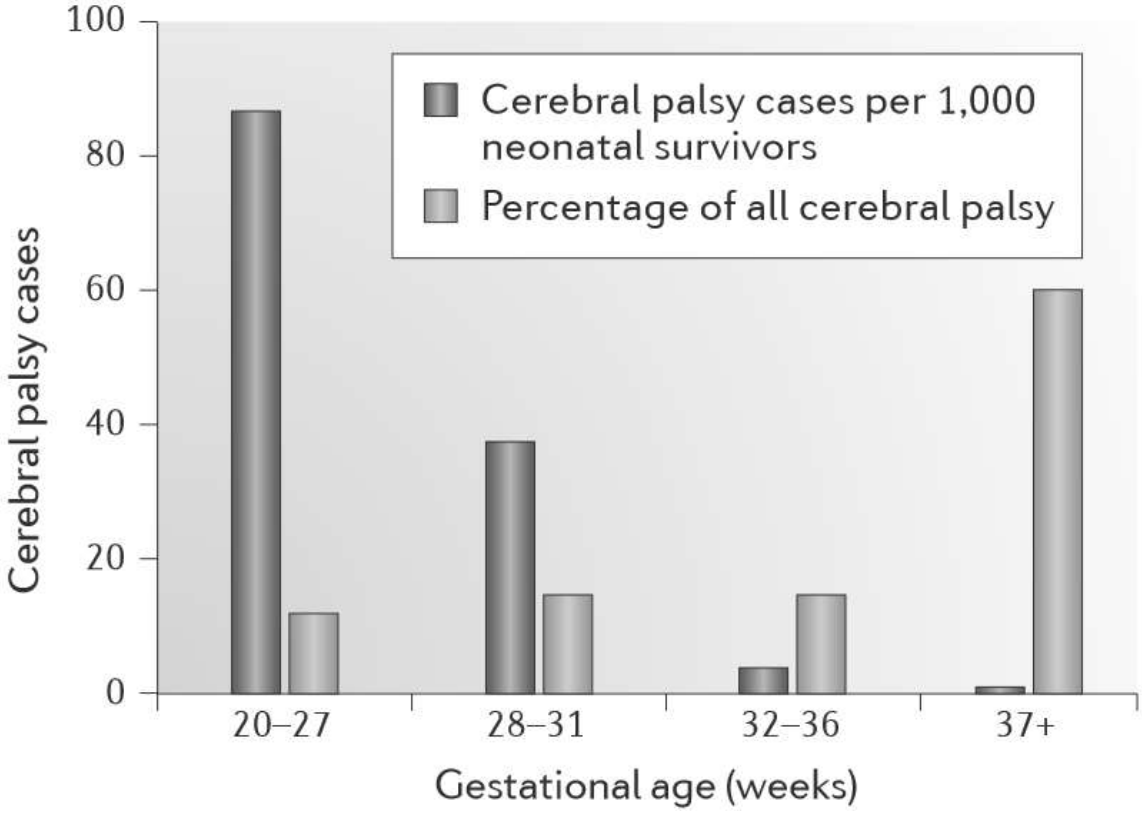

FIGURE 2. Relationship between gestational age and frequency of cerebral palsy from (Graham et al., 2016a).

White matter damage, detectable via ultrasonography or MRI, is another key risk factor (Graham et al., 2016b; Kuban et al., 2009). Perinatal anomalies such as meconium passage, neonatal seizures, and respiratory issues are also strongly associated with CP (McIntyre et al., 2013). Additionally, congenital defects, particularly cardiovascular and skeletal abnormalities, are more prevalent in individuals with CP (McIntyre et al., 2013). The complex developmental nature of CP makes it difficult to quantify cases linked to birth asphyxia alone. Multiple perinatal factors, including chorioamnionitis, perinatal inflammation, transient hypothyroxinaemia, and hypocapnoea have been implicated. These factors may contribute to cerebral vasoconstriction and subsequent brain injury (Collins et al., 2001; Kuban et al., 2015; Reuss et al., 1996). Altered intra-uterine growth and postnatal inflammation further complicate biological processes, potentially increasing CP risk (Leviton et al., 2013). Recent interventions have shown promise in reducing CP risk. Administering magnesium sulphate to mothers in preterm labour has been associated with a 30% reduction in CP incidence among preterm infants. For full-term infants experiencing birth asphyxia, therapeutic hypothermia 72-hour period of brain-body cooling—has shown beneficial outcomes (Jacobs et al., 2013). These advances highlight the importance of comprehensive prevention and treatment approaches in mitigating CP development.

2.2 Cellular mechanisms of the brain injury and lesion

Hypoxia and ischemia are key factors in perinatal asphyxia, particularly in full-term births, which are more susceptible to oxygen shortages than pre-term newborns. These conditions primarily affect the grey matter in regions like the basal ganglia, thalamus, and cerebral cortex. The resulting brain damage correlates with specific lesions and clinical symptoms, such as spastic, dyskinetic, and mixed types of cerebral palsy (CP) (Herrera-Marschitz et al., 2011). However, Graham et al. (2016a) suggest that most CP cases are not related to asphyxia, indicating that other mechanisms may contribute to the development of CP. Cellular energy depletion occurs when ATP is exhausted due to mitochondrial failure, which disrupts ATP-dependent processes, such as Na+/K+-ATPase membrane potentials. This disruption facilitates the influx of Ca2+ through NMDA receptors, triggering a cascade of events that lead to cell death, either by necrosis or apoptosis. These processes have led to the investigation of neuroprotective substances, such as magnesium sulphate, which may block NMDA receptor activity and mitigate cell death (Graham et al., 2016a). The excess Ca2+ in the cytoplasm

promotes oxidative stress by activating Ca2+-dependent oxidases and inhibiting antioxidant processes. This results in an increase in reactive oxygen species (ROS), which further impairs mitochondrial function (Graham et al., 2016a). The accumulation of ROS exacerbates cellular damage, accelerating the rate of cell death. This effect is particularly prominent during early brain maturation in the second trimester, when the brain's scavenging systems have limited capacity to cope with oxidative stress (Jensen et al., 2003). Injuries, including hypoxia or ischemia, can lead to two forms of cell death: necrosis and apoptosis. Necrosis occurs immediately after injury, causing focal, nonspecific cell death. In contrast, apoptosis is a more prolonged process, which tends to be cell-specific and often targets pre-oligodendrocytes in the brain (Jensen et al., 2003). Necrosis and apoptosis contribute to white matter injuries in full-term births, which are typically associated with pre-term births. Necrosis is often seen as focal lesions that can develop into macroscopic cysts, such as in cystic periventricular leukomalacia, or cause gliosis in non-cystic forms (Mukherjee & Chakravarty, 2010).While, apoptosis of pre-oligodendrocytes leads to hypomyelination (Graham et al., 2016a). Cell death in the brain is mediated through both extrinsic and intrinsic signalling pathways, which activate common mitochondrial and nuclear networks, see figure 3 below for reference (Graham et al., 2016a). Extrinsic pathways are triggered by inflammatory cytokines binding to death receptors like FAS and by NMDA receptor-mediated excitotoxicity due to glutamate. In contrast, intrinsic pathways are activated by internal signals from stressed mitochondria, leading to caspase-mediated and non-caspase-mediated cell death in the nucleus (Mukherjee & Chakravarty, 2010). Under caspase-induced stress, mitochondria release cytochrome c, initiating caspase-mediated cell death. Alternatively, apoptosis-inducing factors activate non-caspase pathways that cause DNA damage, often facilitated by free radicals like nitric oxide (NO) (Graham et al., 2016a; Pagnozzi et al., 2017). The distinct pathways involved in necrosis and apoptosis present potential targets for intervention, which could help reduce cell death and prevent the progression of brain damage in conditions like CP.

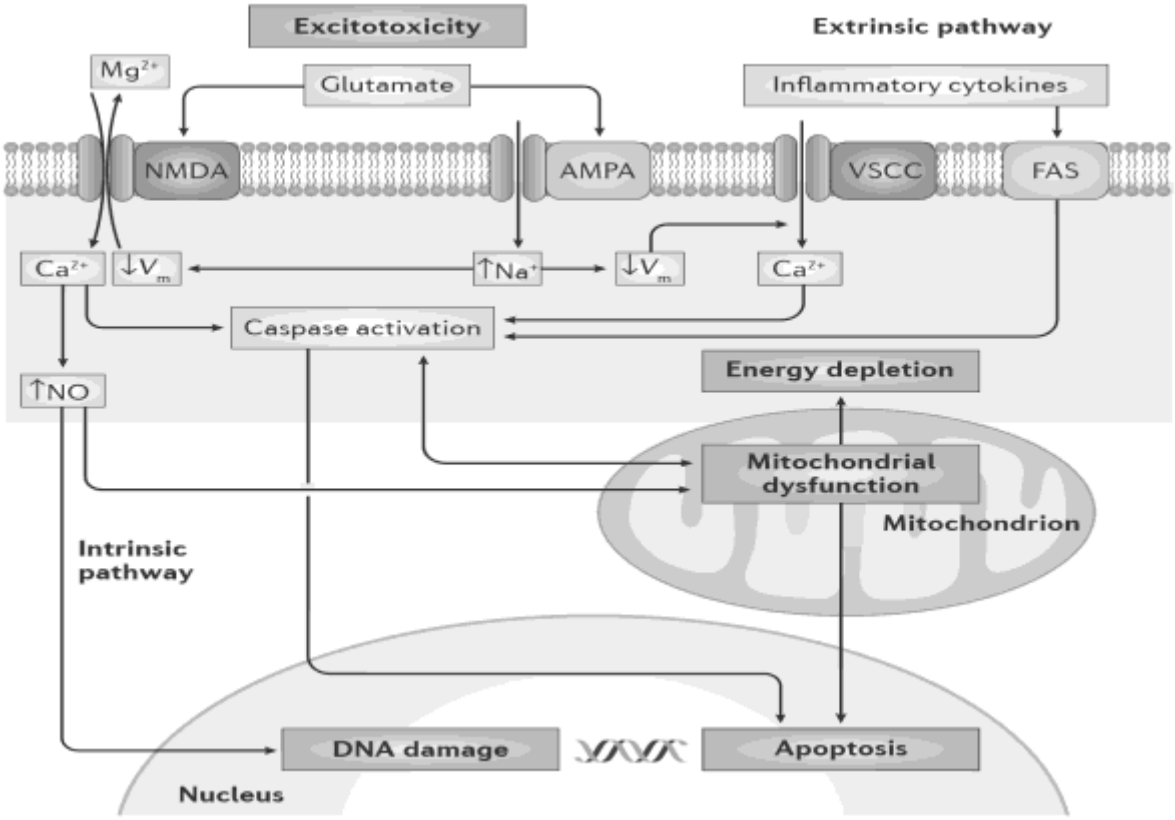

FIGURE 3. Cell death signalling pathway in brain injury related to CP from (Graham et al., 2016a).

2.3 Classification systems: functional, motor and dispersal models

Cerebral palsy (CP) encompasses a group of permanent movement and posture disorders caused by non-progressive disturbances in the developing brain. Its classification is crucial for understanding its characteristics, planning management, and predicting outcomes. CP is categorized by motor type, impairment distribution, and severity, providing a framework to describe its diverse manifestations and guide therapeutic approaches (Graham et al., 2016a). Functional capacity classifications, discussed in the following sections, delve into the types, classifications, and impairment patterns across CP populations (Lundy-Ekman & Weyer, 2022). Since CP presents uniquely in everyone, classification systems consider functional impairments, muscle tone abnormalities, and impairment distribution to ensure tailored assessment and intervention (Graham et al., 2016a; Lundy-Ekman & Weyer, 2022).

Motor type or muscle tone is based on the nature of the movement abnormality, with spastic, dyskinetic, ataxic, and mixed types being the primary categories. Spastic CP, the most common type, is characterized by increased muscle tone and stiffness, often leading to joint contractures. Dyskinetic CP involves involuntary movements, while ataxic cp is marked by difficulties with balance and coordination. Mixed CP presents a combination of these features (Graham et al., 2016b; Lundy-Ekman & Weyer, 2022). Dispersal classification focuses on the areas of the body affected. Terms such as hemiplegia, diplegia, and quadriplegia describe whether one side, the lower limbs, or all four limbs are involved. This categorization helps in understanding the extent of physical disability and planning mobility aids or interventions (Graham et al., 2016b; Lundy-Ekman & Weyer, 2022). Functional classification, often assessed using tools like the gross motor function classification system (GMFCS), evaluates the degree of functional impairment. This system ranks individuals from level I (minimal impairment) to level V (severe impairment), guiding expectations and therapeutic goals (Graham et al., 2016b; Lundy-Ekman & Weyer, 2022). By combining these classification dimensions, clinicians can comprehensively describe the condition and predict developmental trajectories. This nuanced understanding of cp highlights its complexity while emphasizing the importance of individualized care. Each classification mode will be expanded in the below sections starting with functional classification, then muscle tone and finally dispersal (Graham et al., 2016b, 2016a). Cerebral palsy is commonly classified using the gross motor function classification system (GMFCS, see figure 4below) (Graham et al., 2016a). The GMFCS presents five different levels of functional classification for individuals with cerebral palsy. Classification at Level I characterise the ability of a child to perform gross motor tasks including running and jumping, with challenges in speed, balance, and coordination of movement present (Graham et al., 2016a). Level II classification represents additional challenges with endurance capacity, balance on uneven surfaces, and lower capacity when performing gross motor tasks. Furthermore, it may require use of an aid when walking. Level III, walking in most indoor settings requires a mobility device, for longer distances a wheelchair may be required. Stairs may be climbed using handrailing under supervision. Level IV, most often requires physical assistance or powered mobility devices to move. Walking short distances at home with physical assistance and or a body support walker. Outdoors and in the community motor powered mobility devices are mandatory. Level V indicates low muscle control of head, trunk posture, and limb movement. Mobility occurs in a wheelchair in all situations (Graham et al., 2016a).

Cerebral palsy may also be categorised based on the muscle tone observed, often represented as dystonia, ataxia, or spastic. Cerebral palsy is commonly presented in five different groups, with four of the five types showing abnormal muscle tone. Muscle tone is defined as the amount of resistance to stretch in a relaxed muscle, in a healthy neuromuscular system relaxed muscle provides little resistance to passive stretch (Lundy-Ekman & Weyer, 2022). However, in cerebral palsy muscle tone is abnormal and muscle tone is presented on a scale from hypotonia to hypertonia. Hypotonia representing lower than normal resistance, while hypertonia, represents greater than normal resistance to stretch. The classification of abnormal muscle tones represents the muscle tone present (Graham et al., 2016a; Lundy-Ekman & Weyer, 2022).

Hypotonic CP, is categorised due to very low muscle tone, associated with inadequate muscle contraction required to maintain regular head and trunk postures. Individuals with hypotonic CP have little to no capacity to actively move. Spastic CP, and spasticity represents the velocity dependant component of hypertonia. This type is considered velocity dependent as there is little resistance at low stretch velocities, with increasing resistance present at higher stretch velocities. This makes muscle more stiff than normal often leading to contractures, due to abnormally short muscles and tendons. Often leading to abnormal patterns in the gait cycle, seen in toe walking or scissor gait (Meyns et al., 2016).

Furthermore, spastic CP is categorized based on the area of the body that is impacted, termed hemiplegic, tetraplegic, or diplegic. These terms will be explained in the following section. Dyskinetic CP is a term used to describe fluctuating states of muscle tone. Most seen is choreoathetoid with dystonia. Represented by three types of spontaneous movements, choreiform, athetoid or dystonic. Choreiform includes jerky, abrupt, or irregular movements, while athetoid presents as slow, or writhing movements and dystonic represents with spontaneous sustained muscle contraction. In addition, mixed type CP represents a combination of spasticity and dyskinesia. The final classification given to CP is ataxic, this type does not present with abnormal muscle tone, instead movements are uncoordinated and shaky during voluntary movement (Graham et al., 2016b; Lundy-Ekman & Weyer, 2022).

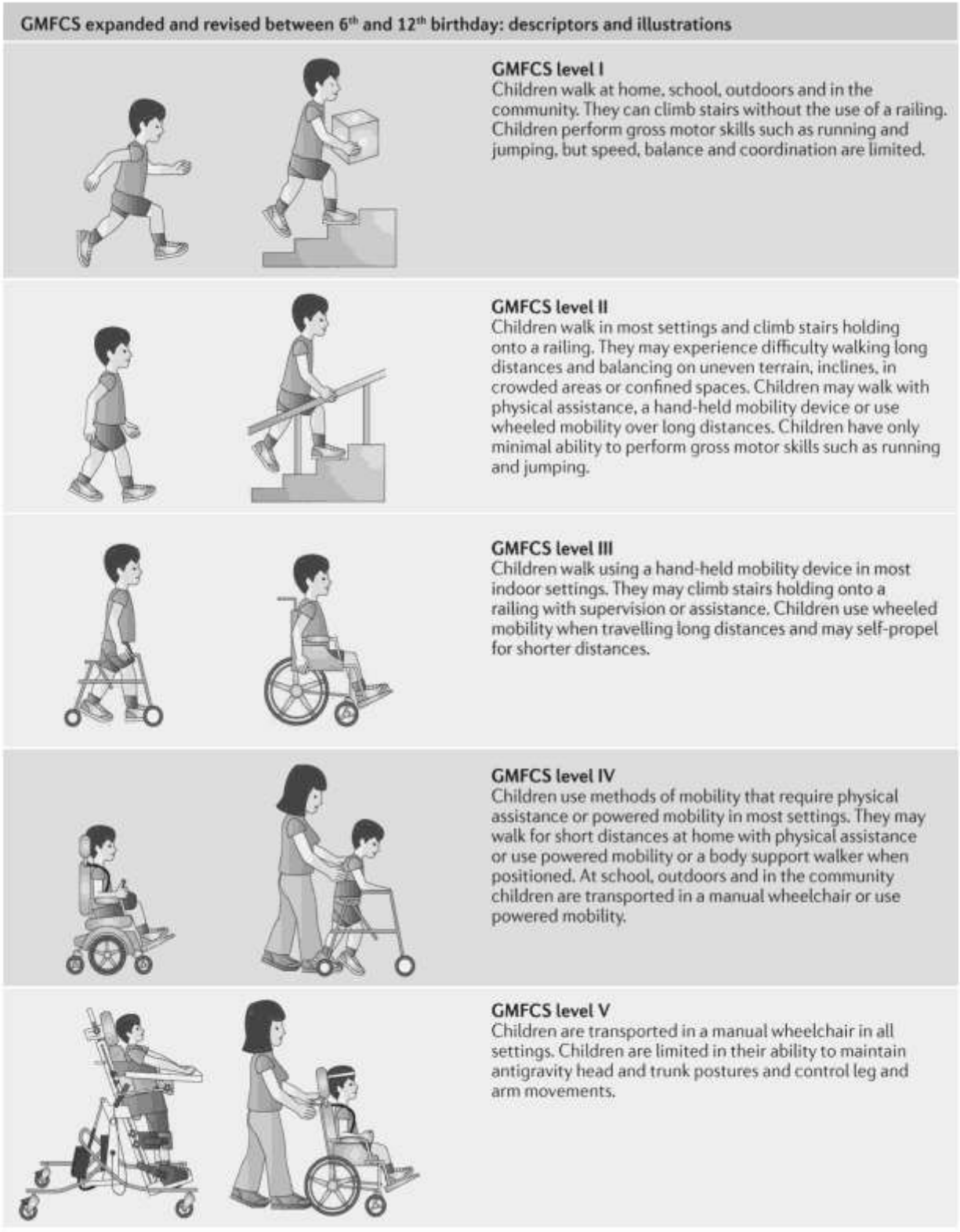

FIGURE 4. The Gross motor function classification systems (GMFCS) for children with cerebral palsy from (Graham et al., 2016a).

