Kinetic relationships between ankle plantar flexor and hip flexor power during gait in mildly affected adults with spastic hemiplegic and diplegic cerebral palsy

A case series study based on a ballistic strength training rehabilitation program

Silje Marie Rydningen Torberntsson

Master Program in Health Science – Physiotherapy by Research

Department of Global Public Health and Primary Care

Autumn 2019 – Spring 2020

Preface and acknowledgements

First of all, I want to thank my supervisor Silje Mæland and my wonderful college in this project Beate Eltarvåg Gjesdal, who has been great support and made this project possible. I am so thankful for all your advices regarding the master thesis, but also all the good times we have spent together these two year. You spread lots of joy while working which has made it a pleasure to take part in the project. You are absolutely outstanding!

Secondly, I am grateful for the kindness and help from Lars Peder Vatshelle Bovim and Bård Erik Bogen in the rehabilitation laboratory Sim Arena, Western Norway University of Applied Science. I truly respect and appreciate everything I have learned from you.

Greetings to all the subjects participating in this project. Your effort and encouragement have been important to initiate future research in this field to improve rehabilitation options for individuals in the same position. Thank you so much!

And finally, thanks to my fantastic family and friends for always being supportive.

Bergen, May 2020
Silje Marie Rydningen Torberntsson

Content

Preface and acknowledgements 2

Content 3

Abstract 6

List of Tables 7

List of Figures 8

Abbreviations 9

Definition of concepts 10

Theoretical background 11

Chapter 1: Normal gait 12

1.1 Normal gait cycle 12
1.1.1 Spatiotemporal gait parameters 14
1.1.2 Joint gait kinematics 14
1.1.3 Joint gait kinetics 17

1.2 Kinetic analysis during in gait 18
1.2.1 Ankle joint structure and power generation 18
1.2.2 Hip joint structure and power generation 19
1.2.3 Ankle-hip ratio (A2/H3) 21

1.3 Management of gait deviations to improve ankle power generation in late stance 21
1.3.1 Ankle foot orthosis 22
1.3.2 Botulinum toxin type A 22
1.3.3 Surgical treatment 22
1.3.4 Traditional resistance training 23
1.3.5 Ballistic strength training 23

Chapter 2: Spastic cerebral palsy and gait characteristics 25

2.1 Pathology of spastic cerebral palsy 25
2.2.1 Gross motor function classification system 26
2.2.2 Spastic hemiplegic cerebral palsy 27
2.2.3 Spastic diplegic cerebral palsy 27

2.2 Gait cycle in spastic cerebral palsy 28
2.2.1 Spatiotemporal gait parameters 28
2.2.2 Joint gait kinematics 28
2.2.3 Joint gait kinetics 29

2.3 Kinetic analysis in spastic cerebral palsy during gait 29
2.3.1 Ankle power generation 29
2.3.2 Hip power generation 30
2.3.3 Ankle-hip ratio (A2/H3) 31

Chapter 3: Rationale and aims **32**

3.1 General aim *32*

3.2 Hypotheses *32*

Chapter 4: Material and methodology **33**

4.1 Case series study *33*

4.2 Ethical considerations *33*

4.3 Inclusion and exclusion criteria *34*

4.4 Study setting and sample *34*

4.5 Ballistic "Leg sled hop" intervention *35*

4.5.1. Exercise 1: Jump Squats on the leg sled 36

4.5.2 Exercise 2: Single leg hopping on the paretic leg 36

4.5.3 Exercise 3: Bounding on altering legs (jogging) 36

4.6 Experimental protocol *36*

4.6.1 Kinematic measures 38

4.6.2 Kinetic measures 40

4.6.3 Calibration 40

4.6.4 Performing gait trials 40

4.7 Analysis of gait data *41*

4.7.1 Calculation of joint kinematics 41

4.7.2 Calculation of joint kinetics 41

5.0 Manuscript following guidelines for authors of Elsevier Gait & Posture **42**

Chapter 6: Additional findings **59**

6.1 Peak ankle and hip power in late stance of gait *59*

6.2 Exercise log of ballistic strength training *61*

Chapter 7: Methodological and process considerations **62**

7.1 Case series study design *62*

7.2 Study setting and sample *63*

7.3 Confounding variables *64*

7.4 Outcome measures *64*

7.4.1 Three-dimensional gait analysis 64

7.4.2 Measurement error and considerations 65

7.5 Ballistic strength training *66*

7.5.1 Relevance of the exercises 66

7.5.2 Familiarization and time interval 67

7.5.3 Side-effects 68

7.6 Clinical perspectives to future research *68*

7.7 Summary and conclusion *69*

Chapter 8: References **70**

Chapter 9: Appendices **80**

Appendix 1: Approval – REK *80*

Appendix 2: Recruiting film and poster *81*

Appendix 3: Information letter and consent 82
Appendix 4: Ballistic strength exercises 85
Appendix 5: Borg Scale 6-20 89
Appendix 6: Training diary 90
Appendix 7: Organization of test days 91
Appendix 8: CGM lower body marker set 93
Appendix 9: Gait analysis protocol 94
Appendix 10: 3D capture – Registration form 98

Abstract

Background: In normal gait, the ankle plantar flexors provide most propulsive energy during push-off, with smaller contribution of hip flexors. However, the interplay between these two joints remains unclear in spastic cerebral palsy. The objective of this study was to evaluate the kinetic relationship between the ankle plantar flexor and hip flexor power in late stance of gait (A2/H3) in mildly affected adults with spastic cerebral palsy. By implementing a ballistic strength training program, it was hypothesized that these exercises would exaggerate ankle plantar flexor power so the need for hip flexor power compensation would decrease, and thereby result in an increased A2/H3 ratio.

Method: Ten adults (35.7±12.6) with spastic hemiplegic and diplegic cerebral palsy, Gross Motor Function Classification System I-II, was recruited to attend an eight week ballistic strength training program mainly prescribed to most paretic limb. Three-dimensional gait analysis with a force plate was used to investigate the impact on ankle and hip power generation in push-off before, during and after intervention. At least three gait trials at self-selected speed was analyzed for each limb to calculate peak ankle and hip flexor power in the sagittal plane and relationship between them (A2/H3 ratio).

Results: 7 participants completed the study protocol. 6 out of these 7 participants increased A2/H3 ratio on the most paretic limb, while 5 increased on uninvolved limb. As expected, the change was more evident on the most paretic limb compared to the uninvolved limb.

Conclusion: Findings from this study provide a better understanding of the interplay between power patterns in the ankle and hip joint in spastic cerebral palsy with a possible implication to clinical practice. However, the results cannot direct any casual relationships between change in A2/H3 ratio and ballistic strength training. Until evidence is found, we assume that ballistic strength training is feasible to alter A2/H3 ratio in adults with spastic cerebral palsy, yet further analytic investigation is needed.

Keywords: Spastic cerebral palsy, adults, ankle/hip power ratio, ballistic strength training, gait

List of Tables

Table 1 Clinical conventions of joint angles in sagittal-, coronal-, and transverse plane.

Table 2 Inclusion and exclusion criteria.

Table 1 Included participants, characteristics, anthropometric measurements and diagnosis description *(scientific article).*

Table 2 Peak ankle and hip ratio (A2/H3 ratio) on both most paretic and uninvolved limb before, during and after ballistic strength training of the ankle plantar flexors *(scientific article).*

Table 3 Self-selected gait speed before, during and after ballistic strength training *(scientific article).*

Table 4 Kinetic graphs of ankle and hip power generation normalized for 100% of gait cycle for both right and left limb *(scientific article).*

Table 3 Peak ankle plantar flexor (A2) and hip flexor (H3) power generation values in the late stance phase of gait.

Table 4 Mean number of repetitions, slope and Borg scale (6-20) the participants expressed after each session with three different ballistic strength exercises.

List of Figures

Figure 1 Stance period: Initial contact and loading response refers to contact of forefoot with the ground, acceptance of weight onto standing limb and shock absorption (eccentric muscle contraction). Mid-stance is a phase of forward progression of the limb over standing foot (eccentric muscle contraction). Terminal stance is forward propulsion (concentric muscle contraction). Swing period: Initial swing is the period of acceleration of the swinging limb. Mid swing is a transitional period from acceleration to declaration of the swinging limb. Terminal swing is a period of declaration of the swinging limb.

Figure 2 Average power patterns of ankle in sagittal plane for 19 health subjects walking at mean natural cadence of 105 steps/min.

Figure 3 Average power patterns of the hip in sagittal plane for 19 health subjects walking at mean natural cadence of 105 steps/min.

Figure 4 DF weakness; A) toe drag, B) high steppage gait, C) hip hiking, D) circumduction. Excessive ankle dorsiflexion is represented by: E) prolonged heel contact, F) excessive knee flexion.

Figure 5 Ankle joint power graphs for 10 children with CP showing mean and standard deviation. Mean cadence was 131 steps/min.

Figure 6 Hip joint power graphs for 10 children with CP showing mean and standard deviation. Mean cadence was 131 steps/min.

Figure 7 The positions when preforming three different ballistic strength training exercises on the leg sled - exercise 1, 2 and 3 respectively.

Figure 8 Experimental protocol.

Figure 9 Each participant individual change in A2/H3 ratio on paretic limb after 8-weeks of ballistic strength training.

Abbreviations

3DGA	Three-dimensional gait analysis
ABD	Abduction
ACSM	American College of Sports Medicine
ADD	Adduction
AFO	Ankle foot orthosis
ASIS	Anterior superior iliac spine
BTX-A	Botulinum toxin – A
CGM	Conventional gait model
COM	Center of mass
CP	Cerebral palsy
DF	Dorsiflexion
EMG	Electromyography
EV	Eversion
FUNCAP-CP	Functional Capacity for walking in adults with Cerebral Palsy
GMFCS	Gross Motor Function Classification System
Hz	Hertz
INV	Inversion
Kg	Kilogram
M	Mean
Min	Minutes
N	Newtons
PF	Plantar flexion
PhD	Doctor of Philosophy
PSIS	Posterior superior iliac spine
QoL	Quality of life
RCT	Randomized controlled trials
ROM	Range of Motion
SCP	Spastic cerebral palsy
SD	Standard deviation
TBI	Traumatic brain injury
QTM	Qualisys track manager software
W	Watt

Definition of concepts

A1	Ankle plantar flexion power absorption at mid-stance.
A2	Ankle plantar flexion power generation during late stance.
A2/H3	Relationship between proportions of ankle plantar flexion (A2) and hip flexion (H3) during the push-off phase.
Cerebral palsy	Cerebral palsy describes a group of permanent disorder of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy, and by secondary musculoskeletal problems (1).
Concentric activity	Muscle length is shortening while generating positive power, accelerating the center of mass.
Coronal plane	Divides the body into posterior and anterior portions.
Eccentric activity	Muscles absorbs negative power through lengthening in an eccentric muscle contraction.
Gait	The manner in which walking is performed.
H1	Hip extension power generation in first half of stance.
H2	Hip flexion power absorption in mid-stance.
H3	Hip flexion power generation in late stance/initial swing.
Power	The product of the joint moment and joint angular velocity. It is measured in watts normalized to body weight. It determines the propulsive forces of the body.
Sagittal plane	Divides the body into left and right (longitudinal).
Transverse plane	Divides the body into cranial and caudal portions (horizontal).

Theoretical background

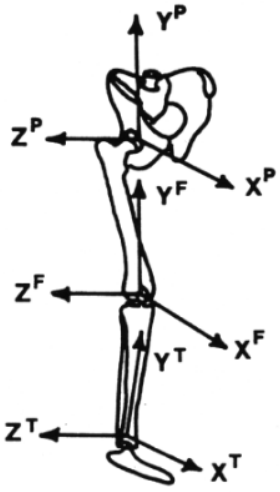
The ultimate goal in gait rehabilitation is to understand functional abilities in relation to limitations with the purpose of enhancing performance. In normal gait, most power is generated by the ankle plantar flexors during the late stance phase of gait, with smaller contribution of hip flexors at push-off (2–4). Yet, the interplay between these two joints are less recognized when applied to gait in spastic cerebral palsy (SCP). The hip flexors appears to be more active during the gait cycle in this population group due to reduced ankle plantar flexor power output (2,5–7). This suggest a complex gait pathology with compensatory tradeoff mechanisms between the hip and ankle which may progress to worse deficiencies with age. Alteration of ankle power generation has been recommended in several neurological disorders to improve functional capacity for walking (2,8–10). With this theoretical background, it is thought that adults with cerebral palsy (CP) may benefit from a training regime targeting the ankle plantar flexors on higher velocity to improve power generation (8,11).

In order to understand the neuromuscular and musculoskeletal gait pathology associated with SCP, it is fundamental to recognize the precise structure of joint- and limb biomechanics during normal gait. This thesis is structured into several chapters with smaller sub-sections explaining this phenomenon. The first chapter describes normal gait cycle mechanisms and clinical gait analysis with a detailed description of kinetic expressions of ankle and hip power and management of gait deviations. The second chapter contains a description of gait in SCP, with primary focus on reduction in ankle power output and A2/H3 ratio. The third chapter describes the rationale and aim with the project, followed by material and methodology in chapter four. In this section, study design and ethical considerations are presented. Further, the protocol for exercise intervention is described in detail followed by instrumentation for gait testing and procedure for data analysis. The scientific article is presented in chapter five. Additional findings that can possible be contributing causes of the results, yet not included in the scientific article are presented in chapter six. Lastly, chapter seven is methodological considerations and conclusion, followed by references and appendices in chapter eight and nine.

Chapter 1: Normal gait

1.1 Normal gait cycle

Gait is a complex repetitious motor skill of the lower limbs resulting in forward progression of the center of mass (COM) (12). The gait cycle is commonly divided into a number of distinct phases (figure 1), characterized by periods of loading and unloading. In general, the gait cycle is normalized to 100%, where the stance and swing phase last on average for 60% and 40%, respectively (13). The stance phase is defined as a period from heel contact to toe-off, where the reference limb is constantly in contact with the ground (12). It is normally subdivided into five phases of initial contact, loading response, mid-stance, terminal stance, and push-off (14). During stance phase, two important tasks of weight acceptance and single limb support is accomplished. The remaining 40% is the swing phase and is defined from toe-off to heel contact. In this phase, the reference limb is advancing forward without being in contact with the ground. The swing phase contains initial swing, mid-swing, and terminal swing (14). An efficient gait cycle requires dynamic stability in stance, pre-positioning of the foot, sufficient foot clearance during swing, adequate step length and energy conservation (15,16). The gait cycle and functional level of lower extremities in forward ambulation can be assessed in several ways. In clinical practice, visual observation is often used to evaluate movement disabilities and making subjective decisions regarding treatment management. However, an accurate objective quantification is usually required in research to identify and enhance gait pattern. Clinical evidence and literature support the efficacy of three-dimensional gait analysis (3DGA) as a valid and reliable assessment tool to verify repeatability, consistency and variability in gait (17). The method enables comparison of treatment outcomes and movement before, during and after intervention (14). By providing information of different gait parameters, it assists in a better understanding of gait deviations to make clinical decision for treatment strategies and exercise prescription (18,19). In a biomechanical perspective, the gait cycle is usually described in terms of distance and timing events (spatiotemporal data), muscle activation (electromyography), joint angles (kinematics) and ground reaction forces, joint moments and power (kinetics) (20). The analysis is based on the human body being modelled as series of rigid body segments, linked together by joint rotation centers (4,21).



The joint rotation centers required for lower limb analysis is pelvis, hip, knee, and ankle. Each rigid body segment is defined by assigning a local three-dimensional coordinate system to it (18). The orientation of the coordinate system within one segment compared to another segment defines the relative movement between them. Every segment has three planes used in the description of gait pattern (22). X is referred as sagittal-, Y is coronal-, while Z is referred as transverse plane. Although the largest joint rotations and the major portion of work occurs in the sagittal plane in normal gait, three-dimensional data can highlight more complex interaction occurring in the coronal and transverse plane compared to subjective evaluation (23).

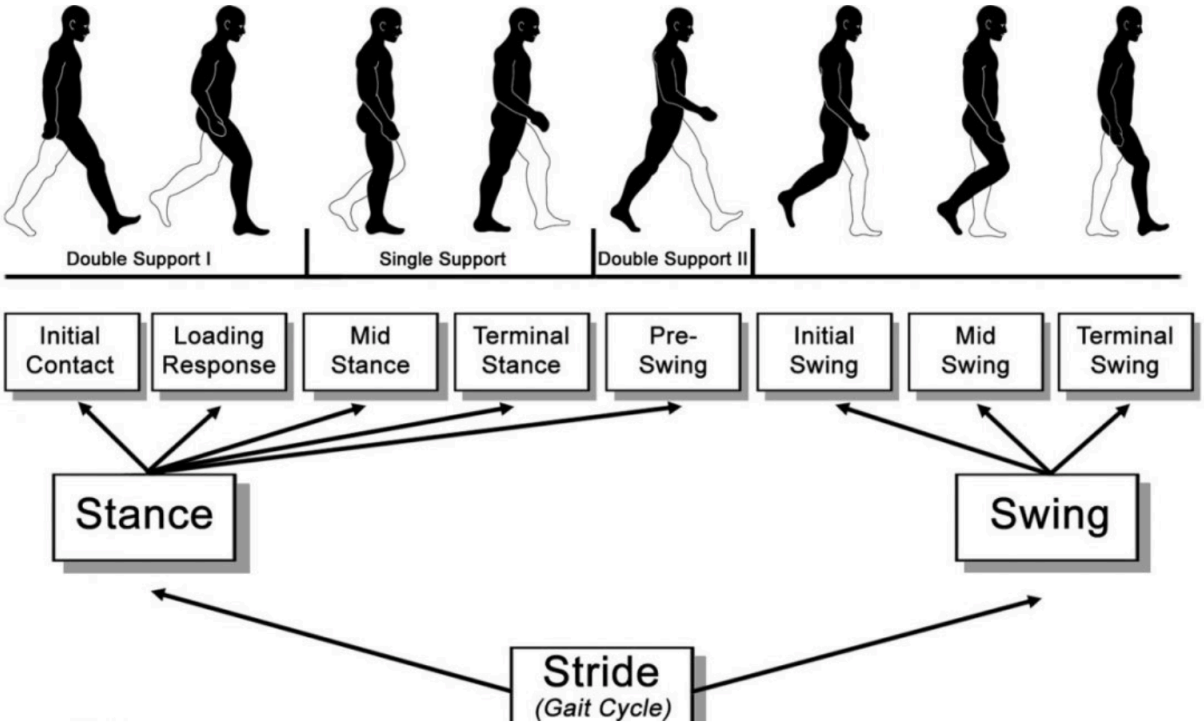


Figure 1 – *Stance period: Initial contact and loading response refers to contact of forefoot with the ground, acceptance of weight onto standing limb and shock absorption (eccentric muscle contraction). Mid-stance is a phase of forward progression of the limb over standing foot (eccentric muscle contraction). Terminal stance is forward propulsion (concentric muscle contraction). Swing period: Initial swing is the period of acceleration of the swinging limb. Mid swing is a transitional period from acceleration to declaration of the swinging limb. Terminal swing is a period of declaration of the swinging limb. Copied from Prokinetics (24).*

1.1.1 Spatiotemporal gait parameters

Spatiotemporal parameters refers to distance and timing of gait events (16). Definition of these parameters allows objective reports of when, where, how long, and how rapidly the limb is in contact with the ground (14). They include speed, cadence, stance time, step width, stride length and step length (13). Step length is described as the distance between two sequential heel contact, whereas stride length is the distance between two sequential heel contacts of the same foot (12). Step width is the medio-lateral displacement between the center points of the heels, whereas cadence is commonly defined as the number of steps per minute (25). The selected gait speed is normally measured in meter per second (m/s) (12). Humans prefer to walk at speeds that minimize the metabolic cost of transport, which usually rage between 1.2-1.4 m/s (26). Gait speed is a well-known indicator of overall gait performance and is commonly used to evaluate the effects of treatment (27). Unfortunately, when used alone, gait speed neither assists in understanding the nature of the gait deficiencies or is it helpful in directing training programs (27). However, it is important to be aware of how neuromotor patterns adapt to changes in gait speed and how it complicates the potential increase of elastic energy storage and recovery in tendons.