Classification is often presented in combination with an impairment dispersal, referred to as either unilateral or bilateral, see figure 5 below (Bar-On et al., 2018). The two impairment dispersals can further be divided, unilateral may represent monoplegia or hemiplegia. Monoplegia usually affects one limb, most frequently the lower limb. Hemiplegia usually affects one side of the body, with the upper limbs most frequently affected. While bilateral impairment presents three groups diplegia, triplegia or quadriplegia (see figure 5 below). In diplegia each limb is affected, with lower limbs more than upper limbs. Triplegia, represents unilateral upper limb impairment, combined with bilateral lower limb impairment. The lower limb is most frequently affected on the same side as the upper limb impairment. Quadriplegic involvement represents all limbs and the trunk, it may also be referred to as whole body involvement. Therefore, the levels of impairment faced by individuals with cerebral palsy are highly individualised, based on the severity and complexity of the lesion (Graham et al., 2016b; Sloot et al., 2021).

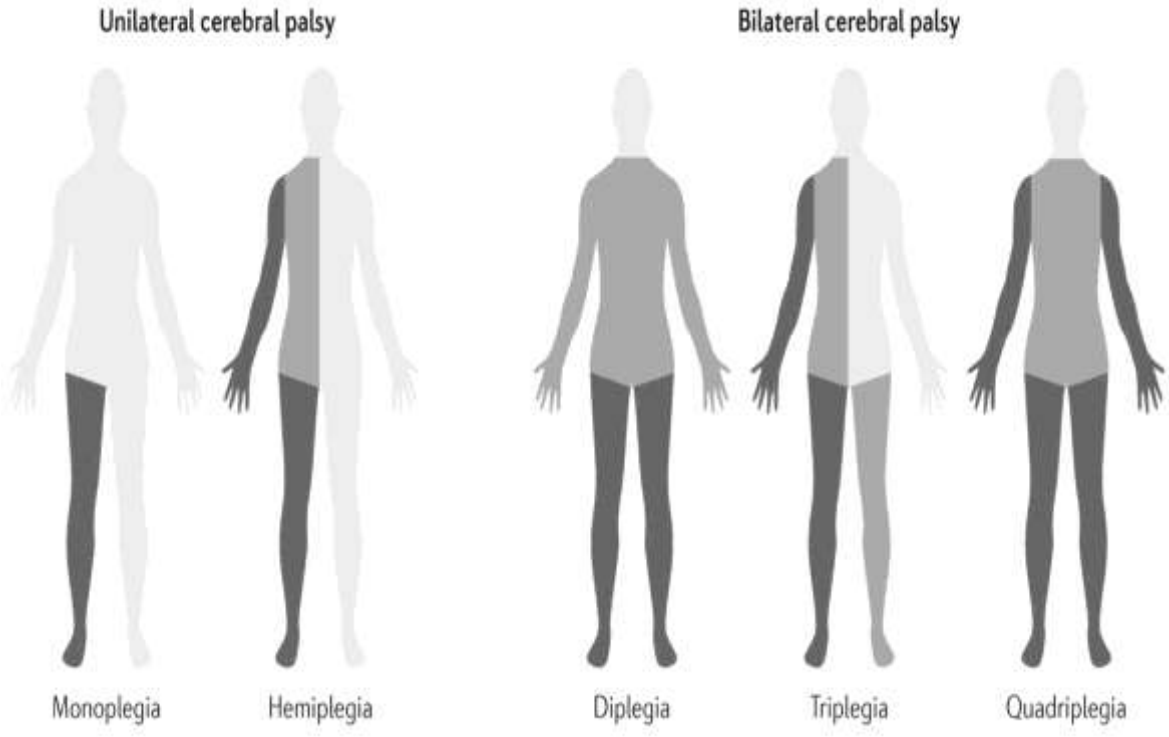

FIGURE 5. Unilateral and bilateral cerebral palsy description taken from (Graham et al., 2016a).

2.4 Motor control systems

A motor control system is comprised of the following components: the cerebral cortex, responsible for processing and commanding motor signals for performance. The frontal motor area, pre and supplementary motor areas, prefrontal cortex, parietal cortices, and association areas responsible for conscious and unconscious visual, auditory, tactile, and proprioceptive feedback (Mukherjee & Chakravarty, 2010). Combined with subcortical areas of the basal ganglia, cerebellum, and brainstem. Notably the red nuclei are involved in stretch reflexes down to the spinal cord which consists of motor units and spinal cord reflexes. Including the cutaneous reflex, withdrawal reflex and muscle reflex which is of major interest in the case of stretch hyper-reflexia (Lundy-Ekman & Weyer, 2022).

The most important motor functions related to stretch hyper-reflexia are related to muscle receptors and muscle stretch reflexes. In this case the tonic (static) stretch reflex by passive stretching is used to test muscle tone and excitability in the case of stretch hyper-reflexia (Lundy-Ekman & Weyer, 2022; Mukherjee & Chakravarty, 2010). Muscle function is largely dependent on the excitement of anterior horn motor neurons, combined with sensory feedback from muscle spindles to the spinal cord. Muscle spindles are responsible for giving feedback on the length and tension of a muscle (Lundy-Ekman & Weyer, 2022). They consist of two types of fibres, intrafusal and extrafusal. Intrafusal fibres are innervated by smaller gamma efferent fibres, while extrafusal fibres are innervated by large alpha efferent fibres. They are also often separated into two types of sensory endings, primary and secondary. Primary receptors are group Ia afferent fibres and secondary are group II afferent fibres (Enoka, 2015). The myotatic reflex, is a role carried out by the muscle spindle. When a muscle is stretched this causes excitation of the spindle and causes the reflex contraction of the muscle and its synergists. In contrast the dynamic stretch reflex is elicited when a fast stretch of the muscle is performed (Lundy-Ekman & Weyer, 2022). The result is stimulation of Ia afferent fibres from the nuclear bag of the muscle spindle through the monosynaptic pathway. The dynamic response subsides rapidly within milliseconds if a sustained static stretch continues. The static stretch is facilitated by the nuclear chain fibres of the muscle spindle, via group II afferents and certain Ia afferent fibres, which act via interneurons polysynaptically (Mukherjee & Chakravarty, 2010).

Muscle tone is maintained by muscle spindles acting through the stretch reflex. See figure 6 below for reference on muscle spindle structure. This background activation is necessary to maintain posture and stability. Tone is responsible for keeping muscle at a given length and is altered during movement to maintain stability. Gamma fibres are mostly responsible for this action and react to any signals sent to alpha motor fibres (Enoka, 2015). This is termed alpha-gamma co-activation and is present during contraction, to contract both intra and extrafusal fibres dependent on the position and force output from the brain towards the spinal cord. The stretch reflex is often tested in clinical settings using one of two tests. Static testing is performed by passive stretching or tone testing. While dynamic testing occurs via muscle and tendon jerk techniques (Lundy-Ekman & Weyer, 2022; Mukherjee & Chakravarty, 2010).

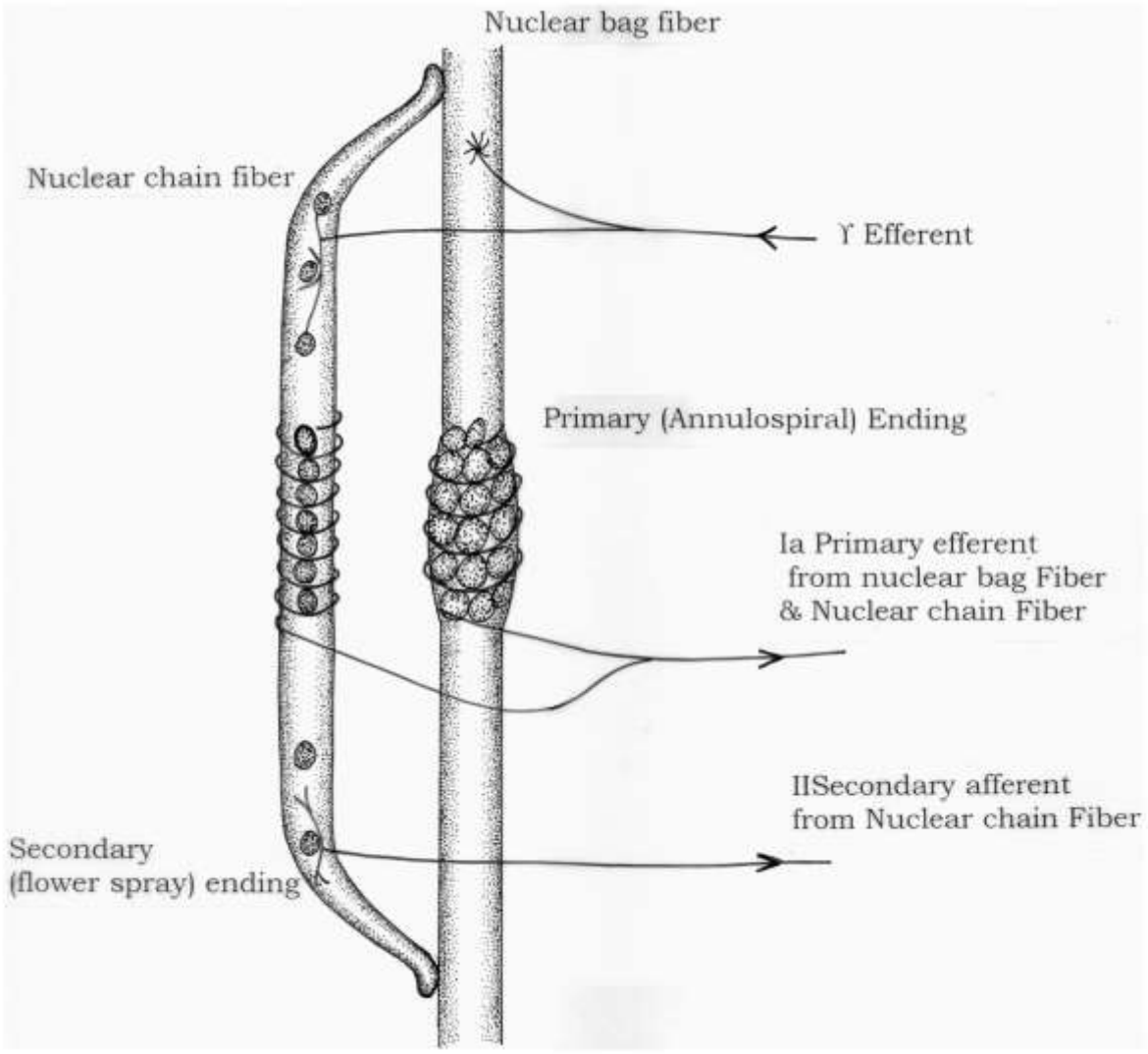

FIGURE 6. Muscle spindle outlining key structural components from (Mukherjee & Chakravarty, 2010)

Furthermore, interneurons play a vital role in mediation of spinal cord function, due to excitation or inhibition. As they are implicated in all segmented and stretch reflex pathways, via peripheral and descending fibre tracts. Interneurons involved in spasticity will be examined in the following sections. Renshaw cells located in the ventral horn medially to motoneurons in lamina VII (Enoka, 2015; Mukherjee & Chakravarty, 2010). Signals from alpha motoneuron axons also stimulate Renshaw cells, in response inhibiting these motoneurons and others innervating the synergist muscle group/s. This creates a negative feedback loop along the alpha motor neuron- Renshaw cell- alpha motor neuron pathway. Resulting in recurrent inhibition, ultimately altering motor neuron excitability. Additionally, Renshaw cells appear to inhibit gamma motor neurons and Ia inhibitory interneurons (Mukherjee & Chakravarty, 2010).

Another vital role of interneurons is reciprocal Ia inhibition. When a muscle is stretched, Ia afferent fibres are activated producing monosynaptic excitation of homonymic alpha motor neurons. Combined with disynaptic inhibition of alpha motor neurons of antagonistic muscle groups, thus resulting in reciprocal inhibition (Enoka, 2015; Lundy-Ekman & Weyer, 2022). Ia interneurons obtain the same excitatory and inhibitory inputs from segmental afferents and supraspinal tracts as alpha motor neurons. Reciprocal inhibition reduces activity in antagonist when the agonist is active, thus the agonist can act unrestricted. This prevents unwanted antagonist activity when an agonist is voluntarily activated. Allowing for optimal output of the agonist muscle groups as they are unopposed by their antagonist counterparts (Castle et al., 1979; Mukherjee & Chakravarty, 2010). Interneurons are also responsible for inhibiting group II afferents, combined with their role in the stretch reflex arc. They are also responsible for production of the flexor extensor response via excitation of flexor alpha motor neurons and inhibition of extensor motor neurons. Moreover, interneurons are responsible for non-reciprocal inhibition, presynaptic inhibition, and flexor reflex afferent activity (Lundy-Ekman & Weyer, 2022). Presynaptic inhibition is related to the amplitude of electrical post synaptic potential (EPSP), as a response to Ia afferent stimulation which is affected by preceding depolarization of the Ia afferent fibre via axo-axonic synapses within exact interneurons. The exact interneurons responsible for presynaptic inhibition are commanded via descending tracts,

resulting in autonomous inhibition of insignificant afferent outputs (Enoka, 2015; Lundy-Ekman & Weyer, 2022) .

The mechanisms related to spasticity are complex, however relatively well understood. It appears that excitatory and inhibitory mechanisms both play a role in spasticity. To what extent is still unclear, however it is hypothesised that inhibitory processes play a greater role than excitatory processes (Mukherjee & Chakravarty, 2010). Correlated to the pathophysiology of spasticity and spastic paretic syndrome, the mechanisms are broadly grouped into two categories. Firstly, spinal mechanisms which are related to the spinal neurons and motor systems. Secondly, they may be grouped into supraspinal and suprasegmental mechanisms. Prior to expansion of these mechanisms, it is worth mentioning the components of both motor control systems and the motor functions of the spinal cord (Mukherjee & Chakravarty, 2010). Spinal reflexes play a vital role in human movement and motor control. A reflex is a non-voluntary movement provoked by stimulation of sensory receptors. In the case of stretch hyper-reflexia the reflex response is altered due to damage to the motor tract, resulting in hyper-activity of the reflex arc and disruption of muscular function. This results in disinhibition of the alpha motor neurons.

2.5 The stretch reflex and the stretch reflex threshold

The stretch reflex (SR) is a fundamental neuromuscular response that occurs when a muscle is stretched, triggering an automatic contraction to counteract the stretch and maintain stability. It plays a crucial role in maintaining muscle tone, posture, and joint integrity (Lundy-Ekman & Weyer, 2022). The SR involves three key components: muscle spindles, Ia afferent neurons, and alpha motor neurons. Muscle spindles, located within the muscle fibres, act as sensory receptors that detect changes in muscle length and the speed of stretch (Enoka, 2015). When a muscle is lengthened, these spindles generate signals transmitted by Ia afferent neurons to the spinal cord. There, the signal activates alpha motor neurons, which initiate a reflex contraction in the stretched muscle (Enoka, 2015). The stretch reflex threshold (SRT) refers to the specific muscle length or joint angle at which the stretch reflex is initiated. It is modulated by descending signals from the central nervous system (CNS), allowing the body to adapt muscle tone based on functional demands. For example, during walking, SRT regulation ensures fluid, efficient movement by preventing premature muscle activation (Enoka, 2015). However, in neurological

disorders like CP, this modulation is disrupted. In typically developing individuals, the SRT is modulated by descending neural input from the brain, which adjusts muscle tone to meet functional demands. However, in individuals with CP, damage to UMNs disrupts this modulation, lowering the SRT. As a result, stretch reflexes are triggered prematurely, leading to excessive muscle activation, stiffness, and impaired movement control (Lundy-Ekman & Weyer, 2022).

In CP, pathophysiological changes to the SRT result from upper motor neuron (UMN) lesions, which impair the balance between excitatory and inhibitory signals in the spinal cord. In CP, the neurological damage to the UMN system significantly alters the regulation of the SRT. Normally, the UMN system provides descending inhibitory signals that suppress the overactivity of reflex pathways, maintaining an optimal SRT. However, damage to the motor cortex, basal ganglia, or corticospinal tracts disrupts these pathways, leading to disinhibition of spinal reflexes and an abnormally low SRT often seen in CP (Valadão et al., 2022). Consequently, stretch reflexes are triggered more readily and at inappropriate muscle lengths, resulting in spasticity and involuntary co-contractions (Levin et al., 2024). A lowered SRT triggers reflex activation prematurely, resulting in hyperreflexia, an exaggerated reflex response. This contributes to spasticity, a condition characterized by velocity-dependent increases in muscle tone. The combination of hyperreflexia and spasticity disrupts voluntary movement control, leading to stiffness, reduced range of motion, and poor coordination. This altered SRT profoundly affects motor function, making it a key target for rehabilitation strategies (Valadão et al., 2021). The SRT is critical for maintaining an optimal balance between muscle stiffness and flexibility. In CP, a reduced SRT contributes to spasticity, a condition defined as hyperactive muscle responses to stretch. This hyperreflexia leads to, difficulty in controlling voluntary movements, limited joint range of motion due to persistent muscle activation, abnormal gait patterns, such as scissoring or toe-walking. This disrupts gait efficiency and increases the energy cost of movement (Meyns et al., 2016). Additionally, spasticity and hyperreflexia restrict joint mobility, making tasks like sitting, standing, or transitioning between postures more difficult (Graham et al., 2016b). Furthermore, functional limitations associated with an altered SRT often lead to secondary complications, such as joint contractures, deformities, and chronic pain. Over time, these issues exacerbate the physical challenges faced by individuals with CP. Addressing the SRT through targeted rehabilitation strategies could reduce spasticity and improve motor control, enabling better movement efficiency and functional independence. Given the centrality of the SRT to motor impairments

in CP, it represents a critical focus for both research and therapeutic intervention (Valadão et al., 2021).

Motor control depends largely on the ability to modulate the SRT dynamically. An overly sensitive or low SRT impairs functional movements, such as walking, grasping, or transitioning between postures. Effective modulation of the SRT is essential for enabling fluid, purposeful movements and reducing the energy cost of activity. Without intervention, an altered SRT perpetuates muscle stiffness, joint deformities, and functional limitations (Lundy-Ekman & Weyer, 2022). Studies using the Montreal spasticity measurement (MSM) protocol support the idea that the TSRT serves as a biomarker for muscle activation and control impairments in individuals with neurological lesions. Understanding the mechanisms behind sensorimotor control disorders and spasticity is crucial for developing personalized treatment strategies tailored to individual disease manifestations. The TSRT functions as a behavioural biomarker of sensorimotor disruption and can help design effective interventions and track recovery. For instance, the TSRT angle defines the spasticity-free zone for upper limb training. Additionally, the parameter "l" can reflect neuropathology and differentiate types of hypertonicity, such as rigidity and spasticity and help distinguish dystonic from spastic hypertonicity in children. A higher value of "l" indicates increased velocity sensitivity of the stretch reflex, meaning faster movements may trigger earlier coactivation of motoneurons, which affects the range of spasticity. This should be considered when designing training tasks for individuals with spasticity. A deeper understanding of the sensorimotor control theory underlying the TSRT and "l" measures will improve precision, reduce misinterpretations, and enhance their diagnostic and prognostic value (Levin et al., 2024).

2.6 Neuromuscular and motor control alterations in cerebral palsy

Cerebral palsy (CP) is characterised by diverse motor impairments that depend on the location of the brain lesion. Damage to motor tracts, including the cortical-striatal-thalamic-cortical and cortico-cerebellar-cortical pathways, disrupts motor planning, coordination, muscle strength, motor learning, and fine motor functions (Graham et al., 2016b). Lesions in descending motor tracts, which project to brainstem and spinal relays, often preserve primitive reflex circuits that would normally disappear during maturation. This preservation results in impaired inhibition, irregular movement organization, postural abnormalities, hyperreflexia, and abnormal muscle tone, culminating in spasticity. The interplay of motor deficits, including hypertonia, hypotonia,

and changes in muscle architecture, contributes to secondary musculoskeletal abnormalities. Each individual with CP faces unique challenges, and this study focuses on how exercise affects the tonic stretch reflex component of hyperreflexia (Lundy-Ekman & Weyer, 2022).

Altered motor control and neuromuscular function often arise from central nervous system (CNS) injuries or conditions, including CP, multiple sclerosis (MS), stroke, and spinal cord injury (SCI). These impairments are frequently linked to upper motor neuron syndrome (UMNS), a condition resulting from damage to descending motor pathways between the motor cortex and spinal cord (Graham et al., 2016a). Symptoms of UMNS are divided into positive and negative features, each further classified into neural and non-neural components. Positive features include spasticity, hyperreflexia, clonus, and co-contraction, all which stem from the loss of lower motor neuron (LMN) inhibition due to damage in corticospinal, corticoreticular, and corticobrainstem tracts during the perinatal period. Negative features involve muscle weakness, impaired motor control, and coordination deficits, reflecting disrupted connections between upper and lower motor neurons (Graham et al., 2016a; Lundy-Ekman & Weyer, 2022). See figure 7 for a diagrammatic representation of the symptoms of UMNS.

Motor dysfunction in CP is complex, involving cascading effects from CNS lesions to peripheral nervous system (PNS) alterations, such as disrupted spinal reflexes, muscle architecture, and function (Lundy-Ekman & Weyer, 2022). Proprioception, critical for system integrity and balance, is often affected. For example, a motor lesion may disrupt the spinal reflex loop, resulting in hyperreflexia. This phenomenon manifests as abnormal stretch and withdrawal reflexes and can occur in both incomplete (iSCI) and complete spinal cord injuries (SCI). Hyperreflexia, a hallmark of UMNS is particularly evident in CP as resistance to passive muscle stretch, including spasticity. Neural components of hyperreflexia, such as velocity-dependent stretch reflexes, result from CNS dysfunction, including disinhibition at the supraspinal level and abnormal reflex loop activity (van den Noort et al., 2017). Spasticity reflects hyper-activity of the stretch reflex arc due to reduced descending inhibitory control, leading to excessive alpha motor neuron activation. Typically, during passive muscle lengthening, reflex activity is minimal. However, in UMNS, disinhibition causes exaggerated reflex responses, creating an opposing force to passive stretch (Graham et al., 2016a). Hyperreflexia can be categorized into phasic and tonic types. Phasic hyperreflexia involves abnormal electromyographic (EMG) activity 30–50 milliseconds after muscle stretch, while tonic hyperreflexia presents as sustained EMG activity 80–100 milliseconds post-stretch

(Lundy-Ekman & Weyer, 2022). This increased excitability often results from abnormal development of corticospinal and corticoreticular tracts, which interferes with neural connections in the spinal cord. Spinal reflex alterations, including reduced disynaptic reciprocal inhibition and changes in intrafusal and extrafusal muscle fibre activity, contribute to heightened stretch reflex responses.

The stretch reflex plays a crucial role in motor control by protecting muscles from injury during rapid or forceful lengthening. In CP, abnormal activity in muscle spindles and reflex arcs disrupts this function, reflecting altered feedback and feedforward mechanisms in spinal circuits. These changes can evolve over time after the initial CNS injury, indicating plasticity in the nervous system. This plasticity involves a multifaceted interplay between neural components, with ongoing research investigating these mechanisms and their implications for treatment (Mukherjee & Chakravarty, 2010). Understanding hyperreflexia and its underlying mechanisms is essential for developing effective treatments for CP. Current approaches target various aspects of spasticity, including neural and non-neural components, and highlight the complexity of this condition. Further exploration of these mechanisms will be addressed in subsequent sections of this paper, providing a foundation for therapeutic strategies (Graham et al., 2016b; Mukherjee & Chakravarty, 2010).

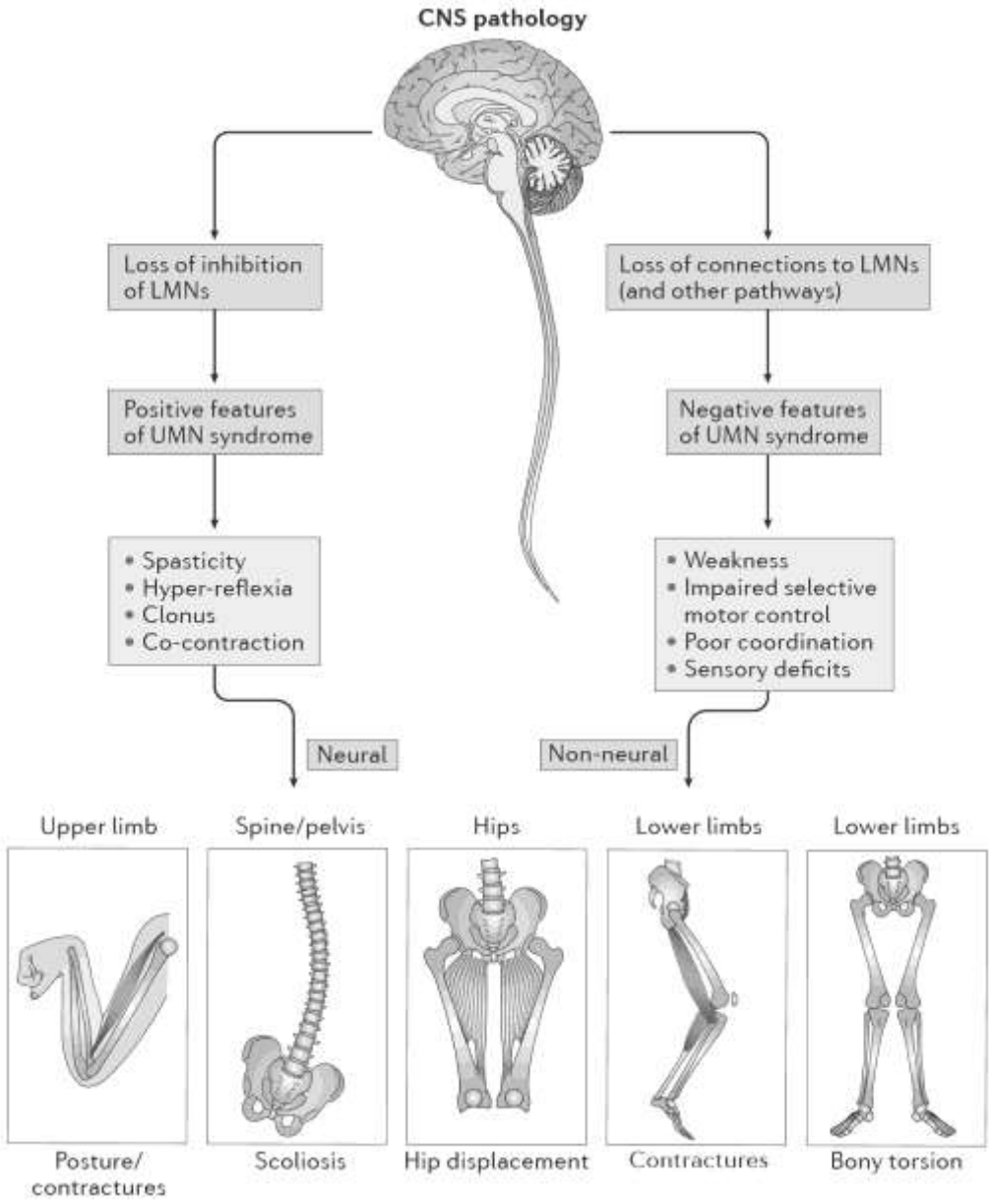

FIGURE 7. Neural and non-neural components of upper motor neuron syndrome and their symptoms from (Graham et al., 2016b).

2.7 Skeletal muscle morphology and function in cerebral palsy

Although cerebral palsy is a result of a central nervous system lesion, the symptoms are not limited to the central nervous system itself. As a result, symptomology extends to the peripheral nervous system and neuromuscular system, where several changes may be seen in, neuromuscular, and skeletal muscle morphology and function see figure 8 for reference (Graham et al., 2016a). These changes are related to both the structural and mechanical function of skeletal muscle and may be observed as changes in muscle fibres, sarcomeres, transcriptional

profiling or stem cells (Graham et al., 2016a). Furthermore, (Howard & Herzog, 2021) suggest that there are a minimum of four factors affecting skeletal muscle force production in cerebral palsy. These are reduced muscle size, and contractile tissue, over stretched sarcomeres, and loss of sarcomeric titin. Research suggest muscle size or physiological cross-sectional area (PCSA) is generally reduced in CP, with some studies suggesting as much as a 27.9% decrease in size compared to TD individuals. Further studies suggest that loss in muscle size may be up to 43% on average (Matthiasdottir et al., 2014). It must be noted that physiological cross-sectional area of muscle is one of the main factors responsible for maximal force production (Enoka, 2015).

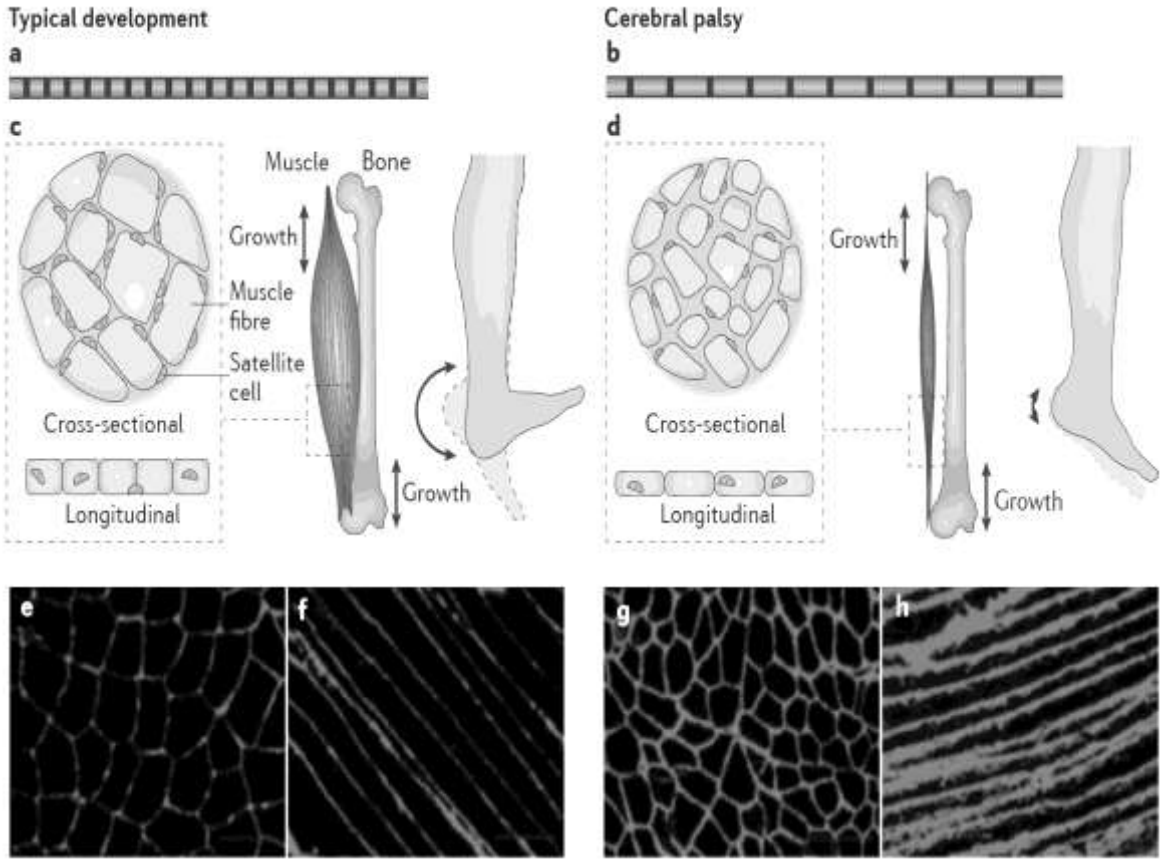

FIGURE 8. Structural properties and changes in skeletal muscle in cerebral palsy (B) compared to typically developing individuals (A).

2.8 Sarcomere structure and dynamics in cerebral palsy

Previous studies have shown that individuals with cerebral palsy have muscles which are shorter, smaller and show decreased diameter compared to typically developing individuals (Castle et al., 1979; Schiaffino & Reggiani, 1996). Furthermore, the distribution of fibre types

is altered, thus the mix of fast and slow twitch fibres is differently distributed (Rose et al., 1994). Studies have however been inconclusive in defining whether the fibre type shift is unidirectional from slow to fast or fast to slow. Research shows mixed results with some individuals presenting a shift from slow to fast fibre type, while others present a fast to slow shift (Tirrell et al., 2012). This may be due to the muscles sampled, individual variances in fibre type distribution, poor sampling techniques, or differences in the level of physical activity, or types of training participants perform (Tirrell et al., 2012). One side effect of changes in muscle fibre type distribution may partially explain the decreased force production capacity seen in individuals with cerebral palsy. More pronounced than the changes seen in fibre type, are the changes in the structure of the sarcomere. Sarcomeres represent the functional structures responsible for muscle contractions. They shorten and lengthen to varying degrees, based on the type of muscle contractions being performed. In individuals with cerebral palsy the sarcomeres can be up to twice as long, and fewer in number. Previous studies from (Lieber & Fridén, 2019), suggest that sarcomere lengths are in the range of 3.5-4 μm. Suggesting, that the tetanic force generation capacity is as low as 20% of normal expected maximal tetanic force production, with consideration for the size of actin and myosin filaments in human skeletal muscle. Furthermore, the studies from (Lieber & Fridén, 2019) suggest that ultrasound may provide limited relevance when investigating muscle adaptations once an upper motor neuron lesion has occurred. As their study found that fascicles were highly stretched, with long sarcomeres, while fascicle length itself remained normal. Skeletal muscle generally present with stereotypical patterning that is the number of sarcomeres in series, and their functional limits. This can be seen by consistency of the number of sarcomeres in series and parallel. The exceptionally long sarcomeres and passive tension seen in contractures, suggest deficiency in the sensory mechanisms normally underlying and regulating, the length-tension relationship (Lieber & Fridén, 2019; Smith et al., 2011).

In addition to the changes in sarcomere length seen in chronic contractures, whereby the muscle is constantly contracting the sarcomere is highly lengthened although the muscle length is significantly shorter. These properties present a paradox in myo-morphology and have been observed in several different muscle groups in CP patients, (Lieber & Fridén, 2019; Smith et al., 2011). Functionally, it is hypothesised that the long sarcomeres result in low active tension and force production. With alterations observed throughout the range of motion, while passive muscle force is much higher than normal (Smith et al., 2011). This may be related to altered cross-bridge cycling frequency seen in individuals with CP, which yet has been studied very

little. Hypothetically, the number of cross bridge cycles is altered in person with CP, that is fewer cross bridges are able to cycle, resulting in lower-than-expected force production (Lieber & Fridén, 2019; Smith et al., 2011). As the number of cross bridges in series, allows for alterations in force production capacity. That is the more cross bridges in series, the greater potential for force production (Lieber & Fridén, 2019). This must be considered in combination with factors such as tendon stiffness, conduction velocity of nerves, and voluntary activation which also affect force production capabilities.

2.9 Non-neural components of hyper-resistance

Non-neural components are related to the muscle tissue properties, identified via three components elasticity, viscosity and shortening properties. The non-neurological component alterations are believed to occur because of muscular adaptations caused by neurological dysregulation. This is seen when muscle contractures occur due to shortened muscles or stiffen due to architectural changes in the muscle tissue. These maladaptive changes may be magnified in children due to the growth process and its effect on tissue development and motor control (Graham et al., 2016a). An explanation may be due to the alterations in musculo-tendinous properties in CP due to chronic changes, caused by contractures. The changes at the muscle and tendon level, may be induced as a biproduct of altered motor control or due to physiological processes which may be altered in CP, due to the motor lesion itself (Sloot et al., 2021). Altered stretch reflex activity has been associated with gait pattern alterations seen in CP. Caused by limited ankle dorsiflexion during the contact, swing, and stance phases of the gait cycle, combined with decreased push of power at the terminal phase (Meyns et al., 2016). The hyper-activity is often related to chronic maladaptation in the musculoskeletal system, seen via contractures, increased muscle stiffness and deformation in developing skeletal bones (Peeters et al., 2023). Due to this assumption aggressive treatment methods are commonplace with the aim to decrease or postpone orthopaedic surgery. Other studies have also argued that hyper-reflexia plays and insignificant role in gait changes (Nielsen et al., 2005). Thus, it is important that we can assess muscle-tendon interactions, during passive and active movements.

Previous studies have suggested that the medial gastrocnemius (MG) muscle belly has a reduced capacity for lengthening during low-speed passive ankle rotations (Barber et al., 2012; Kalkman et al., 2018). Additionally, during the mid-stance phase of walking muscle fascicles

appear to lengthen more, thus suggesting altered musculo-tendinous interactions (Kalsi et al., 2016). These results suggest that muscle-tendon unit function is altered in both passive and active states. The resultant outcome appears to be an increase in fascicle lengthening, which may alter muscle spindle activity (Bar-On et al., 2018). Leading to increased activation of muscle spindles, and increased Ia afferent reflex arc activity, resulting in increased SR response (hyper-activity). Furthermore, (Graham et al., 2016a) hypothesised that muscle architectural changes may be altered in children with cerebral palsy (see figure 6). This may be attributed to differences in sarcomere length, although similar fascicle lengths may appear. Graham et al., 2016 suggested this may be due to the way that sarcomeres are added in series during bone maturation (growth), suggesting the sarcomeres may not be added in series in individuals with cerebral palsy. Therefore, when the bone grows the ankle is pushed into plantarflexion due to structural maladaptation, and eventually contractures. (Graham et al., 2016a). In addition, suggestions of increased extracellular matrix (ECM) may alter MTU interaction, further helping to explain increased muscle spindle activity. A summary of factors contributing to muscle resistance to stretch has been adapted from (Lundy-Ekman & Weyer, 2022). The main factors are summarised in the figure 9 below are, descending motor commands, proprioceptive information, connective tissue properties, weak cross-bridge binding, and titin, all forming a muscles resistance to stretch.