1.1.2 Joint gait kinematics

Kinematic analysis refers to angular variations of joints during movement and is used to identify gait cycles by describing angles and position of body segments in relation to space (28). It is the study of spatiotemporal aspects of motion such as velocities, accelerations and displacements. Joint angles between the proximal and distal segments of the relevant joint can be estimated using a three-dimensional model (21). However, they are limited in explaining causes of motion and the consequences for the musculoskeletal system. Table 1 presents clinical conventions used to describe the joint range of motion around the pelvis, hip, knee and ankle joint in three anatomical planes during gait laid out in rows and columns. Columns represent the three different anatomical planes, while rows represent the different joints levels.

Table 1 - *Clinical conventions of joint angles in sagittal-, coronal-, and transverse plane.*

Joint	Sagittal plane (X)		Coronal plane (Y)		Transverse plane (Z)	
	Positive (+)	Negative (-)	Positive (+)	Negative (-)	Positive (+)	Negative (-)
Pelvis	Anterior tilt	Posterior tilt	Up	Down	Forward	Backward
Hip	Flexion	Extension	Adduction	Abduction	Internal rotation	External rotation
Knee	Flexion	Extension	Varus	Valgus	Internal rotation	External rotation
Ankle	Dorsiflexion	Plantar flexion	Eversion	Inversion	Internal rotation	External rotation

1.1.2.1 Pelvis joint kinematics

The clinical conventions for kinematic joint angle of the pelvis segment is described in terms of rotations around the sagittal-, coronal-, and transverse plane (29). During normal gait, the pelvis has motion about all three axes. In adults, the pelvic range of motion (ROM) ranges from 5º to 15º (30). The magnitude of these motions is dependent on gait speed, with larger motions occurring at faster gait speeds (31). Pelvic tilt refers to the rotation of the pelvis about a line through both hip joints in sagittal plane around the mediolateral axis. In the three-dimensional coordinate system, anterior pelvic tilt is defined as positive excursion (+), while posterior pelvic tilt is negative (-). Second, pelvic obliquity refers to coronal plane motion around the anterior-posterior axis. Positive pelvic oblique excursion angle is defined as pelvis up (+), while negative angle is pelvis down (-). Last, pelvic rotation refers to transverse plane motion, the rotational movement of the pelvis through caput femoris around the midline of the body, longitudinal axis. Pelvic forward rotation is positive excursion rotation angle (+), in contrast to pelvic backward rotation which is defined as negative (-).

1.1.2.2 Hip joint kinematics

Hip joint movement is defined around three main axis, medial-lateral, anterior-posterior and longitudinal axis (32). The movement are restricted due to the size of the joint surfaces, tightness and the attachment of the capsule ligaments (33). Around the medial-lateral axis, sagittal plane motion of flexion and extension occurs. Hip flexion angle is defined as positive excursion angle (+), while extension is defined as negative angle (-). Maximum hip flexion of 30-35º occurs in late swing phase around 85-90% of gait cycle, while maximum extension of 10º is reached near toe-off at approximately 50% of the gait cycle (30). The movements around the anterior-posterior axis, coronal plane motion of abduction (ABD) and adduction (ADD) occur, respectively side bending of the pelvis with the upper body. ADD is defined as

positive excursion (+) hip angle, while ABD is negative (-) (24–28). ADD occurs throughout early stance and reaches a maximum at 40% of the cycle, while hip ABD of 5-7º occurs in early swing phase. Third, transverse plane motion of internal and external rotation occurs around the longitudinal axis. Internal hip rotation is referred to as positive excursion (+) rotation angle, and external rotation is negative (-). In addition to these three major axes, movements can be performed around all axes between the transverse and sagittal axes, and combine them into a circular induction (16).

1.4.3.3 Knee joint kinematics

The movements of the knee joint occur around two main axes. Movement in sagittal plane is defined as flexion/extension around medial-lateral axis, where knee flexion angle is defined as positive excursion (+) and extension angle is negative (-). Peak flexion is achieved at around 15% of the gait cycle when the entire body weight is accepted during single stance. This action is controlled by an eccentric contraction of the vastus lateralis, medialis and intermedius (16). The second peak flexion is produced at around 70% of the gait cycle. This shortens the limb when entering the swing phase which assists with toe clearance. Initially this action is produced by the concentric contraction of the gastrocnemius. Secondly, coronal plane motion of varus (ADD)/valgus (ABD) occurs around the anterior-posterior axis. ADD angle is defined as positive excursion (+), while ABD angle is negative (-). Third, transverse plane motion of internal/external rotation occurs around the longitudinal axis. Internal rotation angle is defined as positive excursion (+), while external rotation is defined as negative (-).

1.2.3.1 Ankle joint kinematics

The structures of the ankle are complex. It permits movements of dorsiflexion (DF) /plantar flexion (PF) and eversion (EV)/inversion (INV) in the sagittal, coronal and transverse plane. The ankle joint, art. Talocrualis is classified as a hinge joint with one degree of freedom. It allows motions in all three anatomic planes. In the sagittal plane motion occurs around medial-lateral axis (YZ plane) which permit movement mainly in PF and DF. DF is given positive excursion angle (+), while PF is given negative excursion (-). Peak DF occurs at around 50% of the gait cycle. The motion is controlled by eccentric contraction of the ankle plantar flexors around MS. The maximum peak of plantar flexion is achieved around 62% of the gait cycle near toe-off (3). A second DF is achieved at around 80% of the gait cycle.

1.1.3 Joint gait kinetics

Kinetic data analysis is the most accurate method to identify gait events regarding the forces that causes motion (4). It can reveal decreased muscle power in the gait cycle, and might be the key to establish a complete understanding of compensatory mechanism in pathologic movement patterns (4). To recognize the concept of kinetics, it is important to have knowledge of some fundamental terms. Different structures of the musculoarticular complex, fascicles, aponeurosis, tendons, joints and skeletal muscles are involved in production of joint motion (33–37). The two main functions of skeletal muscles is to generate movement and produce force (33,36,38,39). Force is generated by the muscle contractile elements to create torques/moments around an axis of rotation at joints, which predicts how a muscle operate during gait (40). A moment or torque is defined as the product of magnitude of force and perpendicular distance from the axis of rotation (41). According to Newton´s third law, the ground produces a reaction force equal and opposite to the body weight while standing still (36). This is called ground reaction force and acts on the center of pressure.

In the context of gait kinetics, power is described in terms of generation and absorption of energy (2,12,42). The ability of a muscle to generate power is influenced by the type of muscle action involved and the velocity sustained (43). Power is the product of the joint moment and joint angular velocity (ω), and is measured in watts (W) normalized to body weight (42). It is defined by the force-velocity relationship, determined by the architecture index of a muscle. It includes physiological and anatomical cross-sectional areas, fiber type, muscle thickness, motor neuron recruitment, firing rate and length and angle of fibers. Efficient power generation and absorption for forward propulsion occurs on high angular speed recruiting mostly fast-twitch muscle fibers (Type II) (25). They are capable of concentric-, eccentric and isometric contractions during gait (5). The muscle length is shortening while generating positive power through concentric activity (44). In contrast, the muscle is lengthening while absorbing negative power through eccentric muscle contractions as it contacts the ground and exerts a braking force (9). Irrespective of equivalent muscle lengths and velocity, more forceful contractions are produced eccentric compared to concentric (45). Yet, concentric muscle work in the sagittal plane is considered to be responsible for the power propulsion during gait (46).

1.2 Kinetic analysis during in gait

1.2.1 Ankle joint structure and power generation

The ankle joint is made up by a fork-shaped joint surface at the talus for the distal tibia and fibula (47). Several muscle groups are acting in this joint, yet the ankle dorsal- and plantar flexors are the main contributors (33). Tibialis anterior is known as the most powerful dorsiflexor (33). However, the plantar flexors gastrocnemius and soleus, also known as triceps surae, are more than four times as powerful as the dorsal flexors (48–50). Their biological muscle architecture with short pennate fascicles, long tendons and aponeuroses make them well suited for force and power generation during ankle push-off (51,52). In fact, triceps surae represents about four-fifths of the total plantar flexion moment (3,30). They are particularly important for forward ambulation, trunk stabilization and leg swing initiation, serving to modulate step length and gait speed (3,53,54). Due to their similar activation profiles and distal insertion onto the Achilles tendon, it is traditionally assumed that gastrocnemius and soleus have similar function during gait. However, gastrocnemius is a biarticular muscle with two heads crossing both the ankle and knee joint. For that reason, it is capable of generating knee flexion moment that is unique from soleus (55,56). On the other hand, soleus is a monoarticular muscle that originates from the proximal fibula and proximal medial border of the tibia, and inserts into the calcaneus (49). Soleus is a major contributor to the ankle plantar flexion in late stance and is crucial in propelling the body forward (49).

Figure 2 gives insight to ankle power pattern in the sagittal plane in normal gait. In fact, around 93% of the mechanical work is produced in the sagittal plane (4). The largest power output in the gait cycle is generated by the ankle joint during push-off, approximately 50% of the gait cycle (47,57). During the first 40% of the gait cycle, the ankle moment at heel contact absorbs negative power by contracting eccentrically (A1) to control the shank as it rotates over the ankle (4). This phase is followed by a region of positive power by concentric ankle plantar flexion activity throughout mid- and late stance phase of gait (60%) (A2), propelling the body over the leading stance limb (58). This second phase is mainly produced by the release of elastic energy stored in the Achilles tendon during A1 absorption period (59). A2 power is known as a strong predictor for modulating step length and gait speed (55–58).

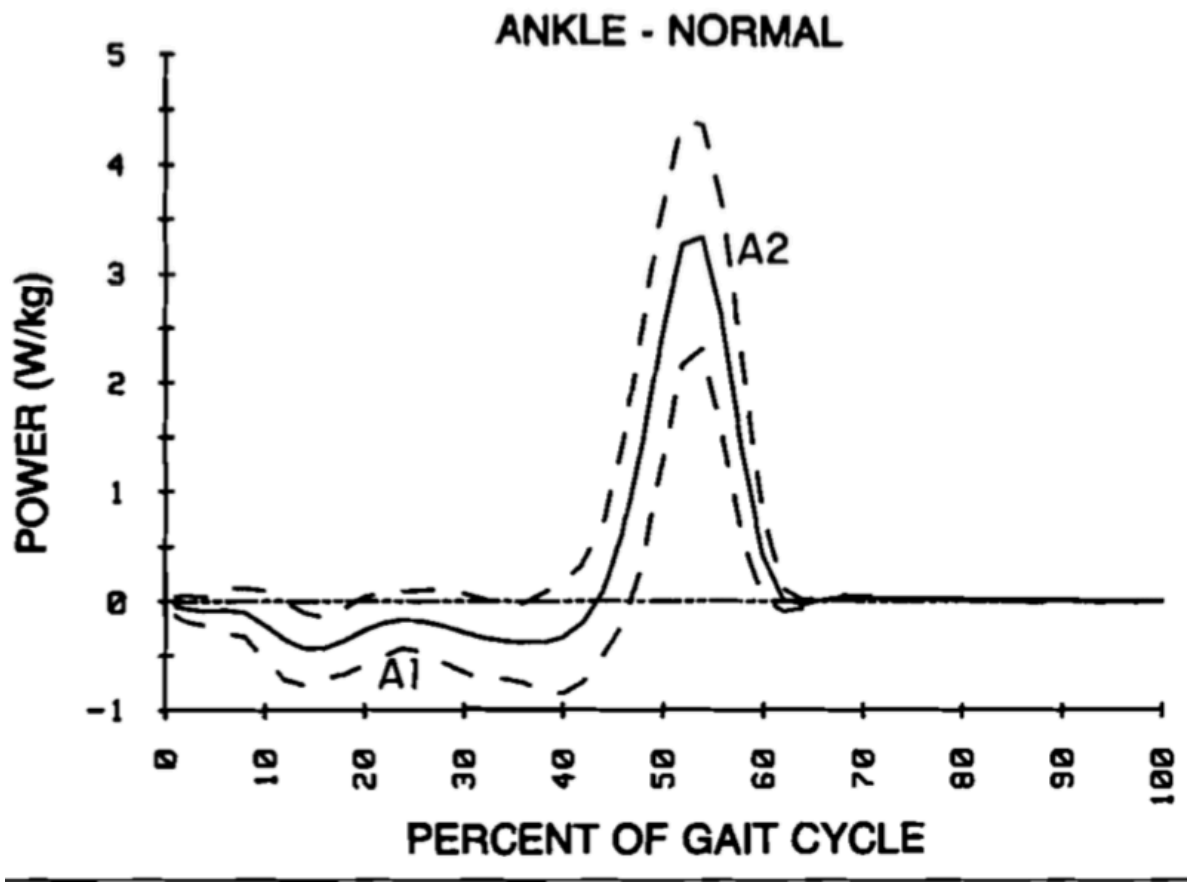


Figure 2 - *Average power patterns of ankle in sagittal plane for 19 health subjects walking at mean natural cadence of 105 steps/min showing M and SD. Redrawn from Winter (3,42).*

1.2.2 Hip joint structure and power generation

The hip joint is surrounded by a complex ligamentous structure and a solid joint capsule (64). The joint capsule has a protective function to restrain the movement of the femur articulating around the acetabulum and to prevent dislocation. Strength and coordination provided by both hip flexors and extensor muscles are active and important during distinct periods of the gait cycle (43). As a function of the large physiological cross-sectional area, the muscles crossing the hip have a higher potential of force generation than the knee and ankle muscles. Out of the muscles acting as extensors in the hip joint, the gluteus maximus is known as the most powerful. Iliopsoas is the most important flexor in the hip joint and has an important stabilizing effect to the upright posture (33).

While walking, a large proportion of work is produced by the hip joint (4). Figure 3 gives insight to the hip power pattern in the sagittal plane in normal gait. The hip power pattern can be subdivided into three phases. The first phase (H1) of concentric activity during initial contact is produced by the hip extensors. The hamstrings and gluteus maximus have the primary responsibility and serves three main functions. They prevents collapse of the body during single support, generate motion as the stance phase begins and controls trunk inclination through active hip extension (4,65,66). The second peak joint power (H2) is produced eccentrically by rectus femoris in the transition from stance to swing. Both adductor longus and adductor brevis eccentrically contract negative power in stance to attenuate hip abduction. These muscles act to absorb some of the mechanical energy generated in H1. H2 slows the backward rotation of the thigh and produce forward inclination of the trunk (4). This peak joint power allows storage of elastic energy that is utilized at the end of stance (66). At the end of stance (H3), a third joint peak positive power is generated by concentric action of hip flexors iliopsoas and sartorius (67–69). This contraction velocity propel the leg into the swing phase (70) by adding mechanical energy to the limb (4). Moreover, it has an important stabilizing effect to the upright posture. Sometimes, a very small concentric phase by extensors at the termination of the swing phase (H4) is identifiable. This phase function to facilitate the preparation for the next gait cycle (23,71).

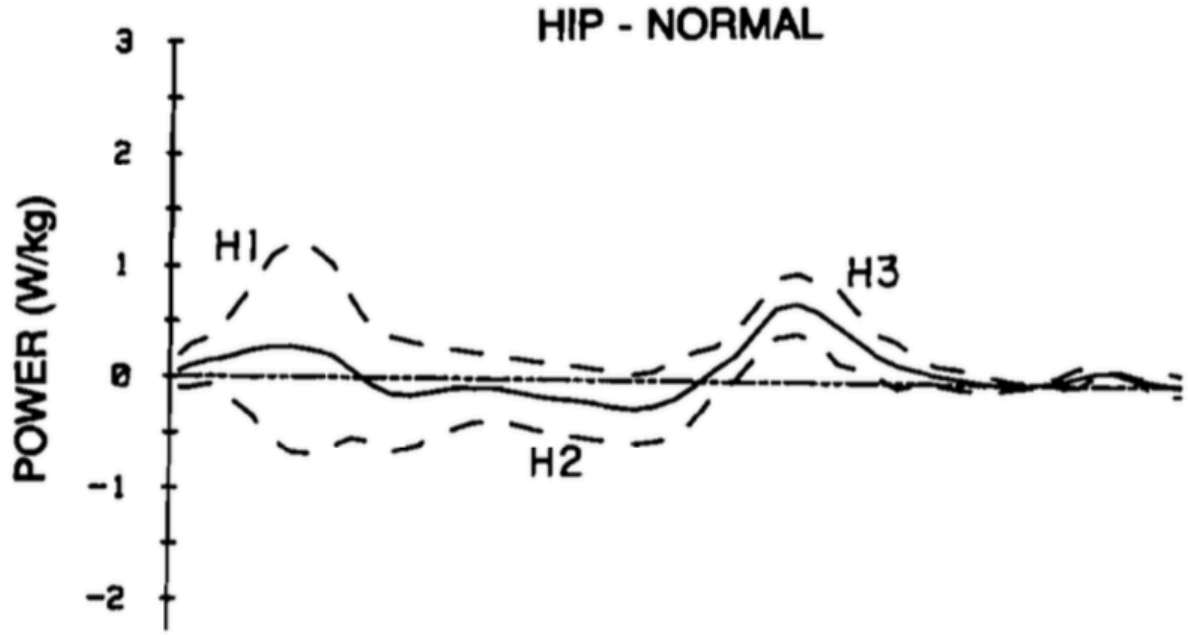


Figure 3 - *Average power patterns of the hip in sagittal plane for 19 health subjects walking at mean natural cadence of 105 steps/min showing M and SD. Redrawn from Winter (3,42).*

1.2.3 Ankle-hip ratio (A2/H3)

In general, most of the positive work in normal gait is produced by the ankle plantar flexors during the late stance phase of gait (A2) with a smaller contribution of the hip flexors at push-off (H3) (2). However, there is an complementary relationship between these joints in propulsive force generation (72,73). The ankle-hip power ratio (A2/H3) characterizes the relationship between proportions of ankle plantar flexion and hip flexion during the push-off phase. It is the region of positive power produced by the concentric plantar flexor activity during late stance, divided on positive power produced by concentric activity in the hip flexors during late stance (5,6). According to Winter and Olney (2,3,27), normal A2/H3 power is 6.8W/kg, range 4.89-8.0. A higher ratio indicates a higher proportion of ankle involvement compared with hip.