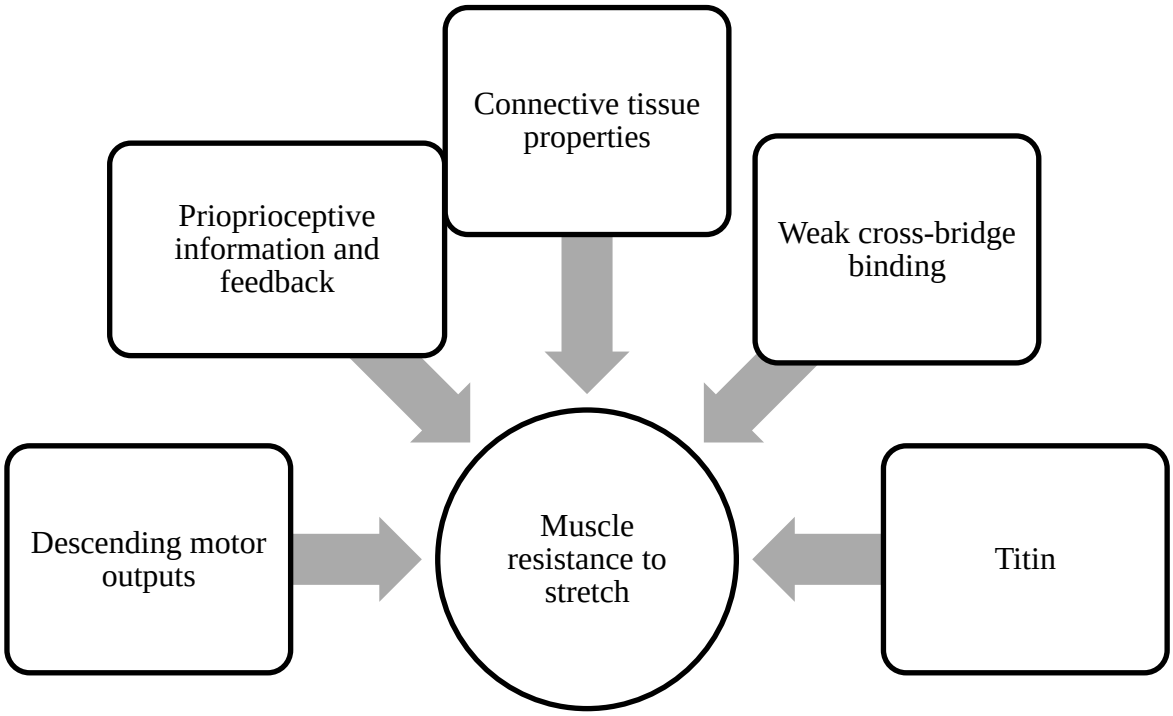

FIGURE 9. Summary of factors affecting muscle resistance to stretch created with information from (Lundy-Ekman & Weyer, 2022)

2.10 Exercise rehabilitation for cerebral palsy

Unfortunately, there is currently no cure for CP, however exercise and physical activity (PA) have the potential to impact health outcomes and activities of daily living. Individuals with cerebral palsy most often have reduced physical activity resulting in lower cardiorespiratory fitness and muscle strength. (Verschuren et al., 2016), presented the figure 10 attached below as an example of the changes in PA seen due to GMFCS. It clearly identifies the change in PA due to GMFCS scores, with higher scores correlating with lower scores in PA. This is logical as we would expect lower scores from less mobile individuals with significant alterations in motor control. Based on a review of the current literature, it appears that individuals with CP spend between 76% to 99% of their days being sedentary, less than 18% partake in light PA, and between 2% to 7% perform vigorous exercise (Reid et al., 2015). Thus, the potential for development of chronic health conditions is increased. Poor physical activity has been studied extensively, it may lead to the development of chronic metabolic and cardiovascular disease. These may include diabetes, asthma, hypertension, stroke, joint pain and arthritis. Therefore, improving the amount of physical activity and health outcomes presents a vital part of the

exercise paradigm for individuals living with cerebral palsy (Verschuren et al., 2016). The recent guidelines set out by (Verschuren et al., 2016), may provide us with a starting point for physical activity guidelines for those with CP see table 2 below.

Exercise rehabilitation presents an important and unique tool for exercise, health and medical professionals in managing and treating cerebral palsy, not just to manage the disease but prevent further potential for the development of comorbidities. Recent evidence suggests that the timing of rehabilitation may affect the response and outcomes of the intervention, with earlier interventions appearing to offer the greatest potential (Reid et al., 2017; Verschuren et al., 2016). The benefits and use of exercise for cerebral palsy rehabilitation are relatively well studied, however optimal use and prescription of exercise remains unelucidated (Valadão et al., 2021). Exercise interventions most often use strength training, gait training, flexibility training or a combination of several methods. Commonly used methods include constraint induced movement therapy (CIMT), bimanual intensive therapy (BIT), tailored exercise interventions, gait training and balance training (Lundy-Ekman & Weyer, 2022). Exercise prescription for individuals with CP should consider a few important factors, the type, subtype, and brain lesion size and locality. Furthermore, the age and cognitive function of the individual should always be considered in developing exercise interventions, as this may affect their implementation and safety (Reid et al., 2015). Other factors which should be considered as with any exercise programs are, the mode, dose, context, relevance and timing of the program (Verschuren et al., 2016). Mode relates to the type of exercise performed, and its relevance to the motor qualities being trained. Dose is the amount of exercise performed, it combines both duration and intensity of exercise. By altering the dose the outcomes may change, thus finding a point suitable for everyone is of upmost importance. Relevance refers to the relevance of the exercise type in helping improve relevant outcomes. This will likely vary depending on the individual, hence our study for individualised exercise programs (Verschuren et al., 2016).

The main aim of exercise interventions is to cause adaptation in the relevant systems. In this case the aim is to alter nervous system, and muscle structure and function, which effects the function of the neuromuscular system (Reid et al., 2015). The neuromuscular system is highly plastic in nature and responds well to training in TD individuals. However, so far data is mixed in identifying the outcomes and changes in the neuromuscular system in CP (Moreau & Lieber, 2022). Consideration of the individualised responses to exercise as a unique biological entity combined with a disordered state. Thus, individualised programming may help maximise

relevant outcomes dependent on the individual's needs and condition. Another important consideration is the amount of exercise that is functionally possible for each individual based on their functional capacity (Verschuren et al., 2016). A key challenge in sustaining physical activity is lifestyle change, which can be especially difficult for people with CP due to various barriers. These include physical limitations, environmental factors, and caregiving challenges, such as time constraints, financial strain, and emotional stress. Families and caregivers have identified these barriers as significant obstacles to meeting physical activity guidelines (Verschuren et al., 2016). Given these challenges, it is crucial to identify individuals who would benefit from exercise interventions to mitigate long-term health risks. One of the most effective tools for this is cardiopulmonary exercise testing, which is the gold standard for assessing exercise tolerance and cardiorespiratory endurance. Exercise testing can help clinicians identify those at risk for poor health outcomes and determine who would benefit from targeted interventions. Despite its clear relevance, clinical exercise testing is underused, primarily due to a lack of clinician understanding and training on test administration and interpretation (Verschuren et al., 2016).

To address these gaps, clinicians should encourage physical activity and consider recommending exercise testing when individuals with CP experience physical exhaustion or limitations in activity. The first step in designing exercise interventions is to assess physical fitness through objective testing. Once fitness levels are determined, it is important to identify the causes of deconditioning, whether due to inactivity, poor nutrition, disease-specific pathophysiology, or a combination of factors (Verschuren et al., 2016). There are established, clinically feasible exercise tests for individuals with CP, supported by solid evidence of their effectiveness. Much of the current research on physical activity benefits comes from studies involving ambulatory children and adolescents with CP. However, applying these findings to real-world interventions remains challenging due to practical limitations and a lack of inclusion of such applications in research (Moreau & Lieber, 2022).

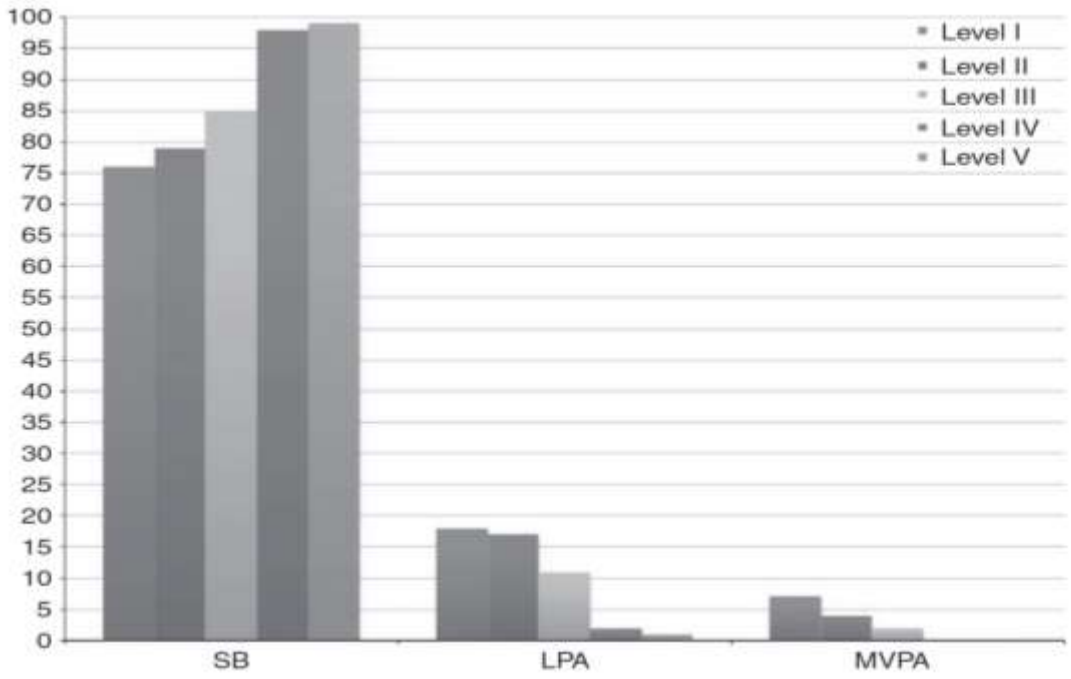

Figure 10. Physical activity based on GMFCS scores, SB- sedentary behaviour, LPA- low physical activity, MVPA- moderate-vigorous physical activity from (Verschuren et al., 2016).

To address this, it is vital to establish guidelines and protocols that promote physical activity as part of regular clinical and therapy centre visits for individuals with CP. These guidelines will help improve clinician comfort with discussing physical activity and guide future intervention studies to further promote healthier lifestyles and improve long-term health outcomes for this population. Table 2 below is comprised of exercise recommendations for cerebral palsy and gives a good outline of the type, frequency, intensity and time all key components of exercise programming (Verschuren et al., 2016).

Table 2. Exercise recommendations for cerebral palsy created with information from (Verschuren et al., 2016).

Exercise	Frequency	Intensity	Time	Type
Cardiorespiratory fitness	1-2 sessions per week working towards three.	>60% of HR max, or 40% of HRR, or between 46-90% of $VO_{2peak.}$	At least 20 minutes per session, for at least 8-16 weeks *frequency dependant.	Regular purposeful exercise involving major muscle groups.
Resistance training	2-4 times per week on non-consecutive days.	1-3 sets of 6-15 repetitions. Equivalent to 50-85% of 1RM	No specific details mentioned, but duration of 12-16 weeks.	Progressive based on single and multi-joint exercises.
Daily physical activity- moderate to vigorous	>5 days per week	Moderate to vigorous intensity	60 minutes	Varied modes and activities
Daily physical activity- sedentary	7 days per week	Sedentary <1.5 METs	<2 hours per day or break up sitting every 30-60mins.	Non-occupational, leisure time activities, such as watching TV, using computer or playing games.

2.11 The effects of exercise on the neuromuscular system

Exercise has a profound impact on the neuromuscular system, which includes both the muscles, and the nervous system involved in movement and coordination. Regular physical activity enhances neuromuscular function by promoting neural adaptations, such as improved motor unit recruitment and coordination between muscles and nerves (Enoka, 2015). This leads to more efficient movement patterns and an increase in muscle strength and endurance. Resistance training, for example, can stimulate the growth of muscle fibres and the recruitment of additional motor units, improving overall muscle performance and force production. Additionally, exercise has been shown to enhance neuromuscular plasticity, allowing the system to adapt to new movement demands and recover from injury more effectively (Lundy-Ekman & Weyer, 2022). One of the key effects of exercise on the neuromuscular system is the improvement in muscle fibre composition and muscle function. With consistent strength training, there is an increase in the cross-sectional area of muscle fibres, particularly type II (fast-twitch) fibres, which contribute to greater force production (Enoka, 2015). In addition, endurance exercise improves the efficiency of type I (slow twitch) muscle fibres, which are

critical for sustained activity and fatigue resistance. These changes result in better overall muscle performance, including increased strength, flexibility, and endurance. The neuromuscular system also adapts by increasing the number of motor neurons and enhancing the connection between the brain and muscles, which leads to improved motor control and coordination (Enoka, 2015; McArdle et al., 2010).

Exercise also has a significant impact on the central nervous system (CNS), enhancing brain function and motor control. Regular physical activity has been linked to improved synaptic plasticity, which refers to the strengthening and formation of new connections between neurons (Lundy-Ekman & Weyer, 2022). This can lead to better motor learning, quicker reaction times, and enhanced motor planning and execution. For individuals recovering from neurological impairments or injuries, such as stroke or spinal cord injury, exercise promotes neuroplasticity, supporting the brain's ability to reorganize itself and regain lost functions. In particular, exercises that require complex movements or coordination can help retrain the CNS and facilitate recovery of lost motor functions (Lundy-Ekman & Weyer, 2022). However, the effects of exercise on the neuromuscular system can vary depending on the type, intensity, and duration of the exercise, as well as the individual's baseline health and neurological condition. In people with neurological disorders such as cerebral palsy or multiple sclerosis, the neuromuscular adaptations may be less pronounced, and exercise interventions need to be tailored to the individual’s specific needs and capabilities (Lundy-Ekman & Weyer, 2022). While regular exercise can improve neuromuscular function in these populations, careful consideration of the mode, intensity, and progression of exercise is essential to avoid overexertion or injury. With appropriate modifications and guidance, exercise can significantly enhance the neuromuscular system’s function and contribute to improved quality of life and mobility (Moreau & Lieber, 2022; Verschuren et al., 2016).

2.12 Assessing stretch hyper-reflexia and the tonic stretch reflex

Assessing hyper-reflexia and hyper-resistance is crucial for identifying clinical status, planning treatment, and evaluating intervention success. These assessments are typically divided into subjective and objective measures. Subjective methods, such as the modified Ashworth or Tardieu scales, remain dominant in clinical practice due to their ease of use and practical implementation in a clinical setting, guiding decision-making and treatment planning (Graham

et al., 2016b). On the other hand, objective measures, which involve manual or motorized instrument assessments, tend to be more reliable (Sloot et al., 2017), though they are often time-consuming and require the operation of complex equipment. This raises the question of whether different methods assess similar characteristics, leading to comparable conclusions that inform health management and planning (Graham et al., 2016b). An important consideration is the time required to perform more objective tests in clinical settings. These tests are typically more complex to organize and conduct, often necessitating additional training for practitioners. While both subjective and objective assessments are crucial, the current methods often remain highly subjective (Malhotra et al., 2009), highlighting the need for reliable, repeatable, and valid objective measures.

Another key issue in using the stretch reflex threshold (SRT) is the method of calculation. Valadao et al. (2022) suggest that much of the literature contains a systematic error in SRT identification due to the stretch reflex latency—the time between the mechanical initiation of the stretch reflex (SR onset) at the muscle spindle and the onset of the EMG signal (SR EMG onset). The variance between the joint angle at SR onset and SR EMG onset (SRT) is positively correlated with stretch velocity. As velocity increases, the error in joint angle calculation also increases due to the monosynaptic SR latency of approximately 30ms. This results in an overestimation of the SRT at higher velocities. Although Levin and Feldman (1994) proposed a latency correction method, later studies did not adopt this approach, introducing a systematic error in SRT calculation. SR latency primarily depends on individual anthropometrics and axonal pathways to the target muscles. Therefore, subtracting a fixed 30ms average latency could introduce a subject-specific error (Valadão et al., 2022). To address this, it is recommended to calculate and subtract the individual SR latency from the SRT. This would help assess the impact of the correction method on the linear relationship between SRT and stretch velocity and validate the TSRT method with latency correction (Valadão et al., 2022). Valadão et al. (2022) implemented latency correction by measuring Hoffman reflex (H-reflex) latency

2.13 The passive muscle stretch technique

The passive muscle stretch technique, often performed using a motor-driven dynamometer, is commonly used to assess hyper-resistance and hyper-reflexia. This method allows for the simultaneous collection of kinematic, torque, EMG, and ultrasound data by performing passive

movements at various velocities and accelerations to differentiate between neural and non-neural components of hyper-resistance. Fast movements help identify velocity-dependent neural components by detecting EMG bursts at the stretch reflex threshold (SRT), with higher velocities leading to stronger EMG bursts (Sloot et al., 2021). Acceleration also influences SRT, with higher peak accelerations leading to earlier EMG burst onset (Sloot et al., 2021). Slower movements are better suited for identifying involuntary background activation and non-neural tissue components (van den Noort et al., 2017), with non-velocity-dependent movements assessing EMG levels and the EMG-joint angle interaction, while non-neural components focus on torque-angle interactions. Figure 12 below represents an example of SRT identification as performed by (Bar-On et al., 2018). Typically, the subject is seated with knee and hip joints flexed, and the foot placed in the dynamometer's footplate to measure torque and passive resistance. EMG data is often recorded from the Soleus (Sol), medial gastrocnemius (MG), and lateral gastrocnemius (LG) during passive plantar and dorsiflexion, following SENIAM guidelines (Hermens et al., 2000). The motor then performs a set number of movements at specified velocities or accelerations, often using randomized patterns. The resulting data includes range of motion, peak torque, passive torque, SRT, maximum angular velocity, average RMS EMG, work, and stiffness. According to van den Noort et al. (2017), slow movements produce variables such as ROM, maximum angular velocity, average RMS EMG, and SRT, while fast movements yield data on angular velocity, RMS EMG, SRT, work, angle of catch (AOC), and intensity of catch. While the passive muscle stretch technique is a reliable, reproducible, and valid method for research, its equipment can be bulky and restrictive. Additionally, it isolates the test limb, which may not fully represent real-life activities like gait (Sloot et al., 2017). Nonetheless, it provides valuable objective and quantitative data on hyper-resistance, which can inform clinical decision-making for patient treatment (Graham et al., 2016b).

2.14 Electromyography and Surface Electromyography

Electromyography is a technique which is used to identify the electrical activity generated in a muscle. In CP it helps us to identify the TSRT see figure 11 below for an example from (Sloot et al., 2017). The EMG signal represents a summation of the action potentials and is the product of nervous system activity and physiological activities of the muscle (Enoka, 2015). One challenge that may be encountered when using sEMG is the noise which may be seen due to

different tissue properties and or cross talk when the electrodes are placed over bony landmarks (Enoka, 2015). Thus, SENIAM has produced guidelines for placement of the electrodes which can be used by practitioners when conducting research into sEMG activity to try and minimise the signal to noise ratio (Hermens et al., 2000). This aims to reduce variations in signal recordings and spectral features, therefore optimising data collection. Research by Staudenmann et al., suggests that electrodes placed in the correct alignment have a low root mean square (RMS) difference in muscle force and sEMG recordings (Staudenmann et al., 2006). Previous studies have shown heterogeneity in surface electromyography (sEMG) activity, during passive muscle stretch. However, high levels of irregularity in the sEMG amplitude and velocity of the SRT were noted during passive muscle stretch. Currently, the reasoning behind these differences is unclear and unconfirmed (Bar-On et al., 2018).

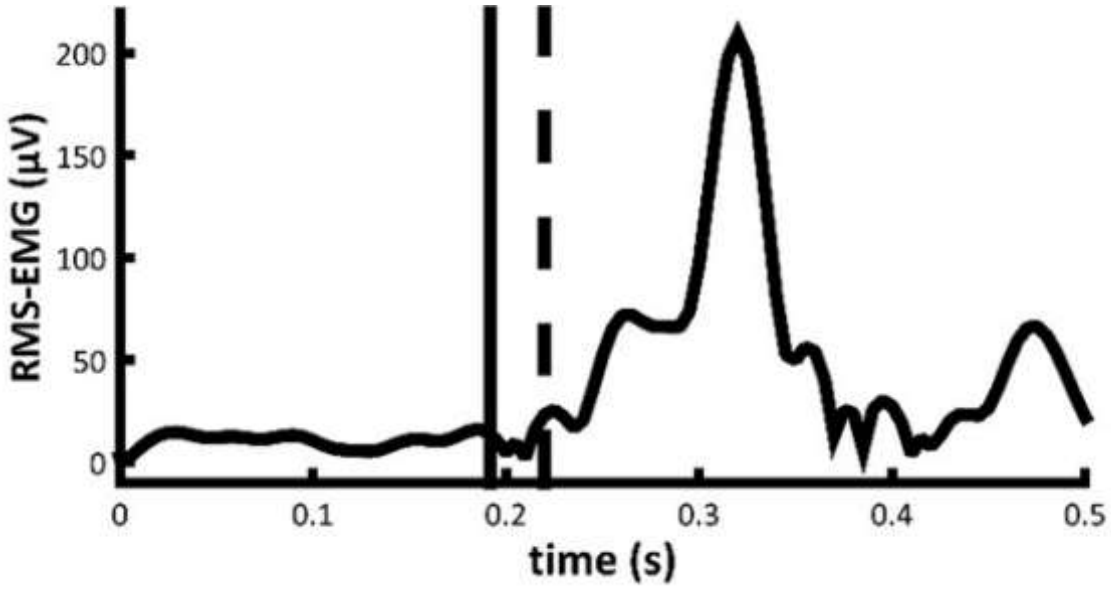

FIGURE 11. Example of Stretch Reflex threshold identification using RMS-EMG. The black dashed line represents EMG onset, and the black solid line represents the stretch reflex threshold at approximately 30ms prior to EMG onset. Adapted from (Bar-On et al., 2018).

3 CASE STUDY AIMS, RESEARCH QUESTIONS AND HYPOTHESES

This case study was conducted as part of the larger EXECP study by Valadão et al. (2021), which aimed to investigate the effects of an exercise interventions on motor, functional, and metabolic outcomes in individuals with CP. The current case study specifically focused on examining the TSRT latency correction method over a longitudinal period in a single subject with spastic CP. This will be termed TSRTcorrected henceforth. EMG data were collected at four time points (pre1, pre2, post1, and post2) and four stretch velocities (55, 110, 200, and 291°/s) using the passive muscle stretch technique to assess changes in the TSRT before and after a 12-week exercise intervention.

The TSRT, a component of stretch hyper-reflexia and a key feature of upper motor neuron syndrome (UMNS), was analysed to determine its velocity-dependent characteristics. Stretch hyper-reflexia, characterized by stretch reflex onset at lower-than-expected velocities and joint angles, is a hallmark of TSRT and can be observed during rapid movements via EMG activity (Valadão et al., 2022). This phenomenon, sometimes referred to as stretch reflex disorder (see Figure 12 below), is central to understanding spasticity, as originally defined by Lance (1980) as a velocity-dependent hyperactive stretch reflex. The study aimed to address gaps in the understanding of TSRT and explore how exercise interventions might influence this measure.
To improve TSRT precision, the study employed the latency correction method (TSRTcorrected), which accounts for variations in H-reflex latency due to differences in individual anthropometrics and nervous system function. This approach, building on the work of Levin and Feldman (1994) and adapted by Valadão et al. (2022), mitigates systematic errors caused by uncorrected stretch reflex latency (~30 ms). Such errors, particularly at higher velocities, can lead to overestimation of TSRT by creating a positive linear relationship between velocity and joint angle error. By applying and validating this correction, the study aimed to standardize TSRT measurement for future research. Using EMG data from EXECP studies for the H-reflex and passive muscle stretch protocols, this research examined whether exercise altered TSRT in the subject and whether TSRT demonstrated velocity-dependent characteristics.

The study also sought to uncover neuromuscular and nervous system adaptations associated with the intervention, providing valuable insights into rehabilitation strategies for individuals with CP. By refining TSRT calculations and exploring exercise as a rehabilitation method, the

research aimed to enhance clinical exercise prescription and improve outcomes for individuals with CP and similar neurological conditions. In summary, the primary focus of this study was to investigate TSRT differences using the latency correction method across four stretch velocities and four time points in a subject with spastic CP. It evaluated whether TSRT was velocity-dependent and how the exercise intervention influenced it. This longitudinal approach provides a foundation for understanding how targeted interventions can optimize treatment for stretch hyper-reflexia and associated neuromuscular disorders.

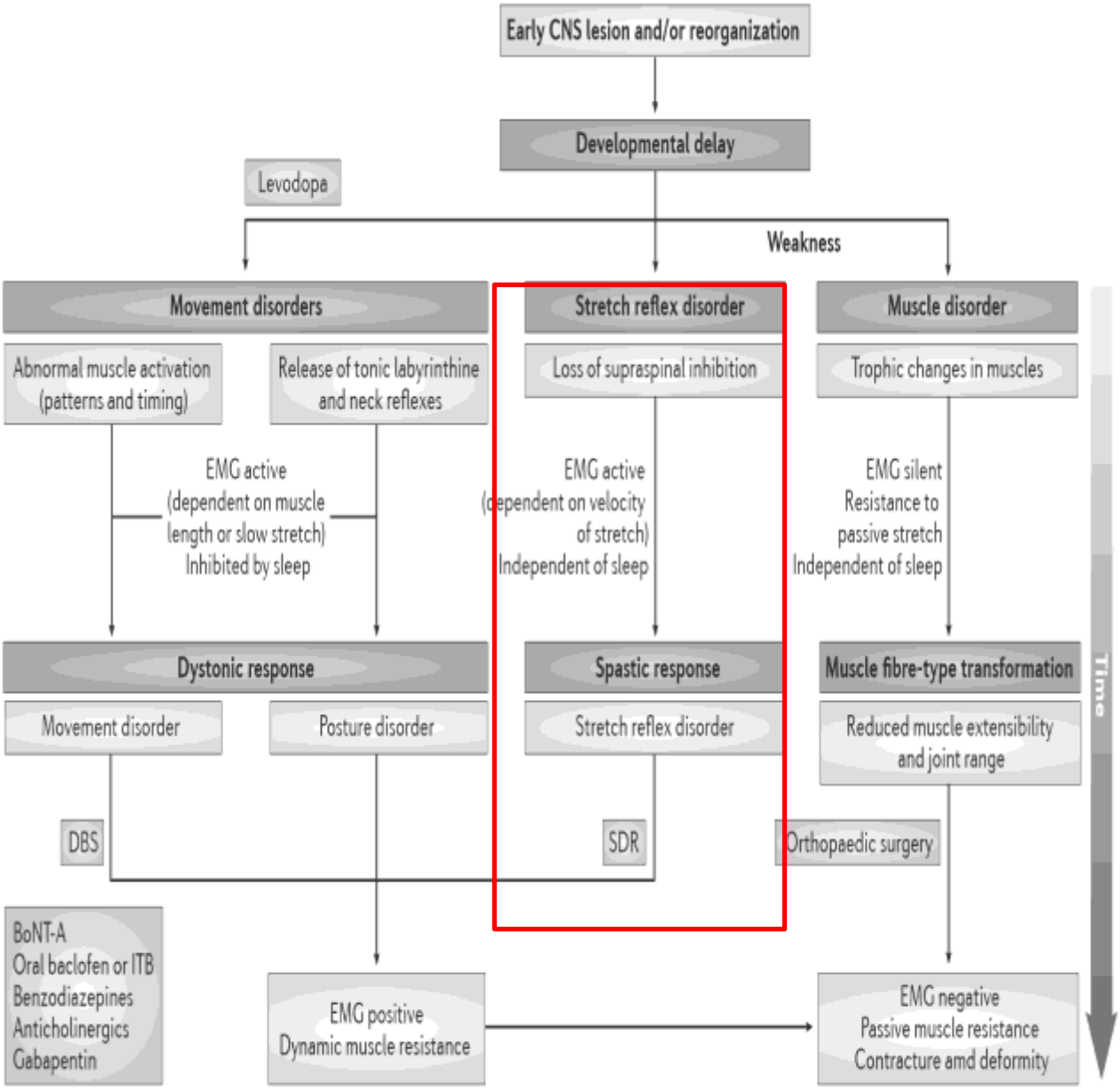

FIGURE 12. Various movement disorders in cerebral palsy highlighting in red the specific focus of this project from (Graham et al., 2016a)

3.1 Research questions and hypotheses

The primary research questions are 1) is the TSRTcorrected velocity dependent in this case study subject for the soleus and medial gastrocnemius? 2) how does TSRTcorrected for the soleus and medial gastrocnemius muscles vary across different stretch velocities (55, 110, 200, and 291°/s) and 3) the effects of the exercise intervention and retention on TSRTcorrected in SOL and MG ?.

The hypotheses include:

- **Null Hypothesis**: TSRTcorrected is not velocity dependent
- **Alternate Hypothesis**: TSRTcorrected is velocity dependent.
- **Null Hypothesis 2**: No significant differences in TSRTcorrected between the muscles at different velocities.
- **Alternate Hypothesis 2**: Significant differences in TSRTcorrected between the muscles at different velocities
- **Null Hypothesis 3**: No significant differences in TSRTcorrected between the muscles pre vs post intervention.
- **Alternate Hypothesis 3**: Significant differences in TSRTcorrected between the muscles pre and post intervention.

4 METHODS

The study designs for this project will be separated into two sections to provide clarity to the reader as to which parts of the study were performed as part of the EXECP study. That is the larger study which provided the case study data. The case study was done in collaboration with the EXECP principal investigators. The subject for this study was one individual with spastic cerebral palsy. Due to data anonymisation no further subject specific characteristics can be provided.

4.1 Sampling and subject recruitment for EXECP

The original EXECP study recruited 24 individuals aged 9 to 24 years, all diagnosed with spastic cerebral palsy. Participants were primarily sourced from hospitals, CP clinics, and physiotherapy centres in the Jyväskylä area of central Finland. Due to a limited number of participants, the recruitment area was extended to surrounding towns and cities. However, the recruitment process was disrupted in the spring of 2020 by the coronavirus pandemic, which prevented several participants from joining the study. The control group, consisting of 24 age- and sex-matched individuals, was recruited from local schools and universities. Recruitment occurred from May 2017 to December 2020. The project accepted male and female subjects aged between nine and twenty-four years, diagnosed with unilateral or bilateral spastic cerebral palsy. Furthermore, a gross motor classification system score of level I to III was required. Participants were not accepted if the met any of the following criteria; a) lower limb surgery and/or pharmacological treatments in the past 6 months, b) undergone dorsal rhizotomy, c) utilised serial casting of the lower limb, d) have performed resistance training of the lower limbs in the previous six months, e) unable to cooperate with instructions during testing and intervention sessions, f) unable to stand with both heels in contact with the floor. In exceptional circumstances participants may be included with criteria a and b.

4.2 EXECP study design

The EXECP intervention was conducted at the Neuromuscular Research Centre, at the University of Jyväskylä, Finland. Participants trained 2–3 times per week for 12 weeks, with the number of sessions adjusted based on their regular physical activity. Each 90-minute session

began with 5–10 minutes of inclined treadmill walking, followed by 60–75 minutes of strength training, and concluded with 10–20 minutes of flexibility exercises. Sessions were supervised by certified physiotherapists or strength coaches to ensure proper exercise quality and form.

The EXECP study employed a non-concurrent multiple baseline design with two pretests and two post-tests, spaced three months apart. A three-month control period was followed by a three-month exercise intervention (EXECP) between Pretest 2 and Post-test 1. Participants underwent post-testing immediately after the intervention and again three months later to evaluate any residual effects. The intervention group, composed of individuals with cerebral palsy, was age- and sex-matched with typically developing controls. A multiple baseline design was chosen over a randomized control trial to account for the expected heterogeneity within the intervention group, allowing participants to serve as their own controls and minimizing variability in the studied variables. The EXECP intervention was designed to be performed year-round, avoiding potential seasonal influences see figure 13 below for a reference of the study design and intervention structure. Participants were instructed to maintain their usual daily activities, training, and therapy schedules, with stable exercise frequency. During the intervention, participants were advised to reduce overall training volume to prevent overload, and any changes in activity during the control period were documented. Testing procedures were standardized to ensure consistency and reduce procedural variability. An outline of the testing session schedule is provided in figure 15 for reference on the details and timing of tests . To minimize bias, data analysis was deferred until after data collection, with testers blinded to the results, allowing them to focus solely on protocol implementation. This design ensures

objective and quantitative outcomes with minimal bias, despite potential measurement variability.

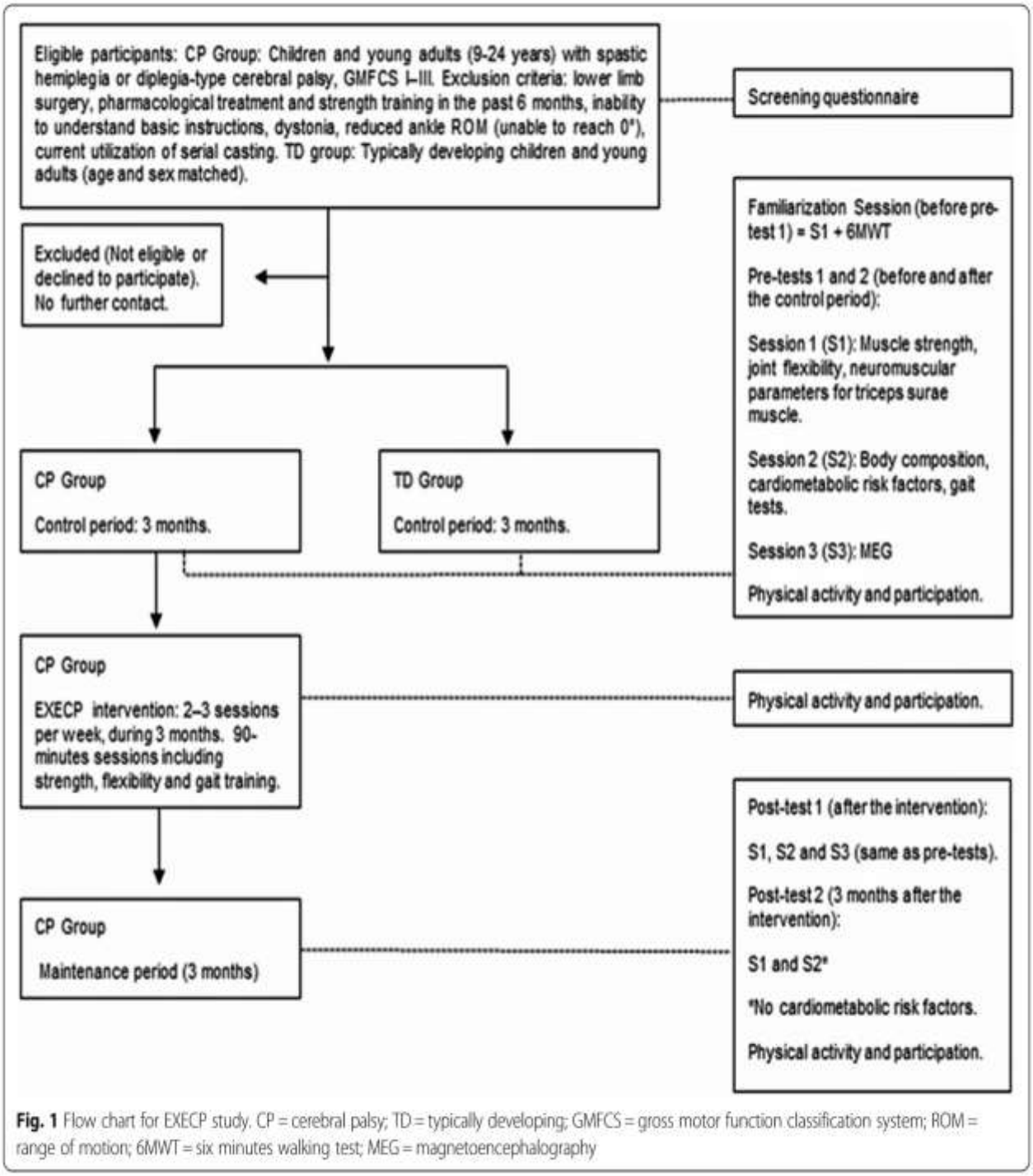

Fig. 1 Flow chart for EXECP study. CP = cerebral palsy; TD = typically developing; GMFCS = gross motor function classification system; ROM = range of motion; 6MWT = six minutes walking test; MEG = magnetoencephalography

FIGURE 13. Flow chart for the EXECP intervention outlining participant selection, testing and exercise intervention from (Valadão et al., 2021).

4.3 Case Study design

This case study involved a single subject randomly selected for analysis, adhering to the EXECP sampling and recruitment protocol previously outlined above and by Valadão et al. (2021). This case study included a single participant with spastic cerebral palsy (N=1). To qualify, the participant had to complete all components of the EXECP exercise intervention and relevant testing session 1. The study focused on an individual with spastic cerebral palsy (N=1) and formed part of a broader investigation EXECP, as detailed above. This exploratory study is one of the first to examine TSRTcorrected longitudinally in this context. It utilized quantitative data collection methods, including electromyography (EMG), to provide a detailed analysis of the h-reflex latency and TSRTcorrected for the case study subject. Ethical approval was as part of the EXECP intervention and granted by the Central Finland Healthcare District Ethics Committee (U8/2017). The project adhered to the University of Jyväskylä's data management policies. Participant confidentiality was maintained through anonymization of data, and informed consent was obtained from participants and/or their guardians. Data were securely stored and managed accordance with institutional standards.