1.3 Management of gait deviations to improve ankle power generation in late stance

The ability to sustain walking capacity is a crucial feature to maintain independence in the society and everyday life (74). Rehabilitation appears to be most effective when it is task specific (75). The primary focus is to reflect the goal of treatment and identify factors limiting gait performance (10,63). With other words, the prescription should reflect how the muscles act during walking. It is critical to consider the relevant muscle groups, required velocity, intensity, repetitions and individual factors to achieve results effectively (8). Moreover, the decision is influenced by functional status, musculoskeletal deformities, age and requires identification and understanding of the impairment. Repeated efforts and several treatment modalities are investigated to develop a general rehabilitation program. Regular clinical interventions include orthopedic surgery to restore muscle-tendon length to improve bony alignment, neurosurgical techniques to reduce spasticity, use of orthotic devices and physical therapy to address muscle length, body positioning and mobility (45). Although reduced ankle power generation and hip compensatory actions is a well-known phenomenon in several neurological gait conditions, strategies targeting the plantar flexor muscles power generation have received little attention (76–78). There are currently few rehabilitation programs assessing this deviation (3,25,67,79), and the optimal guidelines to increase A2/H3 ratio have not yet been established (80).

1.3.1 Ankle foot orthosis

Ankle foot orthosis (AFO) is considered as a conservative treatment option (81). They are primary designed to provide joint stability and to keep tight muscle stretched while preventing excessive plantar/dorsiflexion flexion (17,81,82). The main objective is pre-position of the foot in initial contact, allow heel contact and correct the foot angle in swing phase (81,82). AFO is a relatively common treatment option, used both in activity and rest. Several types with different functions have been developed to meet the various gait pattern and muscle deformities in cerebral palsy. Reported benefits are reduced spasticity, free movement and a more stable COM. Despite the benefits, AFO fails to reduce deformity and have not exposed to improve ankle power output in the late stance of gait.

1.3.2 Botulinum toxin type A

Intramuscular injections of Botulinum Toxin type A (BTX-A) has been clinically used for many decades and have reviled positive outcomes on spastic muscles (83). There are seven types of neurotoxins (A-G), however only A and B are available for clinical use (83). When BTX-A is injected focal into muscles, the release of Acetylcholine is inhibited in neuromuscular synapses, and thereby reduces spasticity (17,83). The dose varies individually based on the degree of spasticity and body weight (84,85). The main objective is to improve movement patterns and energy consumption. Previous studies has illustrated significant improvement in ankle kinematics and kinetics (83,84). The treatment outcomes includes reduced spasticity of the plantar flexors, improved dorsal flexion and increased force generation in the push-off phase (17,84). However, the treatment effect is reversible, and some side effects have been reported, but they are rare.

1.3.3 Surgical treatment

Surgery is commonly used to address bony deformities, correct alignment, muscle contracture, reduce spasticity and improve function (86). Interventions such as muscle tendon lengthening surgery can change a toe-gait pattern to heel-toe pattern by reducing the persistent stance phase loading of the plantar flexors. This may serve to reduce the degree of eccentric lengthening of the gastrocnemius. However, orthopedic surgery involving muscle-tendon lengthening procedures is known to reduce muscle strength, particularly in the presence of pre-exciting weakness. Additionally, it may decrease the ability to generate

moment and power output of the muscle groups. Surgical decisions are therefore particularly crucial in slow-walking individuals with low power generating abilities (2).

1.3.4 Traditional resistance training

The rationale of physiotherapy in gait rehabilitation may be to maintain range of motion with adequate muscle length, to preserve strength, and to improve balance and coordination (17,87). The intervention includes passive muscle stretching. Although interventions including traditional resistance exercise is quite common, they have only shown weak correlations with gait functionality improvements and generation in forward ambulation (88). Preview studies have focused mostly on strengthening knee flexors, knee extensors and hip abductors (77,78). Despite modest increase in muscle strength in proximal parts of the lower extremity, the improvements in muscle strength is apparently not transferred to power generation and walking ability (8,75,76,89). If a muscle contractions are performed at very slow velocities, only a few cross-bridges have time to shorten and reduce the net tension recorded in response to the stretch (40). Moreau et al. (76,90) argued that the majority of exercise prescription may need to prioritize the elastic function of the Achilles tendon and the stretch-shortening cycle on higher movement velocities to maintain functionality. By addressing the imbalance between hip and ankle, strengthening the weaker muscle may preserve muscle length (75,90).

1.3.5 Ballistic strength training

It is though that A2 power is produced by both concentric muscle contraction and return of elastic energy stored in the Achilles tendon (49,53). During the stance phase of normal gait, the calf muscle fascicles barely lengthen, which allows elastic energy storage and release in the Achilles tendon (51,92). This is not the case in several neurological gait disorders. In order to improve power generation for walking in neurologic populations with muscle paresis, prescription may need to target higher velocities as well as higher force (25). Ballistic strength training is a rehabilitation option inspired from sprint which emphasize to increase explosive ankle power generation at pull-off (90,93). By performing loaded exercises at higher velocity, the plantar flexors is undergoing an eccentric activation followed by immediate concentric contraction (94–96). It is thought that this stretching and eventual breaking of the actin-myosin complex generates a higher rate of force development, power

output and motor-unit recruitment when compared to traditional, non-ballistic alternatives (90). Moreover, it produces a higher firing frequency greater force, velocity, power and muscle activation. Although the application is relatively novel and not yet supported in randomized controlled studies (RCT) or in clinical practice, it has revealed positive outcomes in several studies and population groups. Lewies et. al (72) demonstrated that healthy children instructed to increase ankle push-off during gait, had lower hip flexion and extension moment as well as decreased hip power in the late stance (6). By exaggerating the ankle strategy, this pushing of the center of mass decrease the need for a hip extension moment action on the stance leg to pull the center of mass forward (97,98). Van Vulpen et al. (99,100) implemented functional power training in mildly affected children with SCP. Significant improvements were obtained in leg muscle strength, gait distance, and running speed. According to Jonkers et al. (101), higher functioning post-stroke subjects increase plantar flexor power generation (A2) of the paretic limb. For the non-paretic limb, a similar trend was found. Preliminary evidence suggest that ballistic strength training may be safe and feasible, having a positive effect for several neurological disorders (14,17,19–23). Hendrey et al. (8) reported feasibility of ballistic strength exercises compared with usual care after stroke. Moreover, they indicated greater peak jump height, peak propulsive velocity and significantly faster gait speed in the ballistic group. Williams et al. (10,11) found by significantly greater performance in peak jump velocity, height and force production after training ballistically compared to seated leg press and leg extension performed non-ballistically in TBI (traumatic brain injury). They hypnotized that performing higher-level mobility task would improve ankle power output during the push-off phase and may further reduce the need for excessive hip power generation (102–104). However, it is still unknown whether these hypothesis of training principles relative to kinetic relationships hold true in SCP. To our knowledge, no study has yet determined the influence on ballistic strength training on functional gait capacity and power generation adults with SCP.

Chapter 2: Spastic cerebral palsy and gait characteristics

2.1 Pathology of spastic cerebral palsy

Spastic Cerebral Palsy (SCP) describes a heterogenous group of a complex motor disability, originating from a nonprogressive lesion to the central nervous system in the developing fetal or infant brain (17,74,87,105). The motor disorder is lifelong, with a prevalence of approximately 1 in 500 live births (1,106). Prenatal events account for around 75% of cases, while lack of oxygen, extreme premature birth, low birth weight or childhood illness are other causes. The clinical features are multifactorial, often accompanied with disturbances of sensation, perception, cognition, communication and behavior (74). Although the brain lesion is static, the chronic impairment affects the development growth of muscles and bones. In other words, individuals with SCP has a progressive neuromusculoskeletal pathology associated with delay in onset of walking and an abnormal gait pattern compared to typically developed peers (107–109). The present of skeletal muscle spasticity and contracture is considered as the major obstacle to motor function, which occurs secondary to upper motor neuron lesions as a result of improper or absent motor command from the brain (76,87,110). Spasticity is presented as intermittent or persistent involuntary activation of muscles and diminished selective motor control (37). In particular, these deficiencies prohibit a muscle from achieving an operative length on high velocity (45,91,111,112), which serves to limit torque production in the agonist muscle (113), restrictions to relax the muscles, difficulties to move in alternating directions and muscle weakness in affected extremities. Constant spasticity eventually increases energy consumption and produces pain which progressively lead to worse deformities of bone structure around the tightened musculature (114–116). Further, it increases the likelihood for secondary conditions (5). These factors suggest that adults with SCP may experience continuous deterioration in gait function and marked functional restrictions of movement associated with increased pain frequency and physical fatigue (74,116,117). Besides, since organized health care often confines when reaching adulthood, this population are facing even greater challenges to achieve adequate levels of physical activity, social participation and sustain quality of life (QoL) compared to the general population (87,108,118–121).

2.2.1 Gross motor function classification system

Generally, classification of CP often differ between unilateral hemiplegia and bilateral diplegia, with 39% and 38% of cases (17,87). Depending on the degree of the neurological severity sustained, individuals with CP experience functional limitations, ranging from minor impairment to being completely physically dependent on health professionals (122,123). Gross Motor Function Classification System (GMFCS) is a clinical assessment tool used to distinguish the functional capacity level of individuals with CP from I-V (124,125). This scale evaluates the extent to which patients are able to walk and perform motor tasks based on topography and impact level of impairment. It has been shown that even mildly affected adults with CP have significantly reduced isometric strength in all major lower extremity muscle groups required for mechanical power production during gait compared to age-matched pears (7,91,126–128). Recent findings from Williams and collogues (9) indicates that these mainly include the hip extensors, ankle plantar flexors and hipflexors (129). The individuals express various gait deviations with a lack of ability to provide push-off power with the paretic limb from mid-stance phase to the initial swing phase during gait (17). From these patterns, it is important to distinguish the primary gait deviations which are a direct consequence of the underlying deficits associated with the compensatory strategies which are deviations due to the biomechanics constraints and pathology (123). An illustration of common abnormal gait patterns following CP is illustrated in figure 4.

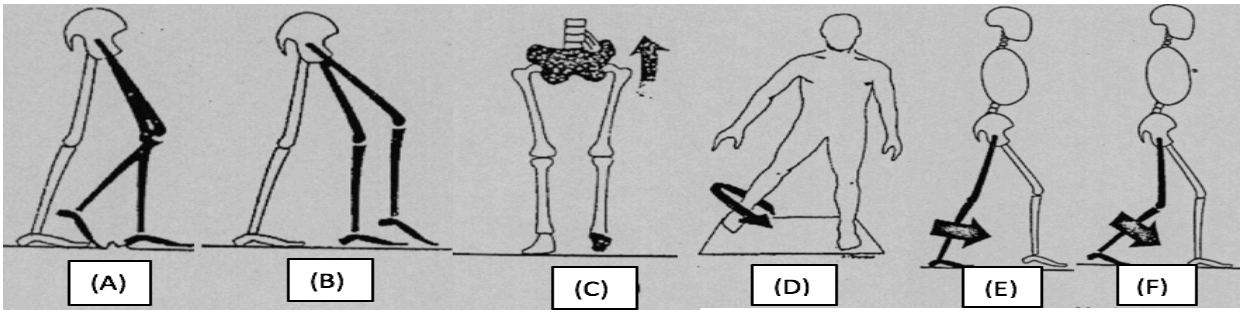


Figure 4 - *DF weakness; A) toe drag, B) high steppage gait, C) hip hiking, D) circumduction. Excessive DF is represented by: E) prolonged heel contact, F) excessive knee flexion* (17,130).

2.2.2 Spastic hemiplegic cerebral palsy

The most widely accepted classification of gait in spastic hemiplegia is based on sagittal plane kinematics reported by Winter et al. (131). It is primary associated with unilateral motor involvement. A typical feature is weaker, smaller and shorter muscle volume and leg length on affected limb, resulting in abnormal movement of the non-pathological limb during gait (130). Stance time on the unaffected side is greater than stance time on the affected side (132,133), which results in a measurable asymmetry (134,135). Joint-angle disturbances of the affected side include reduction of knee flexion phase in stance, reduction of knee flexion range during the swing phase, occasional loss of dorsiflexion of the ankle in swing phase and at initial contact, and generally reduced excursions. As a consequence of excessive plantar flexion, the individuals land with a foot flat or toe contact, leading to loss of stability during stance (136). The swing phase is associated with the affected limb being circulated due to foot drop. Further, Riad et al. (137) reported significantly lower concentric muscle work on the hemiplegic side compared to the noninvolved side for the ankle plantar flexors, knee extensors, hip flexors and hip extensors (46,137). This is mainly due to increased restraint from stretch reflexes from a spastic antagonist muscle that is lengthening during a maximal concentric exertion in the agonist.

2.2.3 Spastic diplegic cerebral palsy

The second most common type of CP among preterm infants is spastic diplegia (138). With both lower limbs affected, their gait pattern are often highly variable as a consequence of calf spasticity, equinus, contractures and overlengthened triceps surae muscle (109,131). These individuals commonly express hip extensor weakness, which may be compensated with a tendency of excessive hip- and knee flexion, hip adduction and anterior pelvic tilt in the swing phase to clear the foot from the ground (139). During stance, a limited dorsiflexion in the ankle joint has been associated tibial internal rotation and foot pronation. Toe-gait gives rise to increased mean plantar flexor moments in stance when compared to heel-toe gait. Further, a jump gait pattern is commonly seen in diplegia, expressed with spasticity in the hamstrings, hip flexors and plantar flexors.

2.2 Gait cycle in spastic cerebral palsy

2.2.1 Spatiotemporal gait parameters

Spatiotemporal deviations regarding gait pattern in SCP is characterized by decreased duration of stance phase in single support, increased double support, delayed heel rise during stance phase and shorter steps (27). Several studies assessing subjects with SCP have reported generally slower self-selected speed with the percent of stance time longer on the unaffected limb over the affected limb compared to populations without gait pathology (101). Slow gait speed was originally thought to be an adopted safety strategy related to poor balance and postural instability. Alternatively, it is likely that postural instability with deterioration of the fine motor control is a consequence of impaired muscle strength and reduced ability to push-off in late stance phase (140). Since weakness is particularly pronounced in the distal muscles of involved lower limb, it is proposed that weakness of the plantar flexors could limit the maximal plantar flexion moment and power required to walk rapidly (59). Reduced power output during gait are regularly accompanied by shorter steps, slower speeds and reduced gait economy following aging, stroke and TBI compared to age-matched controls (30,63,65,141).

2.2.2 Joint gait kinematics

The evidence on kinematic parameters for spastic gait are plural yet varies. Frequent kinematic deviations and associated impairments with CP is early ankle plantar flexion due to overactivity in plantar flexors (17). Increased ankle dorsiflexion in stance is a result of soleus weakness and increased knee flexion in mid-stance. Increased hip flexion is due to contracture or hip extensor weakness. While lack of hip extension is caused by reduced ROM and anterior pelvic tilt. Increased hip adduction is caused by abductor weakness or overactivity of hip adductor.

2.2.3 Joint gait kinetics

Neurophysiological and biomechanical bases for kinetic force in SCP remain poorly understood (99). However, it is well accepted that this population often have longer Achilles tendons and reduced muscle belly volume of gastrocnemius, cross-sectional area, thickness and fascicle length compared to age-matched peers (142–144). This abnormality reduce the capacity to generate muscle power during gait (142–146). The reason is that fascicle length determines the range which muscles can develop force and power, maximum shortening speed, and length at which passive forces are developed (38,144). Besides, since predominance of type I fibers is reported in CP and additional loss of type II fibers in the plantar flexor muscles is a concern when ageing (147), it may and contribute to decreased rate of force development as shortening velocity of slow twitch fibers (I) is 25% less than that of fast twitch (II) (148). Long-term consequences are inefficient gait pattern, shorter stride length, reduced gait speed, increased energy expenditure and difficulties performing movements out of synergy (91,137,149,150).

2.3 Kinetic analysis in spastic cerebral palsy during gait

2.3.1 Ankle power generation

Pathological populations with CP appear to generate less power with the ankle in late stance of gait (A2) compared to non-pathological population (7,59,91) caused by greatly reduced plantar flexor musculature volume and strength (5,6,67). Olney (2) and Eek (7) reported that the ankle plantar flexors on hemiplegic side produced approximately a third of the power generation instead of the normal two thirds. As illustrated in figure 5, the ankle plantar flexion negative work (A1) was small in the group with cerebral palsy, followed by small positive burst during stance that does not occur in the general population (2). It is highly likely that these factors reduce the capacity to store elastic energy during A1 peak power absorption (3,151).

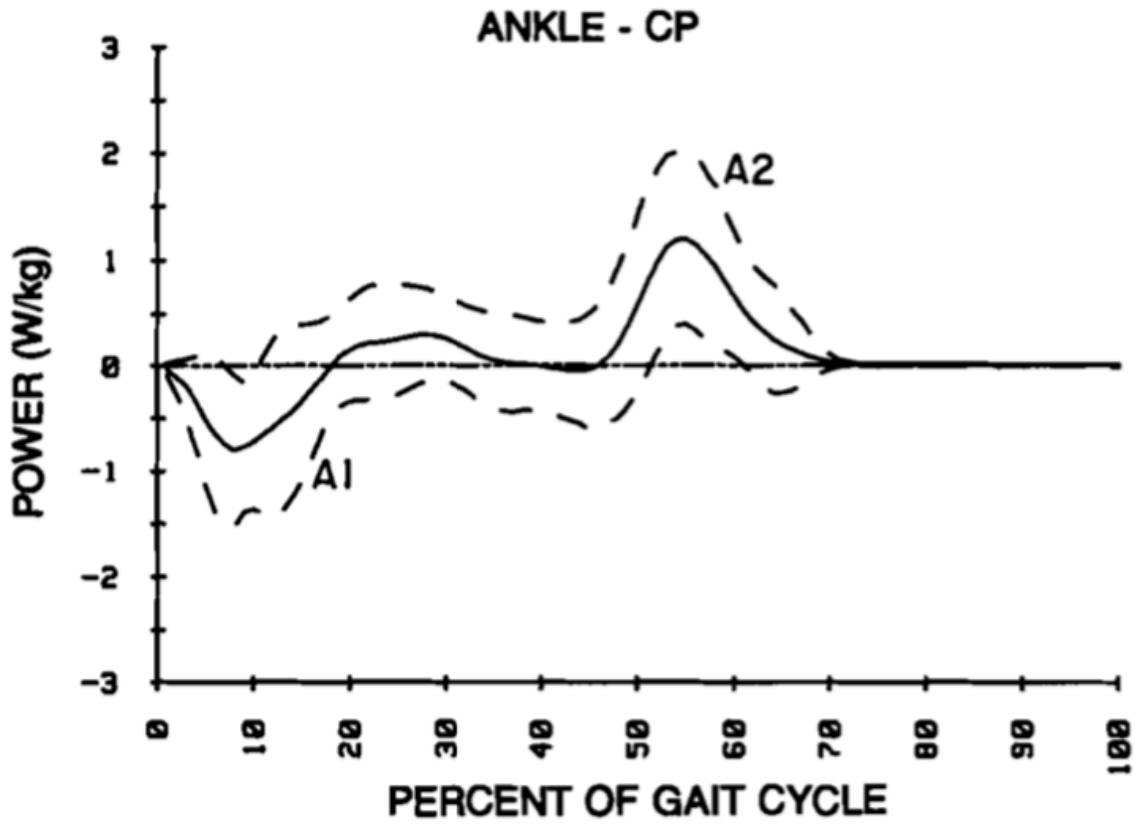


Figure 5 – *Ankle joint power graphs for 10 children with CP showing mean and standard deviation. Mean cadence was 131 steps/min. Illustration: Olney (2).*

2.3.2 Hip power generation

Non-affected muscles tends to compensate for weakness in other muscle group (46,152). Since proximal muscle strength may be less diminished than distal muscle strength in populations with SCP, engaging hip flexor power (H3) during push-off to pull the leg into swing has emerged as a potential compensation strategy for reduced ankle power output (A2) to maintain postural stability (52,59,63,153,154). According to figure 6 presented by Olney (2) and Eek (7), large positive work performed by the hip extensors in early stance (H1) continued late into stance in children with CP. The positive phase of hip flexors (H3) which occurred during late stance was also large. This case also applied in the final burst of positive work before initial contact (H4) produced by the hip extensors (2).

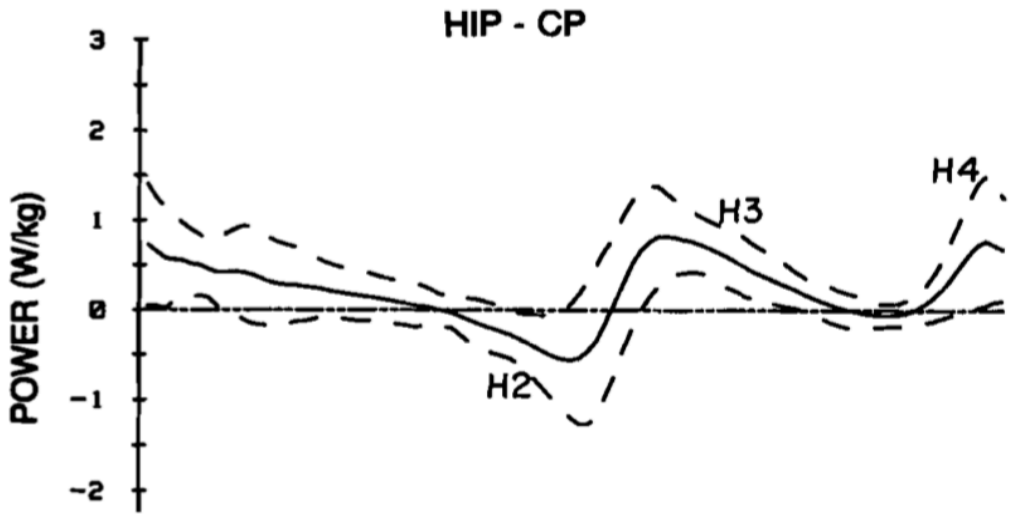


Figure 6 - *Hip joint power graphs for 10 children with CP showing mean and standard deviation. Mean cadence was 131 steps/min. Illustration: Olney (2).*

2.3.3 Ankle-hip ratio (A2/H3)

As mention in chapter 1, most of the positive work of normal gait is generated by the ankle plantar flexors during the late stance phase of gait (A2) with smaller contribution of the hip flexors at push-off (H3) (2). However, individuals with SCP tends to compensate with hip power due to reduced ankle power output (2,5–7,27). This idea suggest that the functional role of iliopsoas to accelerate the leg into swing becomes more important as the contribution of gastrocnemius decreases (6,67,73). However, such proximal compensatory strategy to maintain forward ambulation may be unfavorable and lead to overuse of the hip musculature and increased metabolic cost during gait (46,63,127,154–156). Additionally, it may have an impact on the kinetic relationship between power generation in the ankle and hip joint (A2/H3) for forward ambulation. In a pilot study, Ishiara and Higuchi evaluated the kinetic relationships between the ankle and hip joints during gait in children with SCP (6). They found that the A2/H3 ratios on both the hemiplegic and uninvolved side was lower than those of the healthy control (6). The peak flexion moments generated of the hip was significantly higher, and the ankle did not provide a sufficient force during gait compared to age-matched peers (6). Compared to normal gait, Olney et al. (2) also found less contribution of the ankle compared to the hip in a group of 10 children with CP. A2/H3 was 1.5 (1.0-1.67) compared to 6.8 (4.89-8.0). in the healthy control. This trade-off relationships between the hip joint and ankle have also been studied in TBI and stroke (8–10,27,150,157). However, to our knowledge, there are currently no data on A2/H3 ratio in adult's SCP.

Chapter 3: Rationale and aims

3.1 General aim

It is well recognized that plantar flexor power output at push off (A2) in gait is remarkable affected in high-functioning adults with SCP (2,6,7). Previous findings indicates that increased hip power generation (H3) and metabolic energy cost are compensatory biomechanical causes of weak plantar flexors power output (A2) (59,140,154). However, the mechanisms underlying A2/H3 ratio are not completely understood. It is needed to investigate the association between joint power generation at the ankle and hip joints while walking in this population group (91). Specific treatment is seldom prescribed, partly because the impairment is mild and partly because effective treatment options are limited. It is considered essential to focus on specific muscle group when prescribing exercise to maintain walking ability, motor functions and quality of life. Ballistic strength training is a rehabilitation option which emphasize to increase explosive ankle power generation at pull-off. Although the application is relatively novel and is not yet supported by randomized controlled studies (RCT), it may be task specific for altering ankle power and functional walking capacity in adults with SCP. For that reason, the motive of this research is to explore if eight weeks of ballistic strength training improves power generation of ankle plantar flexors and kinetic ratio between the ankle and hip joint in the late stance of gait (A2/H3) in mildly affected adults with SPC. Knowledge of power patterns gained from this study might provide important implications in clinical practice, give a biomechanical explanation of compensatory actions and generate hypothesis to future analytic studies.

3.2 Hypotheses

Based on previous findings from Williams and Schache (150) and Ishiara and Higuchi (6), a few hypotheses was conducted:

1. High-functioning adults with spastic hemiplegic and diplegic cerebral palsy has reduced ankle power generation (A2) in late stance of gait and compensate with generating more hip flexor power generation (H3) for forward ambulation.
2. A ballistic strength training program for the plantar flexors increases ankle power output during push-off (A2) at self-selected gait speed. Consequently, hip flexor power (H3) decreases, resulting in altered A2/H3 ratio.

Chapter 4: Material and methodology

4.1 Case series study

Case series are a descriptive and observational study designs often used to describe general disease characteristics related to person, time and place (158). Such design can prospectively explore change over a given period of time with objective measurement methods. It is relatively economically advantageous and are considered suitable as it avoids several ethical issues. However, case series design is rated as level IV in the hierarchy of evidence (159). The main reason is the lack of randomization and comparison group, which means that no casual relationships or absolute conclusions considering whether the outcomes are attributed to the treatment or other patient characteristics can be stated. Despite the methodological limitations, the study design was considered convenient to answer the research question in this study since there exist little knowledge on the topic. The findings can be used to generate new hypothesis of treatment efficacy for a further analytic study (26). Moreover, the approach is better applied to clinical practice and was therefore considered highly relevant (160).

4.2 Ethical considerations

It is fundamental in research to aim for good outcomes regarding the population being studied (161,162). We confirm that this project was not a burden to the society and did not harm the participants in a negative way. To ensure safety of all participants, an acceptance of protocol from Regional Committees for Medical and Health Research Ethics (REC) (2018/349) and some precautions from Helsinki Declarations was fulfilled. The recruited sample got adequately information prior to the project. It included receiving an information letter and statement of consent (appendix 3) containing the purpose of the study, time perspective, method and responsible persons. The informants were aware of their right to terminate the study at any time without the need for giving a reason. The data material was treated confidentially, personal data was anonymized and stored in accordance to guidelines. All analyzed data and complete results is presented in this study regardless of whether they contradict the presumed hypothesis (161,163).

4.3 Inclusion and exclusion criteria

Prior to project start, written information (appendix 2) regarding the study was made available at a Norwegian training center for adults with CP and the regional hospital. Further, a recruitment video was published online and shared 105 times on Facebook to reach a wide range of adults diagnosed with mild CP GMFCS I-II living in or nearby Bergen. Participants were selected based on inclusion and exclusion criteria listed in table 2 to ensure that the project hypothesis were tested. In this research project, only high-functioning adults with SCP were included since they often fall out of rehabilitation, and since there exist few kinetic gait data on this group. The participants were inspected with a neurological examination, sensory testing, motor function test and reflex test prior startup. At this point, the most paretic limb was identified to be focused on in the training intervention.

Table 2 - *Inclusion and exclusion criteria*.

Inclusion criteria	**Exclusion criteria**
Adults >18 years old	Treatment with surgery 6 months before project
Diagnosed with SCP	Patients not able to walk without walking aid
Patients with hemiplegia or diplegia	Patients not able to understand instructions
GMFCS level I-II	Severe joint contracture, hypertension, spasticity
Tolerate physical strain	Not completed secondary school
Citizens near Bergen	

4.4 Study setting and sample

Ten grown up citizens with SCP (35.7±12.6) were recruited to participate in this study. All subjects were fully independent and unassisted ambulators, primarily graded as GMFCS level I and II (122). At start-up, the study sample was registered with an individual identifying number in order to collect personal information while assuring anonymity. Data material was conducted in the rehabilitation laboratory “SimArena”, at Western Norway University of Applied Sciences in Bergen. Baseline measurements were established in March 2019, with a three-month perspective. The data collection for all participants was complete in May 2019.

4.5 Ballistic “Leg sled hop” intervention

Guidelines of ACSM (American College of Sports Medicine) claim that “the most effective strength training programs are those who are specific to muscle requirements and task” (93). In this thesis a ballistic strength program was considered task specific to increase power output in the ankle in order to alter A2/H3 ratio. By isolating the plantar flexors and performing strength training explosively on higher angular velocities, it was thought that this exposure would optimize the mechanical function of the calf during gait (9). Figure 7 and appendix 4 illustrate three different ballistic strength exercises prescribed for eight weeks performed on a Power Tower (Total Gym RS Encompass, CA, USA) according to the protocol described by Hendrey et al. (99). Leg sled hop are performed in an inclined supported position on the affected lower limb with the knee relatively straight (8). The subject lands briefly on the forefoot and pushes off again to generate a flight phase. Since the functional impairments is remarkable different within this population group, it was considered advantageous that the intervention can be tailored individually. By adjusting the slope on the slide board, a proportion of body-weight can be decreased to accommodate weak muscles on the affected leg (8,9). Moreover, the load can be manipulated in the mid-point of 30-80% of 1RM where power is best developed. The outcome is likely to be individual in terms of previous experience with training, body characteristics, mental factors, muscle strength and ROM (25). The recruited sample received 2 center-based training sessions every week consisting of a small chat, 10 minutes of warm-up on bike or treadmill, and 15 minutes with 3 different power exercises on the led sled lasting for 5 minutes each. Altogether, giving a total of 160 minutes of warm-up and 240 minutes of high intensity ballistic strength training. In between each exercise, the participants were offered a resting period of approximately 1 minute. Number of breaks, jumps, slope and a general comment were noted for each exercise.

Figure 7 – *The positions preforming three different ballistic strength training exercises on the leg sled - exercise 1, 2 and 3 respectively.*

4.5.1. Exercise 1: Jump Squats on the leg sled

The aim was to target the ankle plantar flexor and hip extensor power with a focus on speed and height of the jump. The participants were placed supine on the leg sled with hips at 30^{o} flexion and feet on the footplate. The resistance was determined individual by the level at which the participants could achieve bilateral ankle plantar flexion and inner range knee extension on push-off while maintaining ankle alignment to prevent excessive ankle inversion or knee hyperextension.

4.5.2 Exercise 2: Single leg hopping on the paretic leg

The aim was to isolate plantar flexors power with focus on speed and jump height. The participants were placed supine on the leg sled with only the paretic foot on the footplate with hip and knee extended. The non-paretic leg was not considered to be trained in this study protocol and was bent up on the platform to maintain stability. The leg sled resistance was determined by the slope each participant was able to achieve flight phase ideally through his or her paretic leg. After performing the exercise, the participant was asked to rate the subjective impression of fatigue in the leg on Borg Scale from 6-20. 6 equals rest, while 20 is maximum effort (Appendix 5).

4.5.3 Exercise 3: Bounding on altering legs (jogging)

The aim was to target the coordination and power production of alternating hip flexion, hip extension, and ankle plantar flexion through quick and fast jogging. The participant was placed supine on the leg sled with hips bent to 30^{o} flexion. One leg is in contact with the footplate, while the other is held in hip flexion. The leg sled resistance was determined by the level at which participant can achieve flight phase bilaterally with good control and coordination. After performing the exercise, the participant was asked to rate the subjective feeling of fatigue in the leg and general exhaustion on Borg Scale from 6-20.

4.6 Experimental protocol

It is important to note that this project is a part of the Doctor of Philosophy project (PhD) “Functional Capacity for Walking in Adults with Cerebral Palsy (FUNCAP-CP)”. For that specific reason, the participants attended a comprehensive experimental testing protocol lasting for about 4 hours (74,117). An overview of the prospective timespan process for

intervention, specific screening, questionnaires and test protocols conducted in this study is illustrated in figure 8. However, in this thesis, only analysis of kinetic data was considered relevant in accordance with the hypothesis. 3DGA were performed at baseline, after six weeks and at week eight when intervention was completed to explore the A2/H3 ratio during late stance of gait, and to evaluate if the exercises had impact on power output relative to baseline (17). Gait data was collected using a motion capture system, (Qualisys AB, Gothenburg, Sweden) with eight high-resolution infrared cameras and a force plate device (Kistler Nordic AB, Jonsered, Sweden). The Qualisys Track Manager software (QTM) was used for the synchronized capture kinematic data at a sampling rate of 150Hz.