Testing sessions

All testing sessions were conducted as part of the EXECP study. Testing sessions were conducted at the NMRC and the Centre for Interdisciplinary Brain Research (CIBR) at the University of Jyväskylä. Each testing phase (pre-test 1, pre-test 2, and post-test 1) comprised three separate sessions (S1, S2, and S3), scheduled 4 hours to seven days apart. Post-test 2, however, included only S1 and a modified version of S2. Session 1 (S1) included assessments of joint flexibility, muscle electrostimulation (H-reflex and PAD), muscle strength, and the tonic stretch reflex threshold (Valadão et al., 2021). Session 1 was the session relevant to the case study performed.

The exercise training intervention was completed as part of the EXECP project and was finished prior to the commencement of this study. The intervention information is included as it is relevant to the current studies research questions. The exercise intervention consisted of strength training, gait training and flexibility training. The strength training program included five multi-joint and five single-joint exercises, designed to balance strength development and motor control while minimizing compensatory movements. Two alternating session designs (A

and B) targeted the lower limbs and trunk with 7–10 exercises per session. Sessions alternated in AB or ABA/BAB patterns, depending on training frequency. Exercises for the ankle plantar flexors included seated and standing calf raises, while dorsiflexors were trained using resistance bands or manual resistance during hip extension exercises. Upper leg training incorporated leg press, squats, and knee flexion/extension, with unilateral exercises addressing limb imbalances. Trunk training focused on isometric holds, such as hollow rocks and Roman chair exercises. Exercise sequencing was varied to promote participant autonomy and well-being. Fatigue and quality were managed by avoiding back-to-back exercises targeting the same muscle group. Trainers ensured proper exercise order and intensity. Training loads adhered to guidelines from the ACSM and NSCA for cerebral palsy. The 12-week program was divided into three 4-week blocks see table 3 from Valadao et al., 2021 for specific information.

1. Block 1: Three sets of eight repetitions at 50% 1RM, with 3-second concentric and eccentric phases, 60 seconds rest between sets, and no rest between repetitions.
2. Block 2: Same volume, but with increased intensity—concentric duration reduced to 1 second, and rest extended to 90 seconds.
3. Block 3: Increased sets (four), reduced repetitions (six), and higher intensity with rapid concentric movements and 2-second eccentric phases.

This progression aimed to improve strength, motor control, and power output. Exercise plans, progression details, and session structures are summarized in an accompanying table 3 below.

TABLE 3. Training progression from (Valadão et al., 2021).

Week	Volume	Load	Movement Duration (s)	Rest (s)	Session A[a]	Session B[a]
1-4	3 sets of 8 repetitions	8 RM	3 concentric 3 eccentric	60	1 - Seated calf raise 2 - Seated dorsiflexion	1 - Seated machine knee flexion 2 - Seated machine knee extension
5-8	3 sets of 8 repetitions	8 RM	1 concentric 3 eccentric	90	3 - Standing calf raise 4 - Hip flexion 5 - Seated horizontal leg press	3 - Hip flexion 4 - Standing calf raise 5 - Seated horizontal leg press
9-12	4 sets of 6 repetitions	6 RM	1 concentric 2 eccentric	90	6 - Roman chair trunk extension 7 - Squat	6 - Isometric hollow rocks 7 - Squat

1 = ballistic muscle action; RM repetition maximum; [a] = each session has a minimum of 7 exercises and a maximum of 10 (i.e. all exercises)

Gait training was conducted using a portable mechanical treadmill with an incline of 6° or 7.3° (Vida XL, Venlo, Netherlands). Participants were instructed to walk at a comfortable pace, avoiding toe walking and aiming for heel strike. A non-motorized treadmill was chosen because its belt moved through hip extension and ankle plantarflexion, promoting proper muscle activation patterns. Verbal feedback was provided during the warm-up to improve gait quality, with participants allowed to stop and rest as needed. Each session included 5-10 minutes of gait training, and participants were given a treadmill to use at home, with the expectation of walking at least 10 minutes daily throughout the intervention.

Flexibility training involved four sets of 45 seconds of passive-static stretching at the pain threshold for each muscle group identified as tight in the pre-tests. Hip flexors were stretched in the Modified Thomas Test position, with one leg in full hip flexion and the other in hip extension. The trainer applied hip extension torque at the distal thigh, with the knee either fully flexed (two-joint stretch) or relaxed (one-joint stretch). The seated butterfly stretch targeted the hip adductors, with the participant's back supported against a wall, hips externally rotated, knees flexed, and soles of the feet in contact. The trainer applied pressure on the thighs to induce hip abduction. Hamstrings were stretched in supine position by flexing the participant's hip to about 90° and applying knee extension torque to the posterior shank. No stretching was performed on the triceps surae due to two reasons: 1) the difficulty of stretching all muscles bilaterally within a 90-minute session, and 2) the inclined gait training already provided a passive dynamic stretch to the ankle joint. Therefore, the focus was on the other muscle groups to assess the impact of the gait intervention on flexibility.

4.4 Passive muscle stretch measurement protocol

The passive muscle stretch measurement protocol was performed a total of four times with two pretests (Pre1 and Pre2) and two post-tests (Post1 and Post 2), spaced three months apart with two three-month control periods between pre1 and pre2 and post 1 and post 2. It used the following protocol to collect EMG data of the soleus and medial gastrocnemius was evaluated using the TSRT and calculated using the previously described SRT latency corrected method suggested by (Valadão et al., 2022). The protocol was performed a total of four times with two pretests (Pre1 and Pre2) and two post-tests (Post1 and Post 2), spaced three months apart with two three-month control periods between pre1 and pre2 and post 1 and post 2 . The participant was positioned with the knee fully extended, and stretches were applied to the triceps surae by

a motor-driven dynamometer (Neuromuscular Research Center, University of Jyväskylä, Finland). The range of motion of passive ankle dorsiflexion was from 20° of plantarflexion to 0° at four angular velocities (55, 110, 210, and 291°/s) The velocities were applied in a pseudo-randomized and balanced sequence, with 10 stretches conducted at each velocity and a rest interval of 2.6–2.9 seconds between stretches (Valadão et al., 2021). EMG data were collected and analysed to identify the joint angle at which the TSRT occurred. In this case study the SRT latency corrected method was used to include an individual h-reflex latency correction to identify the joint angle, as suggested in the aims of the EXECP study. This case study used the collected EMG data from the passive muscle stretch protocol from EXECP to calculate the TSRTcorrected.

4.5 EMG data collection and setup

The EMG data collection was performed a total of four times with two pretests (Pre1 and Pre2) and two post-tests (Post1 and Post 2), spaced three months apart with two three-month control periods between pre1 and pre2 and post 1 and post 2. For both the passive muscle stretch protocol and the h-reflex protocol. In this study EMG activity was recorded from the Sol, MG muscles using self-adhesive electrodes (Blue Sensor N, Ag/AgCl, 0.28 cm^2; Ambu, Ballerup, Denmark), with a ground electrode placed on the tibia. Electrode placement and skin preparation were conducted following SENIAM guidelines previously mentioned by (Hermens et al., 2000). The electrodes were positioned on the muscle belly, aligned with the underlying fibre direction, with a 20-mm interelectrode distance. EMG signals were amplified and high pass filtered (1000X, 10 Hz) using a preamplifier (NL824; Digitimer, Welwyn Garden City, UK) and subsequently bandpass filtered (10 Hz to 1 kHz) by a differential amplifier (NL900D/NL820A; Digitimer Ltd., UK). The signals were acquired on a personal computer at a sampling rate of 5 kHz using a 16-bit A/D converter (CED Power 1401; Cambridge Electronics Design, Cambridge, UK). This case study analysed the EMG data collected from the Sol and MG, to identify the TSRT using Spike2 from Cambridge Electronics Design, Cambridge, UK (Valadão et al., 2021).

4.6 H-reflex recruitment protocol and setup

The H-reflex recruitment protocol was performed to identify the H-reflex and outline the recruitment curve. The protocol was performed a total of four times with two pretests (Pre1 and Pre2) and two post-tests (Post1 and Post 2), spaced three months apart with two three-month control periods between pre1 and pre2 and post 1 and post 2 (Valadão et al., 2021). H-reflexes and M-waves were evoked in the soleus and medial gastrocnemius muscles by percutaneous electrical stimulation of the tibial nerve while the participant lay in a prone position. A single rectangular pulse (1 ms) was delivered using a constant-current stimulator (DS7AH; Digitimer, UK). A circular cathode with a pickup area of 0.77 cm^2 (Unilect4535M, Ag/AgCl; Unomedical, Redditch, UK) was placed over the tibial nerve at the popliteal fossa, and an oval-shaped, 5.1 × 10.2 cm anode (V-trodes; Mettler Electronics, Anaheim, CA) was positioned above the patella. The stimulation site providing the greatest amplitude of evoked responses in SOL and MG was initially identified using a handheld cathode electrode, which was later replaced by a self-adhesive electrode. The full recruitment curve was obtained by starting with a stimulation intensity of 3 mA and increasing in 0.5–2 mA steps (using smaller steps near the maximum H-reflex) at 0.1 Hz until the maximal M-wave was achieved. Peak-to-peak values for the maximum H-reflex and M-wave were then computed (Valadão et al., 2021).

4.7 H-reflex latency calculation protocol

The H-reflex latency calculation protocol was as such, H-reflex latency initially calculated using MATLAB (R2023a, The MathWorks Inc, Natick, United States). H-reflex latency calculation was performed using the following steps see the figure 14 below. The first red circle represents the stimulation point, while the m-wave spans the second magenta asterisk to the third magenta asterisk. The H-reflex is identified by the first peak in EMG activity. As shown in the figure 14, the fluorescent green circle marks the start of the H-reflex. To calculate latency, the difference in the position from the stimulation point (red circle) to the fluorescent green point was calculate.

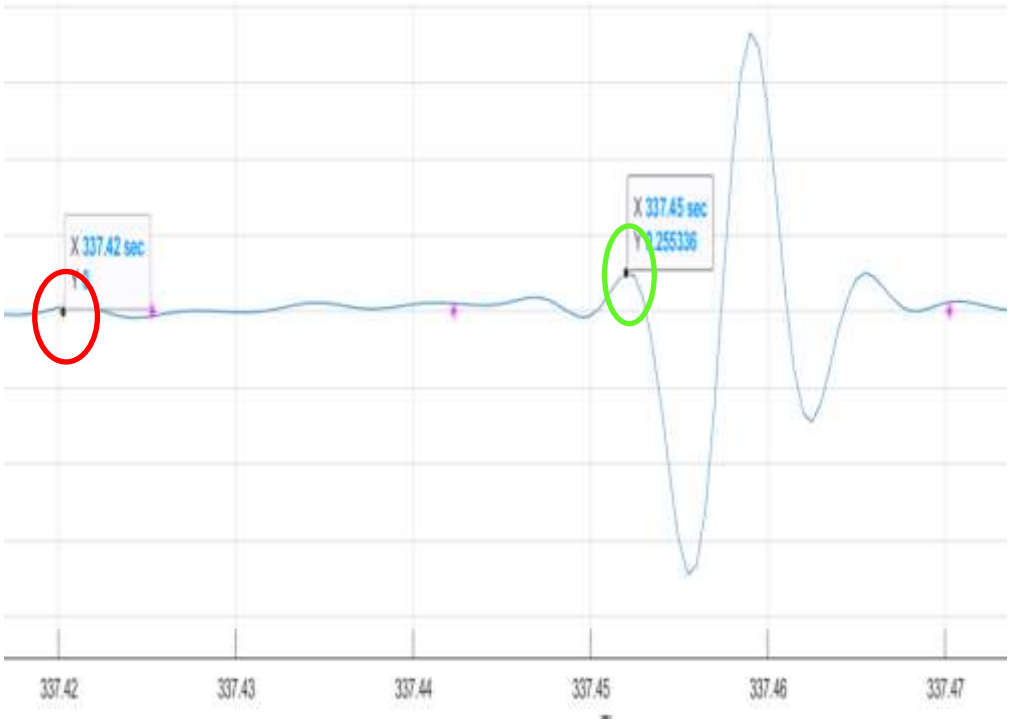

FIGURE 14. Individual H-reflex latency calculation example for the case study subject. The red circle is the stimulation point the green circle the beginning of the h-reflex.

4.8 Data analysis preparation

Stretch reflex threshold EMG data was for this case study was analysed using Spike2 software from Cambridge Electronics Design, Cambridge, UK. The relevant data channels—medial gastrocnemius (4), soleus (3), and angle (2) were selected. The angle axis was zeroed for each file to ensure values ranged between 20 and 0 degrees. This was done through the channel information screen by entering the appropriate axis offset value. This process was repeated for all data sets to standardize the angle range across the data sets and allow comparison of results.

4.9 Filtering of data in spike

The medial gastrocnemius and soleus channels were filtered using the IIR filter function in Spike2 (Cambridge Electronics Design, Cambridge, UK). A custom filter function was created using the band pass fourth order filter function. A Butterworth fourth order filter, with a low of 20Hz and high of 195Hz (20-195Hz) was used as previously suggested by literature and as per other EXECP analysis (Valadão et al., 2021). The filter was applied to both the medial gastrocnemius and soleus channels. New memory channels were created by clicking the apply

digital filter and it was saved as RealWave (Real, 4 bytes per point). The channels were renamed as MG for medial gastrocnemius and SOL for soleus to avoid confusion during analysis. The original MG and SOL channels were removed from the display window by right clicking and deselecting channels 3 and 4. The axes for MG and SOL were then optimized to display data on the same scale, ensuring comparability between the two channels. With these preparations complete, data analysis could proceed for the TSRTcorrected, as detailed in the following section.

4.10 The tonic stretch reflex threshold corrected analysis

The EMG data from the passive muscle stretching protocol from EXECP was used for this case study to calculate TSRTcorrected. TSRTcorrected was analysed in Spike2 from Cambridge Electronics Design, Cambridge, UK. First the steps in the above section were performed to filter the data. Once this was completed the TSRTcorrected data analysis commenced. Each time point (PRE1,PRE2,POST1 and POST2) was analysed individually and manually, for the 40 stretches. 10 stretches were analysed at each of the four velocities (55,110,200 and 291). Signals were not considered for analysis and were discarded if the signal to noise ratio was too high. This was chosen based on laboratory specific conditions from the NMRC at the university of Jyväskylä. This was done using the right click function, selecting the relevant Hcursor1, set position and entering the values. The TSRTcorrected was analysed by placing two vertical cursors were initially used to determine the stretch velocity. One cursor was placed at the start of the angle slope, and the other at the end. The positions of the cursors were then copied and subtracted from each other to calculate the stretch duration in seconds(s). Four distinct values were identified, corresponding to different velocities: 0.4s (55 º/s), 0.2s (110 º/s), 0.1s (200 º/s), and 0.68s (291 º/s). This process was repeated for each stretch to ensure consistency in the results. After identifying the stretch velocities, the TSRT-corrected EMG analysis was performed. Vertical cursors were used to determine the TSRT onset via EMG burst activity, labelled as SRTNON for non-corrected and SRTCORR for the latency corrected positions. The SRTNON cursor position was copied into the Windows calculator, and the individual H-reflex latency for the muscle was subtracted from this value. The result was then used to adjust the SRTCORR cursor position for precise identification of the TSRTcorrected position. A horizontal cursor was placed at the x, y intercept of the SRTCORR vertical cursor to mark the TSRTcorrected position, representing the joint angle of TSRTcorrected. This horizontal cursor value was then copied into the Excel file for analysis. The process was repeated for both the

SOL and MG muscles across all 40 stretches and the four time points (PRE1, PRE2, POST1, POST2). Care was taken to use the relevant H-reflex latency for each time point and muscle, as calculated in MATLAB (version R2023a, The MathWorks Inc, Natick, United States), following the latency correction procedure described earlier.

4.11 Statistical analysis

Since this was a case study no complex statistical analysis was performed instead the results were presented in the form of descriptive statistics. This included mean, standard deviation and coefficients of variation for each measurement point these were calculated using Microsoft excel (Microsoft Corporation, 2024). Mean relative change was also calculated to see the relative percentage of change between time points to allow for a slightly more in depth look at the potential changes over time due to the exercise intervention.

5 RESULTS

5.1 Pre-intervention results

The Pre 1 results in Table 4 highlight mean latency-corrected stretch reflex threshold characteristics for the soleus (SOL) and medial gastrocnemius (MG) muscles. For the SOL, reflex thresholds increased with velocity, peaking at 15.31° at 200°/s before decreasing slightly to 13.02° at 291°/s. Variability (CV%) also increased with velocity, ranging from 4.9% at 110°/s to a peak of 16.54% at 291°/s, indicating less consistent reflex responses at higher speeds. In contrast, the MG exhibited stable reflex thresholds across velocities, with mean values ranging from 19.36° at 55°/s to 18.91° at 291°/s. The MG also demonstrated remarkably low variability, with CV values progressively decreasing from 3.05% at 55°/s to 0.99% at 291°/s, reflecting highly consistent reflex activation.

TABLE 4. Pre1 TSRTcorrected joint angle data mean, standard deviation (SD) and coefficient of variation (CV), for soleus (SOL) and medial gastrocnemius (MG).

Muscle	Velocity (°/s)	Mean	SD	CV (%)
SOL	55	NA	NA	NA
SOL	110	9.59	0.47	4.9
SOL	200	15.31	1.52	9.93
SOL	291	13.02	2.15	16.54
MG	55	19.36	0.59	3.05
MG	110	19.38	0.22	1.15
MG	200	18.90	0.23	1.22
MG	291	18.91	0.19	0.99

The PRE2 data in Table 5 below presents the latency-corrected TSRT for the soleus (SOL) and medial gastrocnemius (MG) muscles across different velocities. In the SOL, the stretch reflex threshold decreased slightly as velocity increased, from 16.69° at 110°/s to 15.54° at 291°/s. Variability (CV%) was highest at 200°/s (10.13%) and slightly lower at 291°/s (9.84%), indicating greater inconsistency at higher velocities. For the MG, the latency-corrected TSRT remained stable across velocities, with mean values ranging from 17.56° at 55°/s to 18.79° at 110°/s. Variability (CV%) was highest at the lowest velocity, 55°/s (18.82%), and significantly lower at higher velocities, reaching its lowest at 110°/s (2.94%). The MG showed greater consistency across velocities, with variability decreasing as velocity increased, reflecting more reliable stretch reflex activation at higher speeds.

TABLE 5. Pre 2 TSRTcorrected joint angle data mean, standard deviation (SD) and coefficient of variation (CV), for soleus (SOL) and medial gastrocnemius (MG).