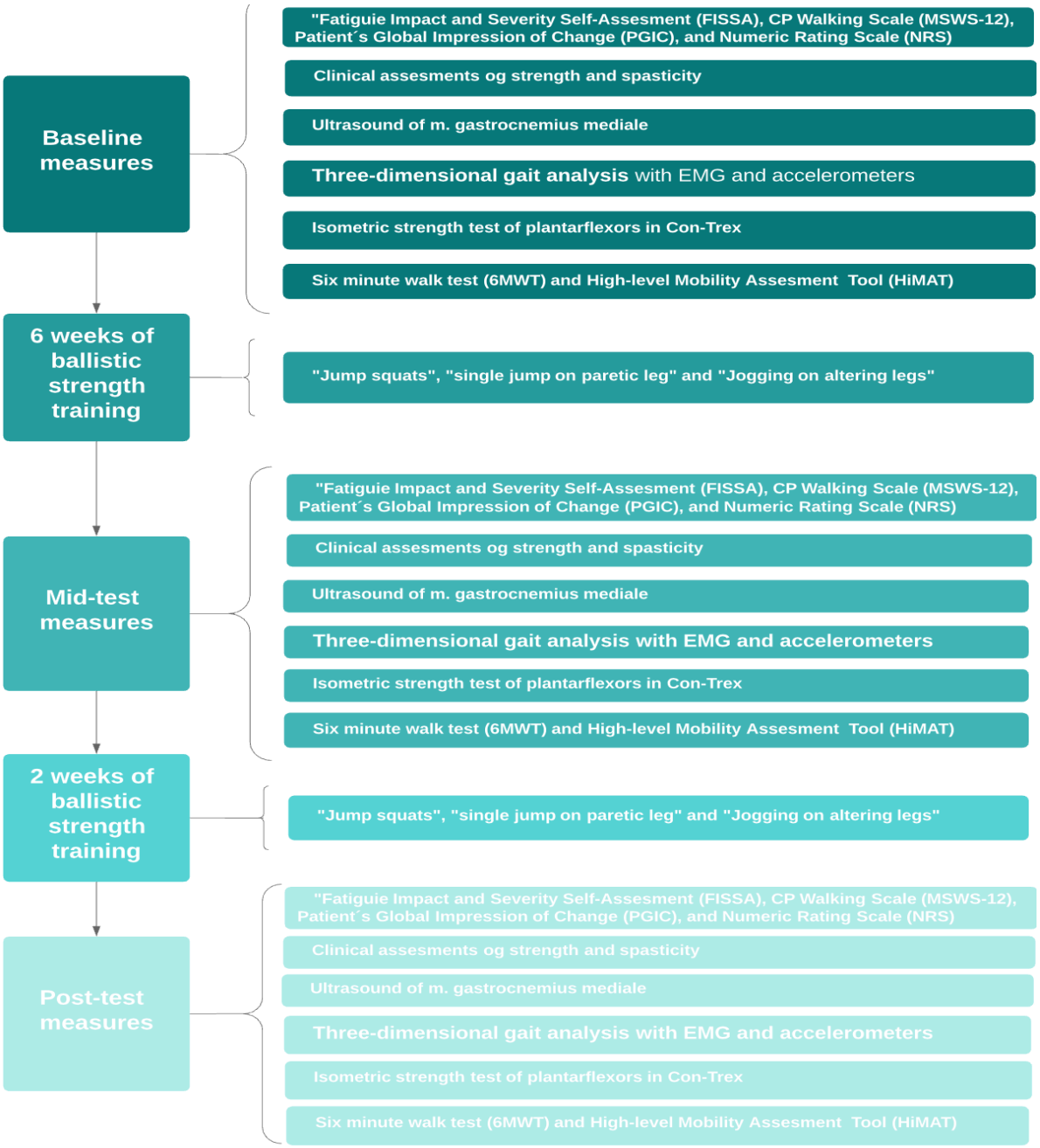


Figure 8 - *Experimental protocol.*

4.6.1 Kinematic measures

4.6.1.1 Marker placement

In order to calculate joint rotation angles during walking, the lower extremity must be modelled as a series of linked segments. Each body segment is defined by reflective markers attached in an accurate standardized position relative to anatomical landmarks on the skin. A minimum of three markers is required to define each body segment. The motion analysis system measures the center of the marker. If the marker is visible for two or more cameras, it is possible to reconstruct its position in the room. Therefore, precise marker placement is critical to obtain accurate data. The placement of the markers is dependent on the model used to compute the joint kinematics. In this project, kinematic data was measured using the "CGM lower-body marker set", whereas the underlying skeletal model was scaled behalf of anthropometric data (123,164,165). Twenty 14mm reflective markers were attached to following anatomical segments; anterior superior iliac spine (ASIS), posterior superior iliac spine (PSIS), thigh, lateral femoral condyle, lateral tibia, lateral malleoli, head of 2^{nd} metatarsal, head of 5^{th} metatarsal, base of 2^{nd} metatarsal, calcaneus on both left and right limb in the dynamic gait trials. A pencil was used to mark the location of the expected center of marker placement on the standing subject. If a marker where fell off, it was easily placed at the same location. Yet, in the static measurement, an additional marker was placed on the medial femoral condyle and at the medial malleoli, giving a total of 24 reflective markers. Complete marker placement is listed in appendix 8.

4.6.1.2 Defining pelvis segment

The pelvis forms a rigid triangular segment created by the left and right ASIS, and the left and right PSIS (33). Origin of pelvis segment is midway between left and right ASIS. A special vector in the pelvic coordinate axis system defines location of hip joint center using pelvis size and leg length (36).

4.6.1.3 Defining hip segment

The thigh forms a three-dimensional rigid body segment created by the hip joint center, a marker on the lateral thigh and a marker on the lateral femoral condyle (33). Origin of thigh segment is knee joint center. The knee is a distance of knee offset from lateral femoral condyle in direction of segment plane (36).

4.6.1.4 Defining knee segment

The lower leg forms a rigid segment created by the knee joint center, a marker on the lateral lower leg and a marker on the lateral malleolus (33). During static calibration, a marker was placed on the medial epicondyle to create the knee joint center. Origin of tibia segment is ankle joint center. Ankle is at a distance of ankle offset from lateral malleolus in direction of segment plane (36).

4.6.1.5 Defining the ankle segment

The foot forms a rigid segment created by the ankle joint center, a marker on the calcaneus and a marker on the 2nd metatarsal (33). Origin of foot segment is 2nd metatarsal (36).

4.6.1.6 Anthropometric Measures

In order to estimate joint center locations and define segment coordinate axis systems for each subject, it is necessary to obtain some anthropometric measurements of the subject. These parameters include height, weight, tibial length, distance between the femoral condyles, distance between the malleoli, and distance between the ASIS (4,21).

4.6.2 Kinetic measures

With a force plate device, calculations of ground-reaction forces, joint moments and powers of each joint can be conducted to estimate gait events such as “Right on, and right off” (4). Ground reaction force data was captured with one force plate integrated in the middle of a 7-meter walkway, capable of capturing X, Y and Z force components sampling on 2100 Hz. Threshold for activation of the force plates was set to 10 N.

4.6.3 Calibration

Two types of three-dimensional calibration of capture volume was conducted prior collection of gait data each test day. First, a dynamic calibration was conducted to establish locations and orientations of cameras within the capture volume and the size of the measurement units. This was accomplished by waving the calibration wand around the L-frame positioned on the force plate up to estimated ASIS which is the highest marker on the subject (166). The calibration time was set to a period of 60 seconds to ensure an approved capture volume. Secondly, a static calibration was conducted prior the gait analysis. Participants were asked to adopt upright standing position to establish position of a global coordinate axis system within the capture volume, location of the origin (0, 0, 0) and direction of three orthogonal coordinate axes (x, y, z).

4.6.4 Performing gait trials

Before recording experimental trials, participants performed a few gait trials to ensure they were comfortable walking barefoot on the walkway. All participants were asked to walk in preferred gait speed to allow natural gait strategies, freely adjust step length, step width and cadence. In order to gain a representative sample of all participants gait pattern, spatiotemporal, kinematic, and kinetic data from three trials at each limb was captured, checked and considered for the analysis.

4.7 Analysis of gait data

4.7.1 Calculation of joint kinematics

Variables extracted from the kinematic analyses were ankle and hip joint angles during gait. The midpoint of the reflective markers attached to the subjects were used to calculate the connecting line between different segments. The markers were labelled and their trajectory for each recording was examined in QTM. Intermittent trajectories were automatically gap filled up to 30 frames and manually assessed in order ensure they represented the correct path. In total, 150 dynamic gait trials and 25 static trials were analyzed. All data were exported to .c3d-files for further analysis in Visual3D (C-Motion Inc, Rockville, MD, USA) when the processing was finished.

4.7.2 Calculation of joint kinetics

Gait events were estimated based on the ground reaction forces. Initial contact was identified as the first frame which ground reaction force exceeded 10N, while “foot off” was the first frame which the ground reaction force was less than 10N. The mid-stance phase was identified as the frame corresponding to a midpoint between the “foot-on” and “foot-off” events. In these analyses, trials were excluded if a foot did not make a clear hit on the force plate, easily discovered when the foot was outside the plate. Trials were also excluded if the impact peak on the vertical ground reaction force signal was larger than the active peak. After selecting three approved gait cycles from the kinetic analyses, the data collection was ready for calculation of power generation in the sagittal plane using Microsoft Office Excel 2016 (Microsoft Cooperation, Redmond, WA, USA) (167). The main interest was especially peak power at push-off in the ankle (A2) and hip joint (H3) to describe the ankles ability to propel the body forward in relation to the hip (A2/H3 ratio). The power values was labeled according to the protocol of Eng and Winter (4). H1 describes hip extensors power generation during early stance phase, H2 is hip joint power absorption, while H3 is hip flexors power generation during late stance. Further, A1 is ankle plantar flexor power absorption, while A2 is ankle plantar flexor power generation during late stance. To assess the relative power of the ankle to the hip in the late stance (A2/H3 ratio), ankle plantar flexor power generation (A2) was divided on hip flexor power (H3) (46). The values for power (W) was normalized with respect to the participants body weight (kg) to determine the total amount of positive work done by each joint (1). All graphs obtained from gait analysis were normalized in time for % of gait cycle.

5.0 Manuscript following guidelines for authors of Elsevier Gait & Posture

Ballistic strength training changes the kinetic relationship between the ankle and hip joint in late stance of gait in mildly affected adults with spastic cerebral palsy

A case series of plantar flexor training primarily prescribed to most paretic limb

Silje Marie Rydningen Torberntsson
Bergen University, Kalfarveien 31, 5018 Bergen, Norway
Western Norway University of Applied Sciences, Inndalsveien 28, 5063 Bergen, Norway

Highlights:

- **Adults with spastic hemiplegic and diplegic cerebral palsy increase hip flexor power to compensate for reduced ankle plantar flexor power in late stance of gait**
- **Ballistic strength training appears to alter power output in the ankle plantar flexors**
- **A2/H3 ratio seems to increase among adults with spastic cerebral palsy following ballistic strength training, suggesting a more efficient gait pattern with less hip flexor compensation**

Abstract:
Background: In normal gait, the ankle plantar flexors provide most propulsive energy during push-off with smaller contribution of hip flexors. Yet the interplay between these two joints remains unclear in spastic cerebral palsy. The objective of this study was to evaluate the kinetic relationship between the ankle and hip joint in late stance of gait (A2/H3) in mildly affected adults with spastic cerebral palsy. A ballistic strength training program was prescribed to explore whether this exercise program would exaggerate ankle plantar flexor power so the need for hip flexor power compensation would decrease, and thereby result in an increased A2/H3 ratio.

Research question: In relation to ballistic strengthening program, will A2/H3 ratio change and promote a more efficient gait pattern in mildly affected adults with spastic cerebral palsy?

Methods: 2 male and 5 women with hemiplegic and diplegic spastic cerebral palsy, GMFCS I and II completed eight-weeks with ballistic strength training mainly prescribed to most paretic limb. Three-dimensional gait analysis with a force plate device was used to investigate the impact on ankle and hip power generation in push-off before, during and after intervention. At least three gait trials at self-selected speed was analyzed for each limb to calculate peak ankle- and hip flexor power in the sagittal plane and relationship between them (A2/H3 ratio).

Results: 6/7 participants increased A2/H3 ratio on paretic limb after intervention. As expected, the change was more evident on the most paretic limb compared to uninvolved.

Significance: Findings from this study provide a better understanding of the interplay between power generation in the ankle and hip joint in spastic cerebral palsy with possible implication to clinical practice. However, the results cannot direct any casual relationships between change in A2/H3 ratio and ballistic strength training. Until evidence is found, we assume that ballistic strength training is feasible to alter A2/H3 ratio in spastic cerebral palsy, yet further investigation is needed.

Keywords: Spastic cerebral palsy, adults, ankle/hip power ratio, ballistic strength training, gait

1.0 Background and objectives

Spastic Cerebral Palsy (SCP) describes a heterogenous group of complex motor disabilities, originating from a nonprogressive lesion to the central nervous system in early development (1–3). The presence of skeletal muscle spasticity and contractures is considered the major obstacle to motor function, which occurs secondary to upper motor neuron lesions as a result of improper or absent motor command from the brain (4–7). Although the brain lesion is static, individuals with SCP has a progressive musculoskeletal pathology (8), indicating that even mildly affected adults have significant reduced strength in all major lower extremity muscle groups compared to age-matched pears (6). There is general agreement in the literature that power generation at push-off appears to be dependent on the hip rather than the ankle during late stance of gait in self-selected gait speeds in SCP (3,9–11). Increased hip joint forces may lead to an overuse injury of the hip musculature and deterioration in walking (11). In a pilot study, Ishiara and Higuchi evaluated the kinetic relationship between the ankle and hip (A2/H3 ratio) joint during push-off phase in children with SCP (12,13). They found that the A2/H3 ratios on both the hemiplegic and uninvolved side was lower than those of the healthy control. A lower A2/H3 ratio indicates a higher proportion of hip involvement compared with ankle in late stance of gait (10,11). The peak flexion moments generated of the hip was significantly higher, and ankle power generation was significantly lower compared with the healthy controls (9–11). However, it appears that no study has yet determined the interplay between ankle and hip in adults with SCP. For that reason, the motive of this research was to explore kinetic relationships between the ankle and hip joint in the late stance of gait (A2/H3) in mildly affected adults with hemiplegic and diplegic SCP. An eight-week ballistic strength training program was prescribed to increase explosive power by performing functional loaded exercises on high movement velocity. By successfully isolating muscle force exerted by gastrocnemius, preventing no or less involvement of the knee and hip during exercise, desired outcome was a more efficient gait pattern with less compensatory actions (14). We expected the largest adaptions in power output to occur in the ankle on most paretic leg during the stance phase (A2), as the exercises mainly focused on the plantar flexors in affected limb (15). In contrast, we expected no or less change in hip power generation (H3) following the intervention. By evaluating the participants ratio before, during and after undergoing eight weeks of ballistic strength training, new knowledge of the interplay between these two joints can be generated.

2.0Methods

2.1 Study design and participants

Ten adults diagnosed with SCP were recruited to attend a prospective case series study in Bergen, Norway from March to June 2019. Prior the project start, the experimental protocol was approved by Regional Committees for Medical and Health Research Ethics (REC West Norway) (2018/349) in conformity with the Declaration of Helsinki. To participate, it was required to fulfill the following inclusion criteria: [1] adult >18 years, [2] diagnosed with spastic unilateral hemiplegic or bilateral diplegic cerebral palsy and [3] high-functioning, Gross Motor Functional Classification System (GMFCS) I-II. Participants were excluded if they had [1] treatment with surgery or botulinum toxin injection less than 6 months before entering the project, [2] if they were not able to walk indoor without walking aid, or [3] if they did not complete primary and secondary school. All participants were given oral and written explanations regarding the objective of the study and gave their written consent.

2.2 Procedure

Both intervention and testing were performed in the Rehabilitation Laboratory SimArena at Western Norway University of Applied Sciences. The intervention was an eight-week ballistic strengthening program performed on a leg sled (Total Gym RS Encompass, CA, USA) inspired by Hendrey et al. (16). It involved three different exercises mainly prescribed for the plantar flexors on most paretic limb to improve ankle power output in late stance of gait (16). All subjects were offered 16 center-based workouts. Briefly, duration of a session was 30 minutes, including a short chat, warm-up for 10 minutes and three exercises lasting for 5 minutes each, described in appendices. Repetitions, load and breaks was tailored to each participant. In addition to the center-based ballistic program, each participant performed a homebased program 3-4 times a week two times a week. Training or other physiotherapy treatment outside the rehabilitation laboratory was allowed yet registered (appendix 6).

2.3 Instrumentation

Kinetic ankle and hip power data was obtained by using three-dimensional gait analysis system (Qualisys AB, Gothenburg, Sweden) with eight high-resolution cameras sampling at a rate of 120 Hz (3). Twenty 14-mm diameter reflective markers were mounted on the skin at specific locations on both lower limbs according to Conventional Gait Model (CGM) protocol (17). Ground reaction force data was captured on 2100 Hz with one force plate (Kistler Nordic AB, Jonsered, Sweden), capable of capturing X, Y and Z force components. Participants initially performed a standing static calibration trial to define joint center locations and anatomical coordinate systems. Thereafter, they were asked to walk barefoot in their preferred gait speed across a 7-meter walkway. In order to gain a representative sample of each participants gait pattern, at least three gait trials for both limbs were recorded, checked and considered for the analysis. Each subject was evaluated before, during and after eight-weeks of ballistic strength program to generate objective information related to change in plantar flexor power and A2/H3 ratio during late stance in gait.

2.4 Data analyses

Visual 3D (C-Motion Inc, Rockville, MD, USA) was used to calculate peak ankle and hip power and A2/H3 ratio in the sagittal plane. All graphs obtained from the gait analysis are presented in percent of gait cycle (%). Kinetic power (W) data are normalized for individual body weight (kg) and labelled according to Eng and Winter (18). H3 indicates hip flexor power generation while A2 indicates ankle plantar flexor power generation during late stance (18). To further assess the ratio between ankle and hip power generation (A2/H3), peak ankle power was divided on hip peak value using Microsoft Office Excel 2016 (Microsoft Cooperation, Redmond, WA, USA). A2/H3 ratio represents the proportions of ankle plantar flexion and hip flexion during the push-off phase, where a higher ratio indicates a greater proportion of ankle involvement compared to the hip.

3.0 Results

3.1 Included participants

Several individuals reported interest in participating in this project. 10 of these potential subjects were recruited based on inclusion and exclusion criteria. One hemiplegic man dropped out before baseline testing because of the timespan of the project. A woman dropped out after 7 sessions with ballistic strength training because of a foot inflammation. One diplegic man was not able to attend at mid-term testing due to sickness. However, his data collected at baseline and after intervention are presented in the results. Due to technical issues, 7 participants were available for analysis. The recruited study sample characteristic before any dropouts are shown in table 1. The average of completed training sessions for the 10 recruited participants were 12.7±5.3. On the first exercise "Jump squats", mean number of repetitions were 202.4±18.76 with a slope on 12.5±2.47°. On exercise 2 "Single jump on paretic leg" mean number of repetitions were 264.3±25.86, with a slope on 8.1±10.36°, resulting on a subjective leg fatigue of 13.7±2.01 on Borg scale 6-20. On the last exercise "Jogging on altering legs" mean number of repetitions were 352.35±80.42, with a slope on 12.2±2.90°, resulting in a subjective leg fatigue of 14.5±1.73 on Borg scale 6-20.

Table 1 - *Included participants, characteristics, anthropometric measurements and diagnosis description.*

ID (n)	Gender	Age (years)	Weight (kg)	Height (cm)	Diagnosis	(most) Paretic side	GMFCS	Completed sessions (n)
FP1	Woman	27	49	167	Hemiplegia	Right	I	13
FP2	Woman	51	64	168	Hemiplegia	Left	I	15
FP3	Woman	28	78	170	Hemiplegia	Left	I	16
FP4	Woman	30	67	165	Hemiplegia	Left	I	7
FP5	Man	53	97	179	Diplegia	Right	II	15
FP6	Man	34	80	168	Diplegia	Left	II	13
FP7	Woman	24	57	154	Hemiplegia	Left	I	16
FP8	Man	24	76	178	Hemiplegia	Right	1	0
FP9	Woman	30	98	160	Hemiplegia	Left	I	16
FP10	Man	56	95	183	Hemiplegia	Right	II	16
10	4/6	35.7±12.6	76.1±17.1	169.2±8.9	2/8	4/6	3/7	12.7±5.3

Kilos (kg), height (cm), number (n).

3.2 Change in A2/H3 ratio following ballistic strength program

Although there was no control group in this study, the results have been compared with previous findings on healthy individuals (18,19). The results illustrated in table 2 indicate that A2/H3 ratio of adults with SCP are lower than the general healthy population, meaning that a higher propulsion of power is generated by the hip compared to the ankle during walking. After completed intervention, 6/7 participants increased A2/H3 ratio on paretic limb at either midterm, post-test or both compared to baseline. Mean values for the paretic leg at start-up was 1.91±0.84, during the intervention it was 2.00±0.72 and after it was 2.40±1.07. To compare, 5/7 participants increased A2/H3 ratio at either midterm, post-test or both compared to baseline on uninvolved limb. Mean values at start-up was 1.93±1.06, during intervention it was 2.02±0.57 and after it was 1.93±0.70.

Table 2 - *Peak ankle and hip ratio (A2/H3 ratio) on both most paretic and uninvolved limb before, during and after ballistic strength training of the ankle plantar flexors.*

	Subject	Before training	During training	After training
A2/H3 ratio (W/kg) most paretic limb	FP1	1.56	1.39	1.91
	FP2	2.37	2.02	3.18
	FP3	1.01	1.62	1.88
	FP5	1.48	-	1.63
	FP6	2.22	3.35	4.44
	FP7	1.25	1.49	1.41
	FP10	3.45	2.14	2.34
M(SD)		1.94±0.78	2.00±0.72	2.40±1.07
A2/H3 ratio (W/kg) uninvolved limb	FP1	1.74	1.49	1.92
	FP2	1.92	1.88	2.19
	FP3	2.37	2.51	2.26
	FP5	1.03	-	0.96
	FP6	1.26	1.45	0.99
	FP7	4.08	2.88	2.80
	FP10	1.13	1.91	2.37
M(SD)		1.93±0.98	2.02±0.57	1.93±0.70

A2/H3 power ratio represents the ratio of A2 to H3.

All data are presented as mean (SD).

3.3 Self-selected gait speed

Gait speed is a well-known indicator of overall gait performance, and reduced power output during gait are accompanied by shorter steps, slower speeds and reduced gait economy (20–23). As presented in table 3, the recruited sample in this study walked on average with slower self-selected speed compared to 1.2-1-4m/s in the general healthy population. However, the self-selected gait speed increased on average from 1.07m/s to 1.16m/s and 1.13m/s during and after intervention.

Table 3 - *Self-selected gait speed before, during and after ballistic strength training.*

	Subject	Before training	During training	After training
Gait speed (m/s)	FP1	1.204	1.185	1.180
	FP2	1.110	1.347	1.373
	FP3	1.246	1.275	1.239
	FP5	0.924	-	0.939
	FP6	1.202	0.923	0.984
	FP7	0.975	1.389	1.402
	FP10	0.796	0.823	0.808
M(SD)		1.07±0.17	1.16±0.23	1.13±0.23

All data are presented as mean (SD)

4.0 Discussion

This study found that mildly affected adults with spastic hemiplegic and diplegic cerebral palsy generates increased hip flexor power to compensate for reduced ankle plantar flexor power in forward ambulation. However, after attending eight-week ballistic strength program, the power output in the ankle plantar flexors were altered, suggesting less compensation with hip flexors. Corresponding to the hypothesis where we expected larger adaptions on paretic limb, 6/7 individuals increased peak A2/H3 ratio on paretic limb and 5/7 on uninvolved limb.

4.1 Interpretation of change in A2/H3 ratio

In addition to study design and methodological limitations, there are several reasons why there is challenging to give a clear conclusion regarding the change in A2/H3 ratio. To generate the results, additional multifactorial analytic studies should be initiated. In the following sections, the change in A2/H3 will be discussed in relation to spatiotemporal-, kinematic- and kinetic parameters in relation to ballistic strength training.

4.1.1 Spatiotemporal parameters

Since gait speed is known to have an impact on nearly all gait variables in the lower limbs, including muscle power requirements (24–28), it was considered relevant to include each participant self-selected speed in relation to change in A2/H3 ratio when interpreting the results. Power is the product of force and velocity (16), and it is consequently possible that if the participants increased gait speed, it is a contributing cause for altering power generation in push-off (16). In fact, 5/7 increased gait speed during or after intervention An increased gait speed demand higher hip and ankle power generation in at push-off (29) which contribute to an overall increase in step length and potentially reduce asymmetry (30). Corfe et al. (31) and Chen et al. (32) found that peak hip power generation had an influence on gait speed in ageing adults. When power output increased with 14%, gait speed also increased from 1.3m/s to 1.4m/s. Similarly, whereas as gait speed reduced from 1.3m/s to 1.2m/s these measures fell by 13%. The participants in this study were asked to walk in preferred speed during gait analysis. Compared to a healthy population, this study sample with SPC walked slower at self-selected gait. Conversely, walking at slower speeds may be mechanically less efficient and less conductive to the storage and recovery of elastic energy in the musculotendon complex (15). However, Brændvik et al. (27) reported that additional factors, especially reduced spasticity and increased muscle strength alter gait speed and step length. Some muscle groups are more sensitive to modifications in gait speed (19,33–36). The fiber work from soleus and gluteus maximus in stance, and tibialis anterior, iliopsoas and hamstrings in swing change the most with increasing speed (15). The fact that no studies has controlled gait speed during three-dimensional gait analysis in SCP may be a reason for the lack of knowledge about A2/H3 ratio and compensatory actions. When the speed varies in between trials, it makes it difficult to compare calculations of joint power across subjects and studies. However, there exist several disadvantages with controlling gait speed, especially in populations with neurological pathology where the gait pattern is rather variable regarding speed, stride width and step length (27). By controlling gait speed, it would perhaps influence the results in a way that it cannot be transferred to their everyday ambulation. Some of the included participants would even been unable to walk safely at a faster speed, yet some would have the ability to do so.

4.1.2 Kinematic parameters

After finding an altered ankle power generation (A2) and improved A2/H3 ratio in adults with SCP, it raised a new hypothesis how this may affect the kinematics. Although only a few studies have evaluated power in relation to gait kinematics, it seems reasonable that an improved A2/H3 ratio will alter the ROM in the ankle joint, and thereby result in a more efficient gait pattern. In fact, Moreau et al. (37) found a significant increase in the ankle ROM after power training. Although the change was too small to have clinical relevance, it could direct important hypothesis for understanding the changes in muscle morphology after strength training. Increased muscle length can be explained by muscle fiber hypertrophy, which could lead to increased muscle length of gastrocnemius. These facts indicate that power training may have a positive effect on muscle length and ROM of the ankle joint. Improved coordination can also be expected as a result of the repetitiveness of the functional exercises. Since the majority of participants in this study both improved A2/H3 ratio and A2 output after the ballistic strength program, it is to believe that these certain exercises altered the shortening velocity and perhaps affected the muscle architecture. Hypertrophy in a muscle may be due to an increase in muscle fiber cross section, more myofibrils in parallel and increased maximal force, or increased maximal shortening speed. Alternatively, better walking performance can be expected to occur over time when exercising. However, without analyzing the ultrasound data, it is impossible to assume if this improvement is due to increased number of sarcomeres in series, parallel, fiber type or fascicle length (38).

4.1.3 Kinetic parameters

A decreased ankle power output and an increased hip power output was prior the project valued as the most undesirable outcome following the intervention. This outcome occurred in one hemiplegic subject (FP10) on paretic limb. Such outcome may indicate that plantar flexors were not isolated, and the hip flexors were trained additionally. Previous findings suggest that training programs focusing on strengthening the weaker muscle is prevented by compensation of other less affected muscle groups. (39–41). This participants baseline A2/H3 ratios on most paretic and uninvolved limb was 3.45W/kg and 1.13W/kg respectively. Interestingly, by comparing his ratios post intervention, the A2/H3 ratios was almost similar and change to 2.34W/kg and 2.37W/kg. Equally, one other hemiplegic subject had a similar change (FP1). Prior intervention her values on both limbs were 1.56W/kg and 1.74W/kg, and

after ballistic strength training it increased to 1.91W/kg and 1.92W/kg. Hypothetically, a similar power output suggests a more symmetrical and efficient gait pattern per stride compared to baseline. In such cases, increasing hip power was perhaps not a disadvantage for gait functionality anyway. According to a previous study on children with CP training muscle strength and gait analysis, increased hip strength improves the plantar flexor power at push-off (42). Further, according to Ross and Engsberg (43) the largest variance in gait and gross motor function in a group children with CP was explained by the hip abductors followed by ankle plantar- and dorsiflexors muscle groups. These facts propose that it may be important to increase both ankle and hip power generation to maintain functional walking (44–46), although such training regime will not result in an increased A2/H3 ratio (32,42).

Even though the plantar flexors in the most paretic limb was the main focus, we expected some transfer to the uninvolved limb following ballistic strength training. After eight weeks of intervention, four participants increased A2 output on uninvolved limb, and interestingly in a greater extent compared to the paretic limb. Although two out of three exercises were targeting both limbs, this outcome was surprising since the task with the highest relevance only focused on the most paretic limb. However, a possible explanation may be the fact that dysfunction on the affected side tends to be compensated for by altering power non-affected side.

5.0 Conclusions and significance

To our knowledge, this is the first study investigating the impact of ballistic strength training and the relationship between power generation in the ankle and hip in adults with spastic cerebral palsy. In conclusion, this study displays possible advantages with this rehabilitation program to alter A2/H3 ratio. However, due to some methodological limitations, small study sample and a lack of control group, these results can only direct hypothesis for a future analytic study. In order to generalize the results, further investigation with high level of evidence should commence. They should recruit a larger study sample with different GMFCS levels and evaluate effect over extended periods of time. In addition, studies should include whether pathological differences between hemiplegia and diplegia require different treatment. It will also be interesting to assess the optimal time interval, frequency and intensity of ballistic strength training.

Conflict of interest statement

The author confirms that there are no known conflicts of interest regarding the work described in the current manuscript.

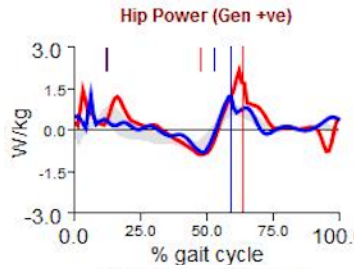
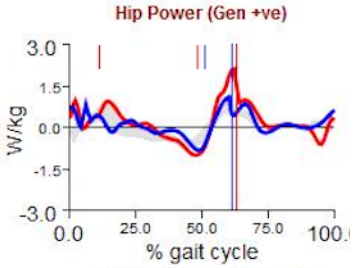
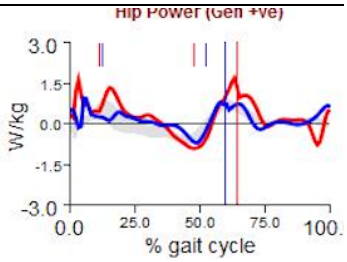
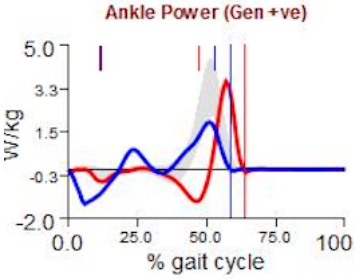
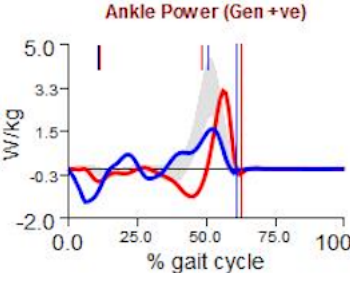
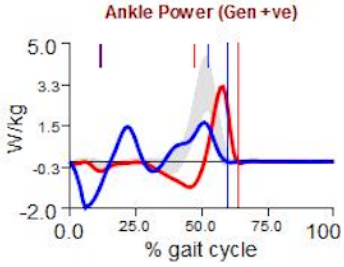
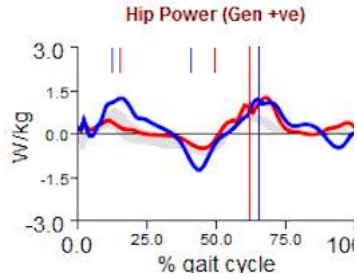
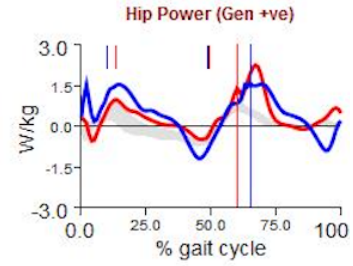
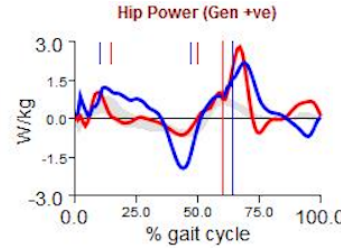
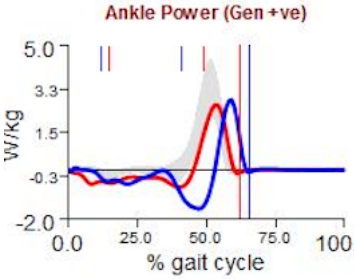
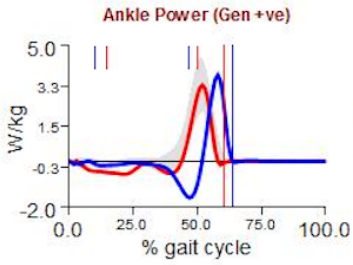
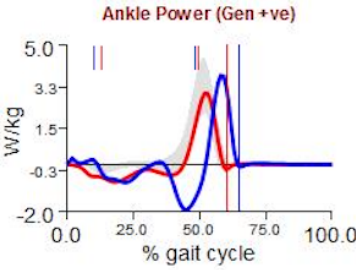
Acknowledgment

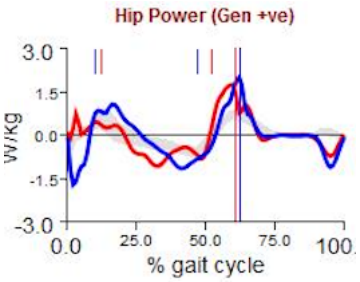
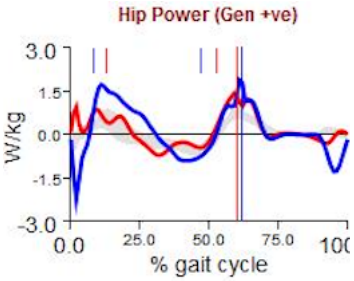
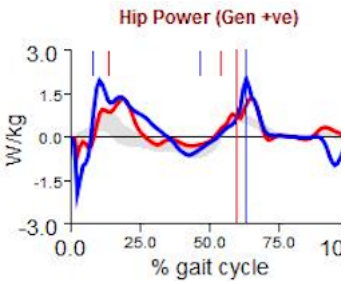
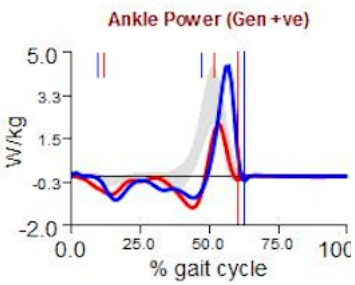
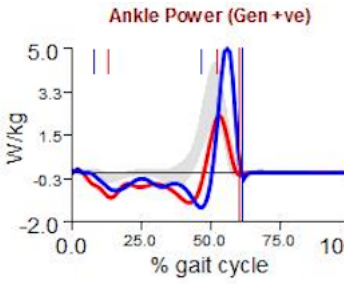
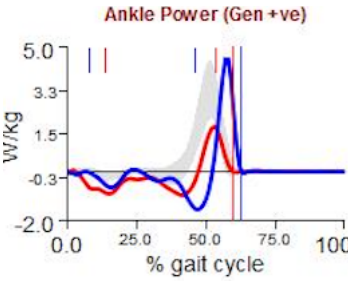
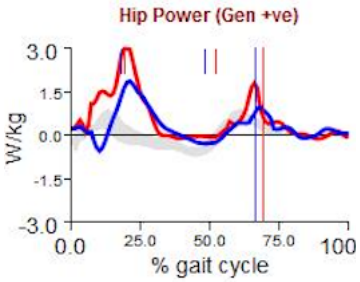
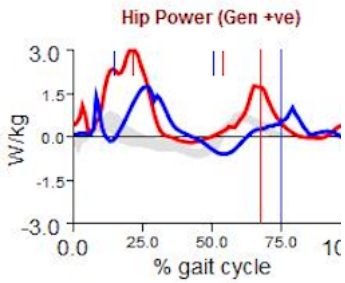
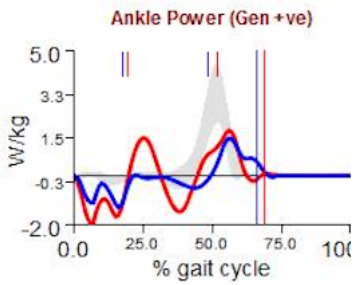
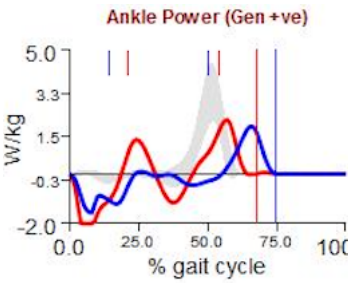
The author would like to acknowledge Dr. Silje Mæland, MSc. Beate Eltarvåg Gjesdal, MSc. Lars Peder Vatshelle Bovim and Dr. Bård Erik Bogen. Data collection for this study was conducted in the Rehabilitation Lab of SimArena, Western Norway University of Applied Sciences (https://www.hvl.no/om/simarena/rehabiliteringslab-f327/).