Muscle	Velocity (°/s)	Mean	SD	CV (%)
SOL	55	NA	NA	NA
SOL	110	16.69	1.28	7.68
SOL	200	16.13	1.63	10.13
SOL	291	15.54	1.53	9.84
MG	55	17.56	3.31	18.82
MG	110	18.79	0.55	2.94
MG	200	18.48	0.7	3.78
MG	291	17.8	0.83	4.66

5.2 Post-intervention results

The soleus TSRTcorrected increased with velocity, starting at 13.04° at 200°/s and slightly decreasing to 10.56° at 220°/s. No reflex activity was observed at 55°/s, and reflexes were intermittent at 110°/s, indicating an absence of consistent reflex responses during slower movements. Reflex variability (CV%) was highest at 110°/s (38.38%) and decreased to 20.7% at 220°/s, reflecting inconsistent reflex activation, particularly at lower velocities. In contrast, the medial gastrocnemius showed stable stretch reflex thresholds across all velocities, ranging from 19.14° at 55°/s to 18.72° at 220°/s. Variability was minimal, with CV values decreasing from 2.23% at 55°/s to 0.77% at 220°/s, demonstrating highly consistent reflex responses. Compared to the medial gastrocnemius, the soleus had greater variability and lower mean threshold values. The high variability, especially at lower velocities, suggests the soleus struggles to maintain consistent reflex activation. See table 6 below for all reference values at the Post 1 time point.

TABLE 6. Post 1 TSRTcorrected joint angle data mean, standard deviation (SD) and coefficient of variation (CV), for soleus (SOL) and medial gastrocnemius (MG).

Muscle	Velocity (°/s)	Mean	SD	CV (%)
SOL	55	0	0	0
SOL	110	7.59	2.91	38.38
SOL	200	13.04	3.85	29.55
SOL	291	10.56	2.19	20.7
MG	55	19.14	0.43	2.23
MG	110	19.27	0.17	0.89
MG	200	18.95	0.16	0.86
MG	291	18.72	0.14	0.77

At the POST2 measurement soleus showed an increase in the TSRTcorrected with velocity, reaching a peak of 12.31° at 200°/s, and then slightly declined to 11.4° at 291°/s see table 7 below. The variability (CV%) ranged from 12.39% at 110°/s to 18.66% at 200°/s, indicating moderate variability in reflex activation. This variability decreased at the highest velocity, with a cv of 10.31% at 291°/s. In contrast, the medial gastrocnemius demonstrated a highly stable reflex threshold across velocities, decreasing only slightly from 18.84° at 55°/s to 18.38°/s at 291°/s. The cv values were consistently low, ranging from 1.55% at 55°/s to just 0.3% at 200°/s, reflecting remarkable consistency in reflex responses. The soleus exhibited moderate variability in its stretch reflex thresholds, with higher cv values at intermediate velocities, which aligns with its role in adapting to postural and low-velocity demands. On the other hand, the medial gastrocnemius demonstrated high stability with minimal variability, particularly at higher velocities (cv = 0.3% at 200°/s). See table 7 and figure 15 below for reference values for the Post 2 time point.

TABLE 7. Post 2 TSRTcorrected joint angle data mean, standard deviation (SD) and coefficient of variation (CV), for soleus (SOL) and medial gastrocnemius (MG).

Muscle	Velocity (°/s)	Mean	SD	CV (%)
SOL	55	0	0	0
SOL	110	10.74	1.33	12.39
SOL	200	12.31	2.3	18.66
SOL	291	11.4	1.18	10.31
MG	55	18.84	0.29	1.55
MG	110	18.59	0.37	1.99
MG	200	18.51	0.06	0.3
MG	291	18.38	0.12	0.65

Relative change was calculated for the SOL and MG at two time points, between the PRE2 measurement and POST1 measurement that is immediately following the twelve-week exercise intervention. As well as at POST1 and POST2 to see if there were any residual effects after three months see table 6 below for reference values. Soleus relative change was calculated for three velocities (110, 200, and 291°/s), as values were not available for 55 °/s due to no SRT activity at this velocity see table 6 for reference values. The PRE2-POST1 showed decreases in the mean joint angle at SRT, of -55% at 110°/s, -19% at 200°/s and -32% for 291 °/s. Medial gastrocnemius on the other hand showed a different profile of relative change in comparison to soleus. In contrast it showed change in the opposite direction from the Pre2-Post1 period, showing a change of between 3-9%. Results from table 8 suggest that the joint angle of the TSRTcorrected was earlier in the passive muscle stretch. Interestingly, the Post1-Post2 period saw a reduction in mean TSRT as highlighted in table 8 showing a range of change from -2% to -4%. Mean absolute change was also calculated to allow for a comparison see table 8 below. SOL showed the largest amount of mean absolute change in the Pre2-Post1 period with values of 9.1°(110°/s), 3.09°(200°/s), and 4.98°(291°/s) respectively. In the Post1-Post2 period the absolute change was mostly in the opposite direction, for with values -3.15°(110°/s), and -0.84°(291°/s). The 200°/s still showed a reduction in its TSRT of 0.73°. In contrast the medial gastrocnemius values showed values of -1.58°(55°/s) -0.48°(110°/s), -0.47°(200°/s), and -0.92°(291°/s) at the Pre2-Post1 time point. In the Post1-Post2 time point medial gastrocnemius values were 0.30°(55°/s) 0.68°(110°/s), 0.44°(200°/s), and 0.34°(291°/s).

TABLE 8. Soleus (SOL) and medial gastrocnemius (MG) mean relative change for TSRTcorrected at four velocities.

Velocity	SOL Pre2-Post 1	SOL Post1- Post2	MG Pre 2-Post1	Post1- Post2 MG
55	NA	NA	9%	-2%
110	-55%	42%	3%	-4%
200	-19%	-6%	3%	-2%
291	-32%	8%	5%	-2%

TABLE 9. Soleus (SOL) and medial gastrocnemius (MG) mean absolute change for TSRTcorrected at four velocities.

Velocity	SOL PRE2-POST1	SOL POST1- POST 2	MG PRE2-POST1	MG POST1- POST 2
55			-1.58	0.30
110	9.10	-3.15	-0.48	0.68
200	3.09	0.73	-0.47	0.44
291	4.98	-0.84	-0.92	0.34

5.3 Summary of results across all time points

The study evaluated TSRTcorrected for the soleus and medial gastrocnemius across four time points (Pre1, Pre2, Post1, Post2) and four velocities. Soleus thresholds increased with velocity up to intermediate speeds and then declined slightly at higher velocities. Variability (CV%) was higher for the soleus across all phases, with the highest value observed at 110°/s during Post1 (38.38%). Variability decreased during Post2, indicating reduced variability after the intervention. Medial gastrocnemius thresholds remained stable across all velocities and phases, with minor reductions at higher velocities. Variability was consistently low, with the lowest value recorded at 200°/s during Post2 (0.3%). Soleus showed larger relative and absolute change values for the TSRTcorrected at different velocities. See tables 8 and 9 for reference. Whether they are statistically significant would need to be confirmed via more complex statistical analysis. See figures 15 and 16 below for a graphical representation of mean TSRTcorrected joint angles for medial gastrocnemius (MG) and soleus (SOL) at PRE1 (P1), PRE2 (P2), POST1 (PO1) and POST2 (PO2) at four velocities and time points as a summary.

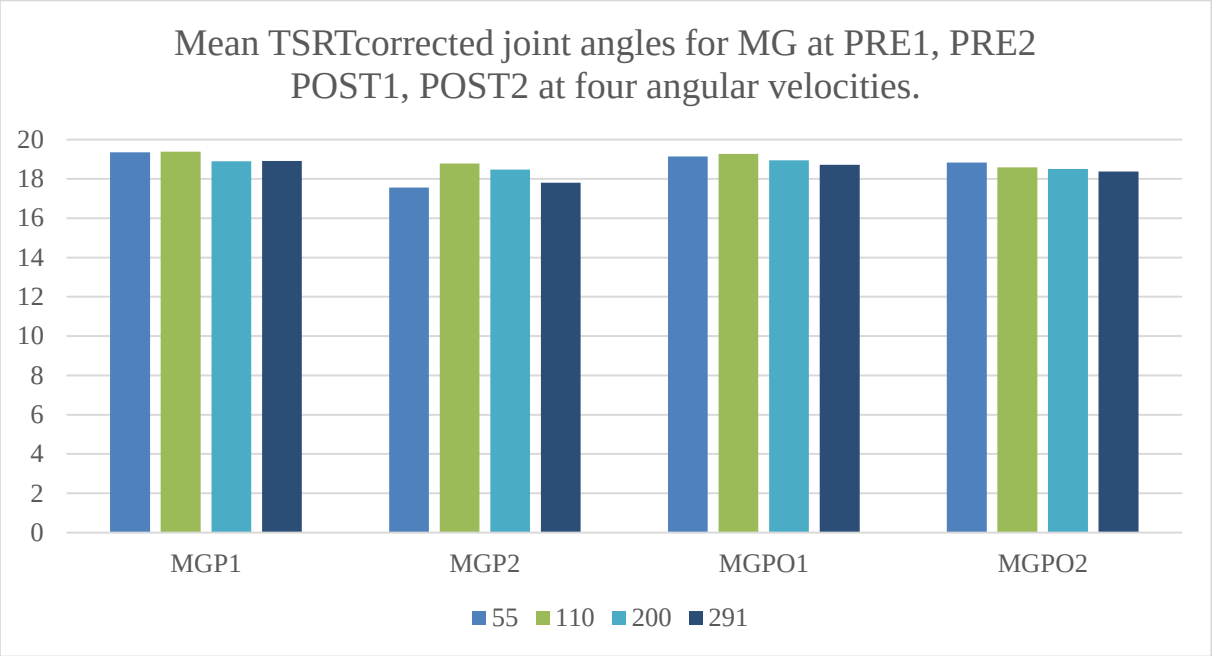

FIGURE 15 . Mean TSRTcorrected joint angles for medial gastrocnemius (MG) at PRE1 (P1), PRE2 (P2), POST1 (PO1) and POST2 (PO2) at four velocities (55,110,200 and 291 degrees per second).

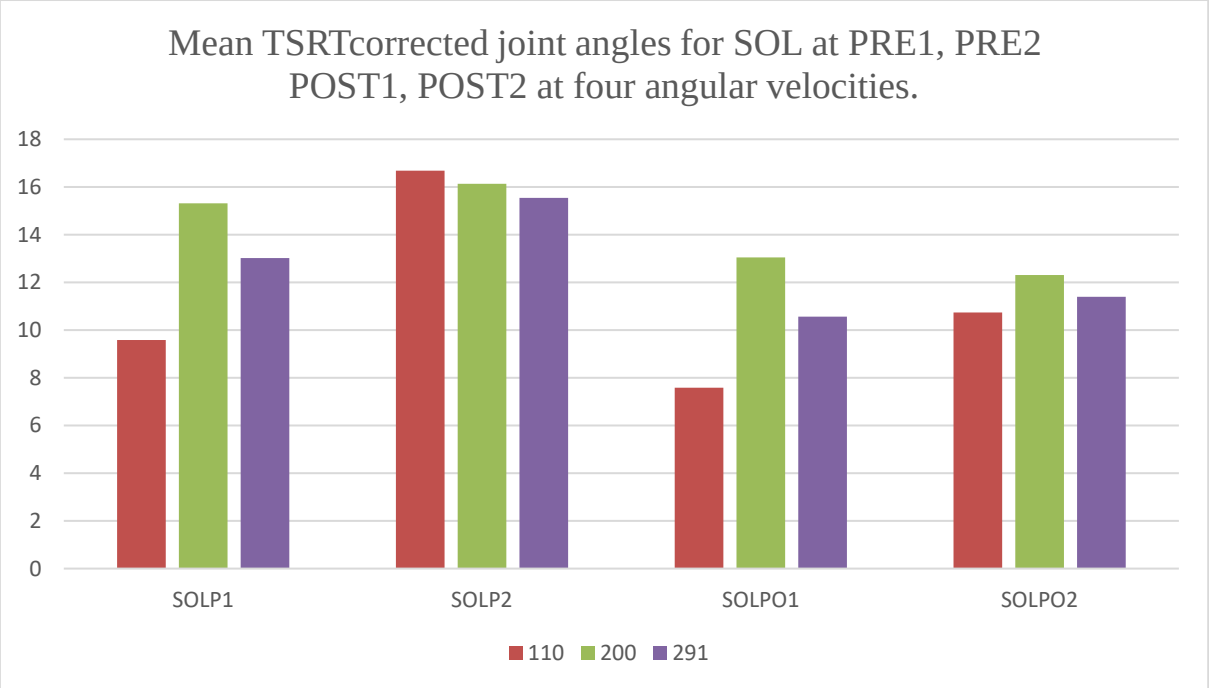

FIGURE 16 . Mean TSRTcorrected joint angles for soleus (SOL) at PRE1 (P1), PRE2 (P2), POST1 (PO1) and POST2 (PO2) at four velocities (55,110,200 and 291 degrees per second).

6 DISCUSSION

The primary research questions were 1) is the TSRTcorrected velocity dependent in this case study subject for the soleus and medial gastrocnemius? 2) does TSRTcorrected for the soleus and medial gastrocnemius muscles vary across different stretch velocities (55, 110, 200, and 291°/s) and 3) at the four time points (pre1, pre2, post1 and post2)? Based on the results presented above it appears that the medial gastrocnemius did not show velocity dependence in its TSRT. This is highlighted by the fact that TSRT were identified at all velocities across all time points. The soleus on the other hand can neither be confirmed nor rebutted. But as the TSRT was not present in the 55°/s sequences at any time point, furthermore the 110°/s returned very few TSRT overall, it is plausible that it showed velocity dependence. It did however show significant TSRT in the 200, and 291°/s sequences at all time points. However, we cannot draw any strong conclusions as a more thorough statistical analysis needs to be performed. The second research question does TSRTcorrected for the soleus and medial gastrocnemius muscles vary across different stretch velocities (55, 110, 200, and 291°/s) is difficult to answer due to the lack of statistical testing. Soleus does show more variability than medial gastrocnemius but whether it is statistically significant can at this point not be elucidated for the soleus or medial gastrocnemius. The third research question, 3) does TSRTcorrected for the soleus and medial gastrocnemius muscles vary across four time points (pre1, pre2, post1 and post2)? Is also hard to confirm with the statistical methodology used in this case study. However, the soleus does show moderate to large relative change values (-55,-19,and -32%) respectively between the Pre2-Post1 time point suggesting some effect of the intervention on the TSRTcorrected at the four time points, see table 6 above for reference. Interestingly the 200°/s shows a further decrease in relative change at the Post1-Post2 period. Suggesting some further reduction in the TSRT at this velocity. This would need to be confirmed with more rigorous statistical analysis. Absolute change in the soleus also appears to be relatively large as suggested by the values in the Pre2-Post1 period with values of 9.1°(110°/s), 3.09°(200°/s), and 4.98°(291°/s). The statistical significance would need to be confirmed by a more complex data analysis. The medial gastrocnemius on the other hand showed much lower relative change values and ranging from 3-9% percent in a positive direction suggesting an increase in the TSRT joint angle between the Pre2-Post1 period. Interestingly the values from the Post1-Post2 period suggest that the TSRT began to normalise as represented by the negative values ranging from -2 to -4.

The findings from this case study do help to reveal distinct functional roles and neuromechanical properties of the soleus and medial gastrocnemius muscles, evident in their TSRTcorrected joint angle profiles. The soleus had lower mean joint angles, indicating earlier reflex activation and greater sensitivity to stretch, aligning with its primary function in postural stability and controlled movement. This sensitivity is supported by its composition of slow-twitch (type I) fibres, which enable rapid stabilization during standing and walking to maintain balance and counteract perturbations (Sinkjaer et al., 1996). Conversely, the medial gastrocnemius showed higher mean joint angles at the TSRT, indicating a delayed reflex onset. This delay facilitates high-speed, forceful movements like running and jumping by allowing greater joint motion for optimal force generation before reflex activation. Its higher proportion of fast-twitch (type II) fibres reinforces its role in dynamic, explosive tasks (Armand et al., 2016). It is well established that resistance training, when appropriately structured, can promote muscle hypertrophy and enhance force and power in children and adolescents with CP. These interventions may also play a critical clinical role in increasing fascicle length, which is important for reducing contractures and improving muscle torque–angle and torque–velocity properties (Moreau & Lieber, 2022). However, the precise mechanisms driving changes in muscle size and fascicle length are not fully understood. Future research with adequately powered studies and large sample sizes is needed to address these uncertainties, enabling clinicians to design targeted rehabilitation protocols to achieve specific muscle functional outcomes (Moreau & Lieber, 2022).

TSRTcorrected variability also differed between the muscles. The soleus exhibited greater variability, particularly at moderate velocities (e.g., 200°/s, CV = 10.12%), reflecting its adaptability to diverse postural and dynamic demands. This variability balances its stabilizing role with responsiveness to changing mechanical conditions. In contrast, the medial gastrocnemius displayed low variability across all velocities, with the most consistent responses observed at 291°/s (CV = 1.07%). This consistency highlights its reliability during high-speed movements, where precise reflex activation is critical. Velocity influenced reflex activation patterns differently in each muscle. For the soleus, reflex threshold joint angles peaked at 200°/s and decreased at 291°/s. This suggests an adaptive mechanism at moderate velocities that permits greater joint motion, while earlier activation at higher velocities helps prevent overstretching and maintains stability (Sinkjaer et al., 1996). The medial gastrocnemius exhibited consistent reflex thresholds across velocities (17.53°–18.01°), demonstrating its ability to support rapid movements without significant changes in reflex sensitivity. This

stability aligns with the muscle's greater stiffness and faster contraction capabilities (Armand et al., 2016).

To summarise the relative mean change data from table 6 suggests that there may have been a positive effect on TSRTcorrected mean joint angle due to the exercise intervention between the PRE2 and POST 1 measurements. Between the POST 1 and POST 2 time point the values returned towards baseline for the 110°/s and 291°/s SRT's suggesting a normalisation or reduction in the beneficial effects of the intervention once it ceased in this case study subject. Interestingly, the 200°/s SRT mean showed a continued decrease of -6% suggesting further reduction in the SRT at the POST2 measurement. These values must be interpreted cautiously as they have not undergone statistical analysis. However, they provide some promise regarding the effectiveness of the EXECP intervention on reducing SRT which may be beneficial for stretch hyper-reflexia. The post intervention period may have also seen a reduction in the benefits, but some residual effects may be leftover. To confirm these a more rigorous statistical analysis should be performed on the study cohort.

Overall, the soleus is characterized by heightened sensitivity and variability, suited to low-intensity, controlled tasks. In contrast, the medial gastrocnemius prioritizes stability and delayed reflex activation, optimized for high-speed, dynamic activities. The soleus activates early to stabilize and prevent falls during slow movements (Sinkjaer et al., 1996), while the medial gastrocnemius's delayed reflex reduces interference, enabling powerful performance in activities such as sprinting and jumping (Kalkman et al., 2018).