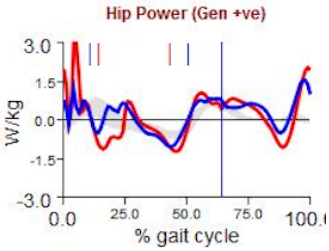
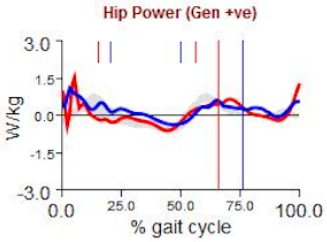
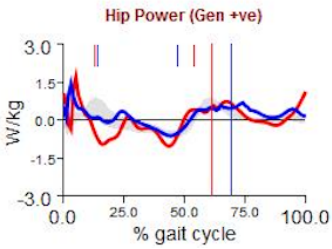
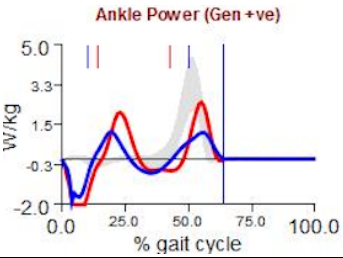
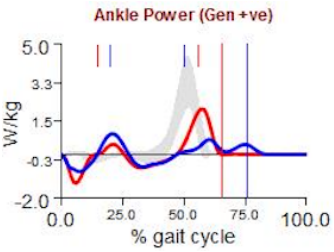
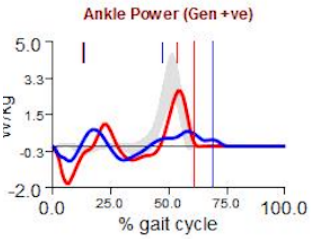
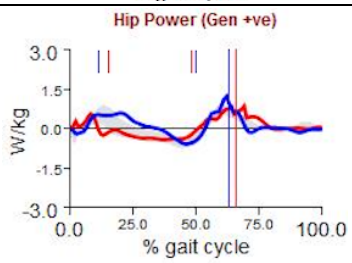
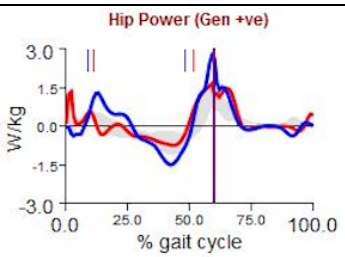
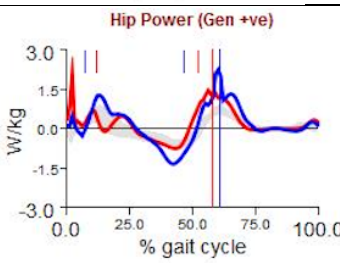
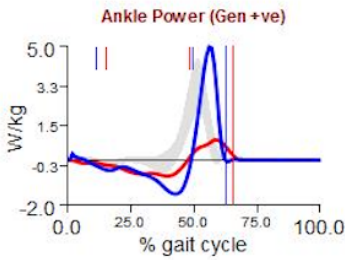
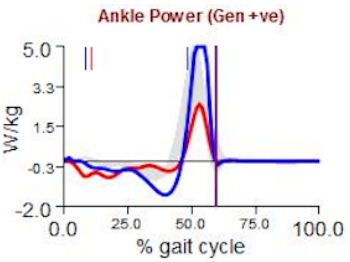
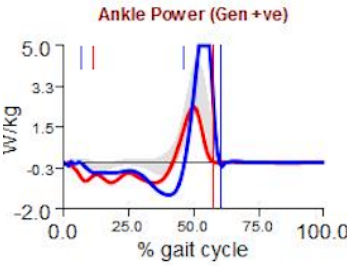
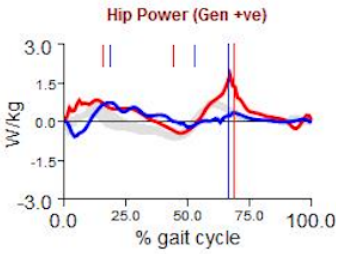
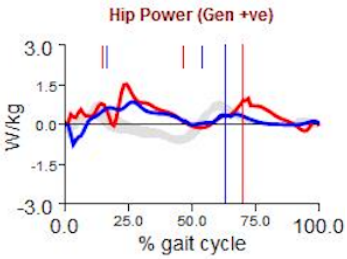
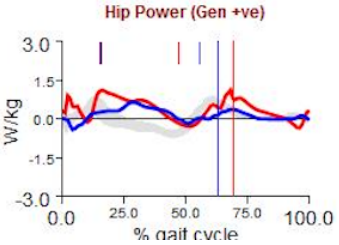
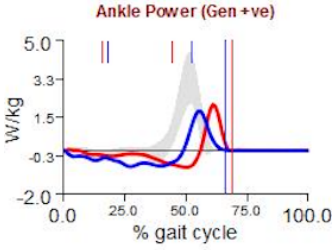
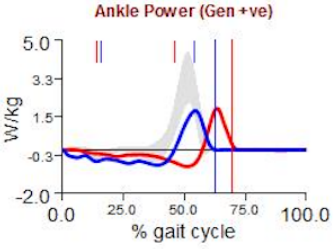
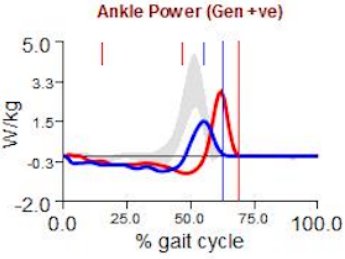
Appendix: Table with graphs of ankle and hip power generation

All graphs presented in the table below are made in Visual 3D, presented in rows and columns. The rows represent each participant in order of ID-number and their hip and ankle power output, while the columns represent outcome at baseline testing, during, and after 8 week of ballistic strength training.

Table 4- *Kinetic graphs of ankle and hip power generation normalized for 100% of gait cycle for both right and left limb.*

Subject		Baseline	During intervention	After intervention
FP1	Hip power (H3)			
	Ankle Power (A2)			
FP2	Hip power (H3)			
	Ankle Power (A2)			

Subject		Baseline	During intervention	After intervention
FP3	Hip power (H3)			
	Ankle Power (A2)			
FP5	Hip power (H3)		Sick, did not participate at mid-test	
	Ankle Power (A2)		Sick, did not participate at mid-test	

Subject		Baseline	During intervention	After intervention
FP6	Hip power (H3)			
	Ankle Power (A2)			
FP7	Hip power (H3)			
	Ankle Power (A2)			
FP10	Hip power (H3)			
	Ankle Power (A2)			

6.0 References

1. Ballaz L, Plamondon S, Lemay M. Ankle range of motion is key to gait efficiency in adolescents with cerebral palsy. Clin Biomech. 2010;25(9):944–8.
2. Martins E, Cordovil R, Oliveira R, Letras S, Lourenço S, Pereira I, et al. Efficacy of suit therapy on functioning in children and adolescents with cerebral palsy: a systematic review and meta-analysis. Developmental Medicine & Child Neurology. 2016 Apr 1;58(4):348–60.
3. Armand S, Decoulon G, Bonnefoy-Mazure A. Gait analysis in children with cerebral palsy. EFORT Open Rev. 2016 Dec;1(12):448–60.
4. Jack R. Engsberg, Sandy A. Ross, Tae Sung Park. Changes in ankle spasticity and strength following selective dorsal rhizotomy and physical therapy for spastic cerebral palsy. Journal of Neurosurgery. 1999;91(5):727–32.
5. Jack R. Engsberg, Kenneth S. Olree, Sandy A. Ross, T. S. Park. Spasticity and strength changes as a function of selective dorsal rhizotomy. Journal of Neurosurgery. 1998;88(6):1020–6.
6. Wiley ME, Damiano DL. Lower-Extremity strength profiles in spastic cerebral palsy. Developmental Medicine & Child Neurology. 1998 Feb 1;40(2):100–7.
7. Damiano DL, Martellotta TL, Quinlivan JM, Abel MF. Deficits in eccentric versus concentric torque in children with spastic cerebral palsy. Med Sci Sports Exerc. 2001 Jan;33(1):117–22.
8. Graham HK, Rosenbaum P, Paneth N, Dan B, Lin J-P, Damiano DL, et al. Cerebral palsy. Nature Reviews Disease Primers. 2016 Jan 28;2:16005.
9. Eek MN, Tranberg R, Beckung E. Muscle strength and kinetic gait pattern in children with bilateral spastic CP. Gait & Posture. 2011;33(3):333–7.
10. Ishihara M, Higuchi Y, Yonetsu R, Kitajima H. Plantarflexor training affects propulsive force generation during gait in children with spastic hemiplegic cerebral palsy: a pilot study. J Phys Ther Sci. 2015 May;27(5):1283–6.
11. Ishihara M, Higuchi Y. Kinetic Relationships between the Hip and Ankle Joints during Gait in Children with Cerebral Palsy: A Pilot Study. J Phys Ther Sci. 2014 May;26(5):737–40.
12. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. Clinical Biomechanics. 1999 Feb 1;14(2):125–35.
13. Milot M-H, Nadeau S, Gravel D, Bourbonnais D. Effect of increases in plantarflexor and hip flexor muscle strength on the levels of effort during gait in individuals with hemiparesis. Clin Biomech (Bristol, Avon). 2008 May;23(4):415–23.
14. Tateuchi H, Tsukagoshi R, Fukumoto Y, Oda S, Ichihashi N. Immediate effects of different ankle pushoff instructions during walking exercise on hip kinematics and kinetics in individuals with total hip arthroplasty. Gait & Posture. 2011 Apr 1;33(4):609–14.
15. Neptune RR, Sasaki K, Kautz SA. The effect of walking speed on muscle function and mechanical energetics. Gait Posture. 2008 Jul;28(1):135–43.
16. Hendrey G, Clark RA, Holland AE, Mentiplay BF, Davis C, Windfeld-Lund C, et al. Feasibility of Ballistic Strength Training in Subacute Stroke: A Randomized, Controlled, Assessor-Blinded Pilot Study. Arch Phys Med Rehabil. 2018 Dec;99(12):2430–46.
17. Baker R, Leboeuf F, Reay J, Sangeux M. The Conventional Gait Model : The Success and Limitations. In 2017. p. 1–19.
18. Eng JJ, Winter DA. Kinetic analysis of the lower limbs during walking: what information can be gained from a three-dimensional model? J Biomech. 1995 Jun;28(6):753–

8.
19. Olney SJ, MacPhail HE, Hedden DM, Boyce WF. Work and power in hemiplegic cerebral palsy gait. Phys Ther. 1990 Jul;70(7):431–8.
20. DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. J Appl Physiol (1985). 2000 May;88(5):1804–11.
21. Farris DJ, Hampton A, Lewek MD, Sawicki GS. Revisiting the mechanics and energetics of walking in individuals with chronic hemiparesis following stroke: from individual limbs to lower limb joints. Journal of NeuroEngineering and Rehabilitation. 2015 Feb 27;12(1):24.
22. JudgeRoy JO, Davis B III, Õunpuu S. Step Length Reductions in Advanced Age: The Role of Ankle and Hip Kinetics. The Journals of Gerontology: Series A. 1996 Nov 1;51A(6):M303–12.
23. Winter DA, Patla AE, Frank JS, Walt SE. Biomechanical Walking Pattern Changes in the Fit and Healthy Elderly. Physical Therapy. 1990 Jun 1;70(6):340–7.
24. Chehab EF, Andriacchi TP, Favre J. Speed, age, sex, and body mass index provide a rigorous basis for comparing the kinematic and kinetic profiles of the lower extremity during walking. J Biomech. 2017 Jun 14;58:11–20.
25. Farris DJ, Sawicki GS. The mechanics and energetics of human walking and running: a joint level perspective. Journal of the Royal Society Interface. 2012;9(66):110–8.
26. Ebrahimi A, Goldberg SR, Stanhope SJ. Changes in relative work of the lower extremity joints and distal foot with walking speed. J Biomech. 2017 Jun 14;58:212–6.
27. Braendvik SM, Roeleveld K, Andersen GL, Raftemo AER, Ramstad K, Majkic-Tajsic J, et al. The WE-Study: does botulinum toxin A make walking easier in children with cerebral palsy?: Study protocol for a randomized controlled trial. Trials. 2017 Feb 6;18(1):58.
28. Schwartz MH, Rozumalski A, Trost JP. The effect of walking speed on the gait of typically developing children. J Biomech. 2008;41(8):1639–50.
29. Eerdekens M, Deschamps K, Staes F. The impact of walking speed on the kinetic behaviour of different foot joints. Gait Posture. 2019 Feb;68:375–81.
30. Requião LF, Nadeau S, Milot MH, Gravel D, Bourbonnais D, Gagnon D. Quantification of level of effort at the plantarflexors and hip extensors and flexor muscles in healthy subjects walking at different cadences. Journal of Electromyography and Kinesiology. 2005 Aug 1;15(4):393–405.
31. Cofre LE, Lythgo N, Morgan D, Galea MP. Aging modifies joint power and work when gait speeds are matched. Gait Posture. 2011 Mar;33(3):484–9.
32. Chen IH, Kuo KN, Andriacchi TP. The influence of walking speed on mechanical joint power during gait. Gait & Posture. 1997 Dec 1;6(3):171–6.
33. Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: Characteristics. Gait & Posture. 1996;4(2):136–48.
34. Olney SJ, Griffin MP, McBride ID. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. Phys Ther. 1994 Sep;74(9):872–85.
35. Olney SJ, Griffin MP, Monga TN, Mcbride ID. Work and power in gait of stroke patients. Archives of physical medicine and rehabilitation. 1991;72(5):309.
36. Lelas JL, Merriman GJ, Riley PO, Kerrigan DC. Predicting peak kinematic and kinetic parameters from gait speed. Gait Posture. 2003 Apr;17(2):106–12.
37. Moreau NG, Holthaus K, Marlow N. Differential adaptations of muscle architecture to high-velocity versus traditional strength training in cerebral palsy. Neurorehabil Neural Repair. 2013 May;27(4):325–34.
38. Thorstensson A, Karlsson J, Viitasalo JHT, Luhtanen P, Komi PV. Effect of Strength Training on EMG of Human Skeletal Muscle. Acta Physiologica Scandinavica. 1976 Oct

1;98(2):232–6.
39. Perry, J. The use of gait analysis for surgical recommendations in traumatic brain injury. J Head Trauma Rehabil. 1999 Apr;14(2):116–35.
40. Esquenazi A. Evaluation and Management of Spastic Gait in Patients With Traumatic Brain Injury. Journal of Head Trauma Rehabilitation. 2004;19(2):109–18.
41. Hosalkar H, Goebel J, Reddy S, Pandya NK, Keenan MA. Fixation techniques for split anterior tibialis transfer in spastic equinovarus feet. Clin Orthop Relat Res. 2008 Oct;466(10):2500–6.
42. Eek MN, Tranberg R, Zügner R, Alkema K, Beckung E. Muscle strength training to improve gait function in children with cerebral palsy. Developmental Medicine & Child Neurology. 2008;50(10):759–64.
43. Ross SA, Engsberg JR. Relationships between spasticity, strength, gait, and the GMFM-66 in persons with spastic diplegia cerebral palsy. Arch Phys Med Rehabil. 2007 Sep;88(9):1114–20.
44. Winter DA. Biomechanical Motor Patterns in Normal Walking. Journal of Motor Behavior. 1983 Dec 1;15(4):302–30.
45. Stansfield W Ben, Hillman J Susan, Hazlewood E M, Lawson A Alastair, Mann M Alison, Loudon R Ian, et al. Sagittal Joint Kinematics, Moments, and Powers Are Predominantly Characterized by Speed of Progression, Not Age, in Normal Children. Journal of Pediatric Orthopaedics. 2001;21(3):403–11.
46. Van Der Linden L Mariëtta, Kerr M Alison, Hazlewood E M, Hillman J Susan, Robb E James. Kinematic and Kinetic Gait Characteristics of Normal Children Walking at a Range of Clinically Relevant Speeds. Journal of Pediatric Orthopaedics. 2002;22(6):800–6.

Chapter 6: Additional findings

When using 3DGA to explore A2/H3 ratio in a population with SCP, several gait parameters can contribute to explain a change in gait and compensatory actions. These include kinetic-, kinematic- and spatiotemporal gait parameters. Yet not included in the scientific article, additional findings listed in this chapter can possible be supply causes of the results and contribute to new hypothesis. Figure 9 illustrate how much each participant change A2/H3 ratio on most paretic limb after 8-weeks of ballistic strength training.

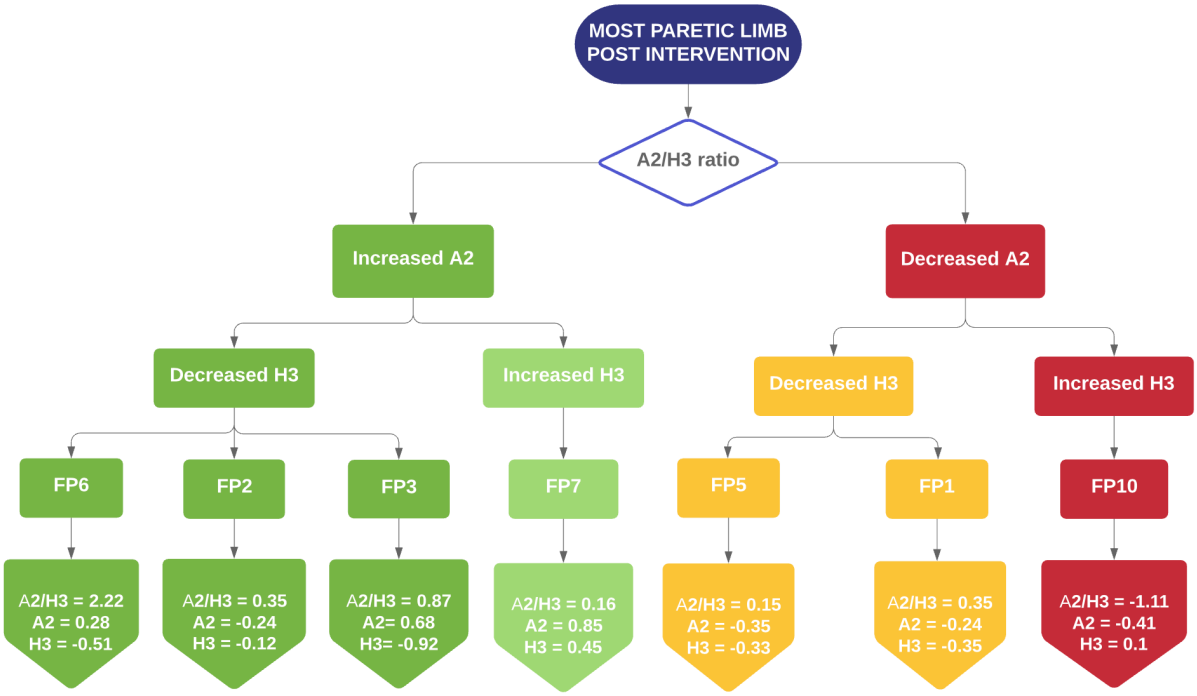


Figure 9 - *Each participant individual change in A2/H3 ratio on paretic limb after 8-weeks of ballistic strength training*

6.1 Peak ankle and hip power in late stance of gait

Although there was a remarkable difference in power output between paretic and uninvolved limb in the study population at baseline, we found larger improvements in ankle plantar flexor power generation (A2) in most affected limb compared to less affected limb after intervention. Table 3 presents the power output before, during and after ballistic strength training. On average in most paretic leg, A2 increased with 0.24W/kg, and H3 decreased with 0.15W/kg from baseline to post test. A2 on the uninvolved limb increased with 0.15W/kg, and H3 decreased with 0.01W/kg from baseline to post test.