6.1 Potential effects of the exercise intervention

Several potential mechanisms could explain the changes observed in muscle response following the exercise intervention. These mechanisms would likely relate to neural adaptations, muscle fibre remodelling, changes in muscle stiffness, and modifications in the stretch reflex pathway. The soleus exhibited heightened sensitivity to stretch, which could be due to enhanced proprioceptive input and better coordination between the muscle spindles and motor neurons. Exercise, particularly those focused on balance and postural control (resistance or functional training), can improve neural efficiency, enhancing the ability of the muscle to respond quickly to perturbations. Exercise can lead to CNS adaptations that improve motor control and response times. For example, increased neural drive or improved synchronization of motor units could

contribute to the earlier reflex activation seen in the soleus, helping to stabilize the body during dynamic movements like walking or standing (Enoka, 2015). The soleus, which is predominantly composed of slow-twitch fibres, is important in maintaining posture. Exercise interventions, especially those involving endurance or low-intensity tasks, may increase the proportion or the endurance of type I fibres, leading to improved performance in stabilization tasks. These fibres are more sensitive to reflex activation, supporting the observed results. In the medial gastrocnemius, which is primarily composed of type II fibres, high-intensity exercise, particularly explosive or sprint training, could lead to an increase in the number or size of these fibres (McArdle et al., 2010). This would result in enhanced force production, supporting the muscle's delayed reflex activation, as greater joint motion would be needed to generate maximal force before the reflex response occurs (A. J. Blazevich, 2006).

The most recent paper from Valadao et al., 2024, showed that the EXECP intervention significantly improved thigh muscle strength. Knee extensors (KE) showed a 19% increase in peak torque, 31% in the rate of force development, and 24% in concentric angular impulse. Knee flexors (KF) exhibited even greater gains, with a 31% rise in peak torque and a 92% increase in concentric angular impulse. These gains align with previous studies reporting 12%–27% improvements in KE torque and 25% increases in concentric torque. KF improvements likely reflect enhanced motor control as well as strength. However, shank muscle strength (PF and DF) did not improve significantly, possibly due to suboptimal training loads and testing protocols that limited adaptation. Trunk muscle strength improvements followed expected patterns. For extensors, load increases reached 900%, as 12 participants achieved maximum exercise duration (60 s) early in the intervention. Flexor strength gains involved a 33% increase in hold duration and weight adjustments, which were more challenging due to long resistance arms. Curve width, indicating steady torque output, did not improve for any muscle group, likely due to the intervention's initial training load structure. Overall, psychological factors, such as willingness to exert maximum effort, may have influenced strength gains, as suggested by the 10-fold increase in trunk extension weight (Valadão et al., 2024).

Muscle and or tendon stiffness can influence the stretch reflex threshold, with more stiffness contributing to a higher threshold for reflex activation (Lieber & Fridén, 2019). Exercise interventions that focus on strength training or plyometrics can increase the stiffness of the muscle-tendon unit, which may contribute to the medial gastrocnemius's consistent stretch reflex threshold at higher velocities (A. Blazevich & Blazevich, 2017). A stiffer muscle can

better resist rapid stretching and will need greater joint motion to activate the reflex. Tendon stiffness, which can also be modified by exercise, may affect how quickly muscles are stretched and how efficiently they generate force. Increased tendon stiffness may reduce the amount of stretch needed to activate the reflex in the medial gastrocnemius, while also providing more efficient force transmission during dynamic movements like running and jumping (Enoka, 2015). The most recent paper from Valadao et al., 2024, showed that the EXECP intervention improved joint flexibility by a mean of 5° in passive knee and hip extensions, a statistically significant but functionally minimal change achieved with just 6 minutes of weekly training. It remains unclear whether the gains stemmed from sensorimotor adaptations (increased stretch tolerance) or structural changes in the muscle-tendon unit. Evidence suggests that flexibility training alone is unlikely to induce mechanical changes in individuals with CP due to limited muscle growth capacity and hindered sarcomere genesis. Given the lack of strong evidence supporting stretching for CP, therapeutic focus should shift toward proven interventions like strength and gait training. Importantly, the intervention showed no negative impact on joint flexibility, addressing a common concern among health practitioners (Valadão et al., 2024).

Exercise interventions, particularly those that emphasize coordination or proprioception could enhance the integration of sensory inputs (from muscle spindles, Golgi tendon organs) and motor outputs. This could lead to improved timing and modulation of the stretch reflex, as seen in the soleus, which showed earlier reflex activation. The exercise could lead to changes in the stretch reflex threshold, influenced by adaptations in spinal cord processing. With regular, targeted training, reflex thresholds may shift to allow for more optimal muscle activation patterns, which could explain the reduced variability in the medial gastrocnemius and its ability to maintain consistent reflex thresholds (Lundy-Ekman & Weyer, 2022). Exercise could lead to more efficient recruitment of motor units in both the soleus and medial gastrocnemius. This may manifest as a faster response time (for the soleus) or more controlled activation patterns (for the medial gastrocnemius), allowing for the observed changes in reflex sensitivity and threshold (Enoka, 2015).

The variability in reflex thresholds and timing could also be influenced by the velocity of movement, as observed in the soleus. Exercises that challenge the muscles at different movement speeds, such as resistance training with varying tempos or agility drills, could enhance the muscle's ability to modulate its reflex response at different velocities, contributing to the observed peak in reflex thresholds at moderate speeds and decrease at higher speeds

(Valadão et al., 2022). Training interventions focusing on balance, such as standing on unstable surfaces, could enhance the sensitivity of the soleus for maintaining postural stability. Increased proprioceptive input, along with more efficient reflex activation, could explain why the soleus showed an earlier reflex onset and greater variability at moderate speeds likely a result of the muscle's adaptive role in dynamic postural control (Sinkjaer et al., 1996). The medial gastrocnemius's delayed reflex activation could be linked to an exercise-induced adaptation that optimizes muscle control in explosive movements. High-velocity training that involves jumping, sprinting, or other fast, powerful movements could improve the muscle's ability to generate force before reflex activation kicks in, leading to a more efficient performance during dynamic actions (McArdle et al., 2010).

Furthermore, the most recent paper published by Valadao et al., 2024 found the following results. The EXECP study observed a significant improvement in motor function, with the group mean 6MWT distance increasing by 33 m (Post1) and 44 m (Post2) compared to pretests. These changes exceed the typical test-retest variability (±43 m), indicating clinical significance. Similar results were seen in Gillet et al.'s study with younger participants (mean age 20 years) combining strength and anaerobic training, while Kirk et al.'s strength-only program for older participants (mean age 36 years) did not improve 6MWT despite gains in gait kinematics and muscle strength. Differences in training protocols, particularly the inclusion of motor coordination exercises, may explain these disparities. Regarding GMFM scores, nine participants achieved a clinically significant improvement of at least two points, with functional gains such as climbing stairs with alternating feet. However, smaller improvements, like balancing on one foot for a few seconds longer, may be less functionally impactful. This underscores the importance of evaluating both quantitative and qualitative outcomes to determine the relevance of such changes (Valadão et al., 2024).

In summary, the observed changes in stretch reflex thresholds and muscle activation are likely due to a combination of neural adaptations, changes in muscle fibre composition and stiffness, improved sensory-motor integration, and optimized muscle recruitment strategies. Exercise interventions tailored to specific functional needs such as postural stability for the soleus or explosive movement for the medial gastrocnemius could drive these mechanisms, leading to the enhanced performance seen in the results.

6.2 Strengths and weaknesses of the study

The strengths and weaknesses of this study are varied. A key strength of this study is that it is one of the first of its kind to assess the latency corrected TSRT at several measurement points. That is its longitudinal nature and allows for more insight into the effectiveness of the exercise intervention immediately pre and post intervention and at a three-month follow-up. A clear weakness is the fact that this was a case study and as such no strong conclusion can be drawn from the results, they may only help give us an idea of potential outcomes related to the exercise intervention and its effect on the TSRT. Another area which could be improved is the analysis of the latency corrected TSRT. It could be valuable to use multiple evaluators to assess the TSRT in this way to see what kind of repeatability there is in the methodology. How does the experience of the evaluator influence their identification of the TSRT. Some guidelines related to false positives or false negative results could also help to improve reliability when assessing the TSRT. Some other strengths of the study are the fact that the latency corrected TSRT was assessed at several time points and velocities. Which help it to identify whether there is a velocity dependent relationship or not. Furthermore, a more comprehensive statistical analysis procedure could help us highlight any significant results. Thus, highlighting whether the intervention was effective in reducing TSRT in spastic cerebral palsy.

7 CONCLUSION

In summary, the medial gastrocnemius shows greater consistency in both its stretch reflex threshold and variability across a range of velocities. This suggests that the medial gastrocnemius may be better suited for tasks requiring stable performance at varying speeds. In contrast, the soleus exhibits greater variability, particularly at moderate velocities, but tends to stabilize at higher speeds, likely reflecting its specialized function in maintaining postural stability during slow or controlled movements, which require more adaptability to changes in movement dynamics. These findings highlight the distinct functional roles of these muscles in dynamic activities and could inform approaches to rehabilitation or training that target specific velocity-dependent muscle behaviours. The soleus exhibited greater sensitivity and variability in stretch reflex activation, aligning with its postural and stabilizing role. In contrast, the medial gastrocnemius demonstrated stable and reliable reflex thresholds, supporting its function in dynamic, high-force movements. The exercise intervention appeared to improve reflex consistency in the soleus while maintaining stability in the medial gastrocnemius. However, as no statistical analysis was performed, we must be careful interpreting them as such, and further in-depth analysis is necessary. These findings emphasize the distinct neuromechanical roles of these muscles and the potential for targeted interventions to enhance motor control in individuals with cerebral palsy who exhibit spasticity.

8 REFERENCES

Barber, L., Barrett, R., & Lichtwark, G. (2012). Medial gastrocnemius muscle fascicle active torque-length and Achilles tendon properties in young adults with spastic cerebral palsy. *Journal of Biomechanics*, *45*(15), 2526–2530. https://doi.org/10.1016/j.jbiomech.2012.07.018

Bar-On, L., Kalkman, B. M., Cenni, F., Schless, S.-H., Molenaers, G., Maganaris, C. N., Bass, A., Holmes, G., Barton, G. J., O'Brien, T. D., & Desloovere, K. (2018). The Relationship Between Medial Gastrocnemius Lengthening Properties and Stretch Reflexes in Cerebral Palsy. *Frontiers in Pediatrics*, *6*, 259. https://doi.org/10.3389/fped.2018.00259

Blazevich, A., & Blazevich, A. J. (2017). *Sports biomechanics: The basics: Optimising human performance*. Bloomsbury Publishing.

Blazevich, A. J. (2006). Effects of physical training and detraining, immobilisation, growth and aging on human fascicle geometry. *Sports Medicine*, *36*, 1003–1017.

Castle, M. E., Reyman, T. A., & Schneider, M. (1979). Pathology of spastic muscle in cerebral palsy. *Clinical Orthopaedics and Related Research*, *142*, 223–233. https://doi.org/10.1097/00003086-197907000-00036

Enoka, R. M. (2015). *Neuromechanics of human movement 5th edition*. Human Kinetics.

Graham, H. K., Rosenbaum, P., Paneth, N., Dan, B., Lin, J.-P., Damiano, D. L., Becher, J. G., Gaebler-Spira, D., Colver, A., Reddihough, D. S., Crompton, K. E., & Lieber, R. L. (2016a). Cerebral palsy. *Nature Reviews Disease Primers*, *2*(1), 15082. https://doi.org/10.1038/nrdp.2015.82

Graham, H. K., Rosenbaum, P., Paneth, N., Dan, B., Lin, J.-P., Damiano, D. L., Becher, J. G., Gaebler-Spira, D., Colver, A., Reddihough, D. S., Crompton, K. E., & Lieber, R. L.

(2016b). Cerebral palsy. *Nature Reviews Disease Primers*, *2*(1), 15082. https://doi.org/10.1038/nrdp.2015.82

Hermens, H. J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology*, *10*(5), 361–374.

Howard, J. J., & Herzog, W. (2021). Skeletal Muscle in Cerebral Palsy: From Belly to Myofibril. *Frontiers in Neurology*, *12*, 620852. https://doi.org/10.3389/fneur.2021.620852

Kalkman, B. M., Bar-On, L., Cenni, F., Maganaris, C. N., Bass, A., Holmes, G., Desloovere, K., Barton, G. J., & O'Brien, T. D. (2018). Muscle and tendon lengthening behaviour of the medial gastrocnemius during ankle joint rotation in children with cerebral palsy. *Experimental Physiology*, *103*(10), 1367–1376. https://doi.org/10.1113/EP087053

Kalsi, G., Fry, N. R., & Shortland, A. P. (2016). Gastrocnemius muscle–tendon interaction during walking in typically-developing adults and children, and in children with spastic cerebral palsy. *Journal of Biomechanics*, *49*(14), 3194–3199. https://doi.org/10.1016/j.jbiomech.2016.07.038

Levin, M. F., Piscitelli, D., & Khayat, J. (2024). Tonic stretch reflex threshold as a measure of disordered motor control and spasticity – A critical review. *Clinical Neurophysiology*, *165*, 138–150. https://doi.org/10.1016/j.clinph.2024.06.019

Lieber, R. L., & Fridén, J. (2019). Muscle contracture and passive mechanics in cerebral palsy. *Journal of Applied Physiology*, *126*(5), 1492–1501. https://doi.org/10.1152/japplphysiol.00278.2018

Lundy-Ekman, L., & Weyer, A. (2022). *Neuroscience: Fundamentals for Rehabilitation*. Elsevier. https://books.google.com.au/books?id=82GkzgEACAAJ

Matthiasdottir, S., Hahn, M., Yaraskavitch, M., & Herzog, W. (2014). Muscle and fascicle excursion in children with cerebral palsy. *Clinical Biomechanics*, *29*(4), 458–462.

McArdle, W. D., Katch, F. I., & Katch, V. L. (2010). *Exercise physiology: Nutrition, energy, and human performance*. Lippincott Williams & Wilkins.

Meyns, P., Van Gestel, L., Leunissen, I., De Cock, P., Sunaert, S., Feys, H., Duysens, J., Desloovere, K., & Ortibus, E. (2016). Macrostructural and Microstructural Brain Lesions Relate to Gait Pathology in Children With Cerebral Palsy. *Neurorehabilitation and Neural Repair*, *30*(9), 817–833. https://doi.org/10.1177/1545968315624782

Moreau, N. G., & Lieber, R. L. (2022). Effects of voluntary exercise on muscle structure and function in cerebral palsy. *Developmental Medicine & Child Neurology*, *64*(6), 700–708. https://doi.org/10.1111/dmcn.15173

Mukherjee, A., & Chakravarty, A. (2010). Spasticity mechanisms–for the clinician. *Frontiers in Neurology*, *1*, 149.

Nielsen, J. B., Petersen, N. T., Crone, C., & Sinkjaer, T. (2005). Stretch Reflex Regulation in Healthy Subjects and Patients with Spasticity. *Neuromodulation: Technology at the Neural Interface*, *8*(1), 49–57. https://doi.org/10.1111/j.1094-7159.2005.05220.x

Pagnozzi, A. M., Dowson, N., Doecke, J., Fiori, S., Bradley, A. P., Boyd, R. N., & Rose, S. (2017). Identifying relevant biomarkers of brain injury from structural MRI: Validation using automated approaches in children with unilateral cerebral palsy. *PLoS One*, *12*(8), e0181605.

Peeters, N., Hanssen, B., Bar-On, L., De Groote, F., De Beukelaer, N., Coremans, M., Van den Broeck, C., Dan, B., Van Campenhout, A., & Desloovere, K. (2023). Associations between muscle morphology and spasticity in children with spastic cerebral palsy.

European Journal of Paediatric Neurology, *44*, 1–8. https://doi.org/10.1016/j.ejpn.2023.01.007

Reid, L. B., Pagnozzi, A. M., Fiori, S., Boyd, R. N., Dowson, N., & Rose, S. E. (2017). Measuring neuroplasticity associated with cerebral palsy rehabilitation: An MRI based power analysis. *International Journal of Developmental Neuroscience*, *58*(1), 17–25. https://doi.org/10.1016/j.ijdevneu.2017.01.010

Reid, L. B., Rose, S. E., & Boyd, R. N. (2015). Rehabilitation and neuroplasticity in children with unilateral cerebral palsy. *Nature Reviews Neurology*, *11*(7), 390–400. https://doi.org/10.1038/nrneurol.2015.97

Rose, J., Haskell, W. L., Gamble, J. G., Hamilton, R. L., Brown, D. A., & Rinsky, L. (1994). Muscle pathology and clinical measures of disability in children with cerebral palsy. *Journal of Orthopaedic Research*, *12*(6), 758–768. https://doi.org/10.1002/jor.1100120603

Schiaffino, S., & Reggiani, C. (1996). Molecular diversity of myofibrillar proteins: Gene regulation and functional significance. *Physiological Reviews*, *76*(2), 371–423. https://doi.org/10.1152/physrev.1996.76.2.371

Sinkjaer, T., Andersen, J. B., & Larsen, B. (1996). Soleus stretch reflex modulation during gait in humans. *Journal of Neurophysiology*, *76*(2), 1112–1120.

Sloot, L. H., Bar-On, L., van der Krogt, M. M., Aertbeliën, E., Buizer, A. I., Desloovere, K., & Harlaar, J. (2017). Motorized versus manual instrumented spasticity assessment in children with cerebral palsy. *Developmental Medicine & Child Neurology*, *59*(2), 145–151.

Sloot, L. H., Weide, G., van der Krogt, M. M., Desloovere, K., Harlaar, J., Buizer, A. I., & Bar-On, L. (2021). Applying Stretch to Evoke Hyperreflexia in Spasticity Testing:

Velocity vs. Acceleration. *Frontiers in Bioengineering and Biotechnology*, *8*, 591004. https://doi.org/10.3389/fbioe.2020.591004

Smith, L. R., Lee, K. S., Ward, S. R., Chambers, H. G., & Lieber, R. L. (2011). Hamstring contractures in children with spastic cerebral palsy result from a stiffer extracellular matrix and increased *in vivo* sarcomere length. *The Journal of Physiology*, *589*(10), 2625–2639. https://doi.org/10.1113/jphysiol.2010.203364

Tirrell, T. F., Cook, M. S., Carr, J. A., Lin, E., Ward, S. R., & Lieber, R. L. (2012). Human skeletal muscle biochemical diversity. *Journal of Experimental Biology*, *215*(16), 2931–2931. https://doi.org/10.1242/jeb.077347

Valadão, P., Bar-On, L., Cenni, F., Piitulainen, H., Avela, J., & Finni, T. (2022). Revising the stretch reflex threshold method to measure stretch hyperreflexia in cerebral palsy. *Frontiers in Bioengineering and Biotechnology*, *10*, 897852. https://doi.org/10.3389/fbioe.2022.897852

Valadão, P., Cenni, F., Piitulainen, H., Avela, J., & Finni, T. (2024). Effects of the EXECP Intervention on Motor Function, Muscle Strength and Joint Flexibility in Individuals with Cerebral Palsy. *Medicine and Science in Sports and Exercise*, *56*(1).

Valadão, P., Piitulainen, H., Haapala, E. A., Parviainen, T., Avela, J., & Finni, T. (2021). Exercise intervention protocol in children and young adults with cerebral palsy: The effects of strength, flexibility and gait training on physical performance, neuromuscular mechanisms and cardiometabolic risk factors (EXECP). *BMC Sports Science, Medicine and Rehabilitation*, *13*(1), 17. https://doi.org/10.1186/s13102-021-00242-y

Verschuren, O., Peterson, M. D., Balemans, A. C., & Hurvitz, E. A. (2016). Exercise and physical activity recommendations for people with cerebral palsy. *Developmental Medicine & Child Neurology*, *58*(8), 798–808.