Table 3 - *Peak ankle plantar flexor (A2) and hip flexor (H3) power generation values in the late stance phase of gait.*

	Subject	Before training	During training	After training
A2, peak power (W/kg) affected limb	FP1	1.94	1.63	1.70
	FP2	2.74	3.09	3.31
	FP3	1.94	2.43	1.89
	FP5	2.35	-	2.07
	FP6	2.53	2.15	2.81
	FP7	5.31	6.69	6.44
	FP10	2.31	2.27	2.94
M(SD)		2.04±0.85	2.28±0.53	2.28±0.64
A2, peak power (W/kg) uninvolved limb	FP1	3.86	3.17	3.40
	FP2	2.89	3.79	3.85
	FP3	4.67	5.35	4.69
	FP5	2.10	-	2.28
	FP6	1.46	1.27	0.96
	FP7	1.73	2.53	2.58
	FP10	2.00	1.83	1.59
M(SD)		3.36±1.37	3.76±1.99	3.51±1.75
H3, peak power (W/kg) affected limb	FP1	1.24	1.18	0.89
	FP2	1.16	1.53	1.04
	FP3	1.92	1.50	1.00
	FP5	1.59	-	1.26
	FP6	1.16	0.88	0.98
	FP7	1.30	2.88	2.30
	FP10	0.58	0.86	0.68
M(SD)		1.30±0.39	1.24±0.42	1.05±0.41
H3, peak power (W/kg) uninvolved limb	FP1	2.21	2.13	1.77
	FP2	1.50	2.02	1.76
	FP3	1.97	2.13	2.08
	FP5	2.03	-	2.38
	FP6	1.14	0.64	0.63
	FP7	1.38	1.70	1.83
	FP10	2.04	1.19	1.24
M(SD)		1.80±0.42	1.87±0.72	1.79±0.52

A2 represents ankle plantar flexion power generation during late stance.

H3 represents hip flexion power generation.

All data are presented as mean (SD)

6.2 Exercise log of ballistic strength training

Table 4 illustrate mean number of repetitions, slope and subjective experience of fatigue for the study sample. The slope on the led sled when exercising "Jumping squats" was on average 12.5±2.47 degrees and 202.4±18.76 repetitions among the participants. On "Single leg jump on paretic leg", the average repetitions were 264.3±25.86 repetitions, and a slope on 8.1±10.36 degrees. On the last exercise, "Jogging on altering legs", the average was 352.35±80.42 repetitions with a slope on 12.2±2.90 degrees. Borg Scale was 13.7±2.01.

Table 4 - *Mean number of repetitions, slope and Borg scale (6-20) the participants expressed after each session with three different ballistic strength exercises.*

Exercise	Subject	M Repetitions (n)	M slope (°)		
Jump squats	FP1	188.7	11.2		
	FP2	222.2	10.9		
	FP3	204.2	13.2		
	FP4	214.1	17.8		
	FP5	204.4	12.6		
	FP6	170.3	11.8		
	FP7	192.4	10.7		
	FP9	196.2	14.6		
	FP10	229.3	9.6		
M(SD)		202.4±18.76	12.5±2.47	Borg Leg 6-20	
Single jump on paretic leg	FP1	312.4	6.6	10.5	
	FP2	240.5	8.3	11.5	
	FP3	250.3	9.9	12.6	
	FP4	244.4	9.8	16.2	
	FP5	257.2	6.6	14.5	
	FP6	242.5	7.6	16.6	
	FP7	275.6	7.6	15.3	
	FP9	257.4	11.0	14.5	
	FP10	298.6	5.1	13.9	
M(SD)		264.3±25.86	8.1±10.36	13.7±2.01	
				Borg leg	Borg body
Jogging on altering legs	FP1	365.6	10.3	13.5	13.1
	FP2	327.0	9.9	12.7	12.9
	FP3	329.3	13.3	12.0	12.4
	FP4	381.0	18.6	14.8	14.8
	FP5	428.8	12.6	16.0	16.2
	FP6	262.2	10.6	14.5	16.9
	FP7	352.6	12.9	15.7	14.6
	FP9	312.8	13.0	15.5	14.5
	FP10	411.9	8.7	13.9	15.1
M(SD)		352.35±80.42	12.2±2.90	14.47±1.73	14.46±1.61

All data are presented as mean (SD)

Chapter 7: Methodological and process considerations

There are several reasons why it is challenging to reach a clear conclusion about the outcome regarding the relationship between power generation in hip and ankle before and after undergoing ballistic strength training. In this chapter, methodological issues will be discussed related to research design, study sample, confounding variables, exposure and analysis.

7.1 Case series study design

It is important to state that the findings in this study are preliminary to generate conclusions regarding treatment efficacy of ballistic strength training on A2/H3 ratio due to the choice of research design. An exploratory case series design is rated lowest in the pyramid of validity (159,168). When a study lacks a control group, no causal relationships can be drawn between treatment and outcome as it is impossible to determine whether the outcomes can be attributed to treatment or patient characteristics (161,163). Additionally, short intervention time and a small and varied study sample may influence the results in this study. In the hierarchy of evidence, blinded randomized controlled trials are considered as gold standard for quantifying efficacy of specific treatment compared to a control group. For instance, it would have been interesting to compare the results from ballistic strength training to a match group training traditional strength training as it is the most prescribed approach in clinical practice. Although RCTs minimize the role of confounding bias and optimizing internal validity, such experimental studies are expensive, time-consuming, demanding to administer, and associated with ethical issues. Despite the methodological limitations in case series, such research design was considered relevant to generate hypothesis of the kinetic interplay between the ankle and hip joint for a future analytic study. Moreover, it is a convenient approach when there is little knowledge about the ballistic strength program and when the aim is to investigate the outcome of a small, heterogenic group with numerous of neuromuscular impairments prospectively.

7.2 Study setting and sample

In clinical research, the intention is to generate new knowledge. By including a wide range of individuals, the study sample is more likely to fit the representative interest. The study sample should be representative for the general population and recruited without selection bias. Selection bias occurs when only subjects who are able to participate for the required time period are included, or only those who are reached (163). This study had some limitations regarding the study sample. A recruitment video was posted online and shared 105 times on Facebook to reach adults with GMFCS I-II living in or nearby Bergen. Although a lot of individuals reported interest, a maximum number of 10 individuals were permitted to participate. By having a maximum limit, potential participants who did not see the video or reported interest to late was not considered to participate. Moreover, 10 participants are a relatively small number of subjects, where dropouts may have large consequences for the result. In this study the response rate on training was 80%, where only 70% was ready for analysis. In order to generalize the results as the common characteristics of adults with SCP, it is necessary to study an increased, randomized number of subjects (159,163). Furthermore, detecting gait deviations based solely on data from high-functioning patients without no control group have limitations. As mention earlier, GMFCS system is commonly used by clinicians to manage treatment programs to group gait deviations (169). There tends to be a common assumption that especially spasticity is strongly related to gait and gross motor development. However it has been proposed that muscle weakness can explain up to 69% of the variance in ambulation of children with CP, while spasticity only have a minimal relationship with motor function (169). Yet, it appears that not all patients fit into a score according to the system (46). Even CP patients with the same score display large variety of gait deviations. When the participants in this study were asked about their GMFCS level, they were not aware of which score they had. In a future study it is therefore desirable to perform analyze of a control group to allowing comparison of differences in the treatment.

7.3 Confounding variables

Confounding variables is another important issue to consider. In a prospective case series design, there is no guarantee that all relevant covariates or confounding variables are included in the analysis (159). Reduction or decrease in spasticity was not evaluated in relation to A2/H3 ratio in this study. Authors of earlier investigations have reported reduction in spasticity after different treatment options (84,85,135,170). By implying the modified Ashworth Scale (171,172) before, during and after intervention, it would be possible to evaluate how this variable is affected by the ballistic strength regime and if there is any impact on the A2/H3 ratio.

7.4 Outcome measures

Although only kinetic data from the 3DGA are included in this study, it is important to keep in mind that the participants went through a large test protocol that possible can affected the participants performance in the gait analysis. A test day contained approximately four hours with a lot of concentration, new inputs in the laboratory and physical strain which may have caused fatigue and in that way affect gait performance.

7.4.1 Three-dimensional gait analysis

Even though 3DGA is valued as a reliable assessment tool, the approach has some disadvantages when evaluating gait (4,21). First, gait trials were preformed indoors on a 7-meter straight, flat and quite narrow walkway. Such a gait condition does not necessarily reflect everyday walking where many directional changes, starting, stopping, and obstacle avoidance strategies are required. Due to the short distance, some participants may not achieve normal and comfortable gait speed because of the need to balance. Secondly, the walkway in the laboratory only contained one force plate. This was considered as an unfortunate component as the study sample had to walk twice as many times back and forward to get three approved gait trials on each limb. Further, all the participants walked barefoot during the gait analysis. Some of them was displeased with this decision and argued that they always walk with shoes both indoors and outdoors. For that reason, the result may not even match the way they walk normally. Walking with and without shoes were not compared, which could have made a difference to the stability of the foot and ankle and possibly influenced the power generation (166,173).

7.4.2 Measurement error and considerations

In spite the fact that 3DGA is validated as gold standard to measure gait parameters, it is quite time consuming and could be considered as an obstacle for clinical use (1,2). Additionally, a ration of measure errors can arise before and during the measurement. First, measure errors can occur already in the QTM software regarding the equipment. Even if the manual calibration was approved to ensure a sufficient volume was examined during analysis, the volume may have differed each time and could have some impact to obtain accurate data. Since the 3DGA system measures motion based on the center of the marker, precise marker placement was considered critical and was always doubled-checked. However, there is always a concern that the markers could been placed incorrect due artificial skin and move relative to the underlying bone (21). It appeared challenging to place pelvis markers on obese subjects due to excessive tissue. A solution was to move ASIS markers laterally to ensure the cameras could capture the location (174,175). Even with these precautions in mind, this issue resulted in that only seven participants trials were complete and analyzed although eight subjects completed all gait trials. Moreover, the CGM-lower body marker set contains recognized simplifications due to the use of manufacturer specific anthropometric regression equations (165,176). With this in mind, some variability of the results between baseline, midterm and posttest could be expected. In this study with did not include any upper body markers. It makes it impossible to analyze any potential compensatory actions produced by the lower back and trunk, which may have affected pelvic and hip motion kinetics.

Further, when transferring the data from Qualisys to Visual 3D, the defining of gait events could possibly been mistaken relative to the marker placement. It appeared challenging to define heel-strike in subjects with toe-gait. Moreover, only ankle and hip kinetics in the sagittal plane was analyzed. The sagittal view provides only part of the information, particularly at the hip joint where hip abductors are critical for the balance control of the trunk in the coronal plane. A majority of individuals with SCP also have coronal plane and transverse plane abnormalities (131). The transvers plane is the most difficult to appreciate on visual inspection and 3DGA is required. 3DGA in combination with EMG recordings have potential to enhance the understanding of pathological movements and the spastic component during walking. It records the sum of multiple motor unit action potential and can reveal additional information about timing of muscle activity (177). However, since EMG not include any further information about power generation, this data was considered not relevant to include hence to the research question in this master project.

7.5 Ballistic strength training

It appears challenging to provide treatment considerations due to multiple combinations of gait deviations and a range of variety in movement patterns across the population group with SCP (46). The brain injury is static and there are currently no biological therapy to correct the brain lesion (17). However, in this research project the main objective was not to treat the cerebral palsy itself, but to improve functional capacity of the plantar flexors during late stance of gait (A2) and alter A2/H3 ratio (55). Previous findings from Häkkinen and komi (178) inclines that traditional resistance training is superior to improve maximum force production, whereas ballistic strength exercises are superior for muscle power and rate of force development in gait. An eight-week ballistic strength training program was therefore prescribed and considered relevant to possible enhance power generation in late stance of gait.

7.5.1 Relevance of the exercises

Although the ballistics strength training program has previously revealed positive outcomes on power output in late stance of gait in population groups with TBI and stroke, the results are preliminary (8,10,11,179). It has been questioned if ballistic strength exercises truly are functional for gait since they are performed when lying on a slide board (8). However, these three exercises were mainly prescribed to alter power output in plantar flexors during late stance of gait. With the right technique, nearly all movement should have been generated by the ankle plantar flexors. Therefore “Single leg jump on paretic leg” was expected to be the most task specific exercise to improve ankle power output for both hemiplegia and diplegia since no knee or hip flexion was allowed. However, some individuals also increased power output in the hip in addition to the ankle which resulted in a reduced A2/H3 ratio. Some even increased power output on uninvolved limb although the primary focus in the training protocol was the limb considered the most paretic identified in clinical testing. In spite the fact that hemiplegic subjects are asymmetrical by definition (5), it was expected that the plantar flexors on uninvolved side were significantly stronger than those on the collateral affected side. With one exception, all individuals with hemiplegia had a higher A2/H3 ratio at the uninvolved limb compared to the paretic limb at baseline and at posttest. However, their ratio were significant lower compared to what we expect in the general population (131). This might be explained by potential power generation from the uninvolved ankle was not fully used. Although it is often though that hemiplegic individuals have one healthy and one paretic

limb, these finding raises the question of whether there truly exist an “uninvolved” side. If not, it raises questions if it was the right decision to only train the most paretic leg on the exercise “Single leg jump on paretic leg”. In retrospect, both limbs should have been trained on this task. Moreover, because of the leg length discrepancies in the hemiplegic group, potential power in the affected limb was possibly not fully trained on “jumping squats”. This may explain why the hemiplegic group had large improvements in both ankle power output and A2/H3 ratio in the uninvolved limb as well. Referring to the group with diplegia where both limbs are affected, only one leg was identified as the most paretic and focused on during training. Bearing in mind their diagnosis, this was an interesting decision since they express weakness and reduced power output in both limbs. Since both limbs are affected, it can be assumed that “jump squats” was the most task specific. This exercise is also less load-demanding and therefore less susceptible to cause injuries.

7.5.2 Familiarization and time interval

Although some cognitive issues and difficulties in movement coordination is associated with the diagnose of SCP (76,87), we experienced that the participants used longer time to adapt to the exercise exposure than estimated before startup. A possible explanation is that their eye-foot coordination is limited. In retrospect, it would be convenient to include a period of learning to ensure right technique and understanding among the participants. In that way the participants could adapt to the training exposure individually, ensure safety and avoid injuries. Another important aspect of the strength exercise implementation was how the intensity and load progressed. To our knowledge, there are currently no standardized protocol, and optimal loads are unknown for a population with SPC. However, when the goal is to improve the rate of force development or muscle power, ACSM recommend ballistic exercises to replicate the high angular velocities attained during walking (93). In this project, load was lowered initially to achieve higher angular velocities during training. Once a participant achieved higher angular velocities in the exercises, the load increased respectively. Although the protocol of Hendrey et al. (8) was used to implement the exercises, the effect of ballistic training may vary individually among the participants influenced by different motivation, personal factor and approach of eight different instructors involved in this project. It is necessary to implement longer intervention time and a follow-up period to observe if the changes in A2/H3 ratio is permanent or reversible.

7.5.3 Side-effects

In this study, 8/9 participants completed the study protocol without any negative side-effects in relation to the intervention. However, one hemiplegic participant (FP4) dropped out of the study after 7 sessions due to a foot inflammation. It was unexpected, since she was young, GMFCS I, was exercising on regular basis and considered to be in a good shape. However, when looking at the notes from the training program, there might be a possible explanation. This participant had on average more repetitions, higher load on the exercises and reported higher numbers on Borg Scale. Looking at these numbers, a higher slope indicates risk of injury, and she was perhaps over trained. Ballistic strength training involves a component of eccentric training, which mainly recruits type II fibers (8,40). An unfamiliar amount of exposure to eccentric work can be a huge strain on the muscles and lead to stiffness. In untrained individuals exercising for the first time or individuals performing novel tasks, some limited muscle dysfunction can be seen immediately after the exercise is completed. A population group with SCP is particularly vulnerable with varied sensory, motor and psychological loss (11). To avoid injuries when performing ballistic strength training in future implementation, an important concern is to carefully adapt the load and intensity to each individual and provide necessary conditions for energy storage and release in the Achilles tendon (180).

7.6 Clinical perspectives to future research

There exist numerous of rehabilitation options developed for spastic cerebral palsy. Ballistic strength training has previously reveled feasibility in several neurological impairments (8–10,90,181), and showed to alter A2/H3 ratio in a small sample of adults with spastic cerebral palsy. In general, studying only one treatment option alone provides an understanding of possible treatment outcomes, but cannot direct any conclusion on actual effect. Further investigation should also emphasize qualitative aspects of how both patients and clinicals experience the training regime. This may provide an insight to how it affects QoL, participation in social community or potential long-term side-effects. Moreover, it is desired to investigate if this training regime is feasible to include in clinicals daily practice. It may give the opportunity to assess optimal loads, frequency and repetitions of ballistic strength training and whether there exist pathological differences between hemiplegia and diplegia.

7.7 Summary and conclusion

Based on the findings of current study, the results provide additional information and a better understanding of how ballistic strength training might contribute to alter A2/H3 ratio in high-functioning individuals with spastic cerebral palsy. This case series study provides potential intervention outcomes but cannot determine if the treatment was effective since there was no control group and the intervention time was relatively short. Further analytic studies comparing ballistic strength training to traditional strength training is needed to improve knowledge of the clinical value of ballistic strength training in adults with SCP.

Chapter 8: References

1. Graham HK, Rosenbaum P, Paneth N, Dan B, Lin J-P, Damiano DL, et al. Cerebral palsy. Nature Reviews Disease Primers. 2016 Jan 28;2:16005.
2. Olney SJ, MacPhail HE, Hedden DM, Boyce WF. Work and power in hemiplegic cerebral palsy gait. Phys Ther. 1990 Jul;70(7):431–8.
3. Winter DA. Biomechanical Motor Patterns in Normal Walking. Journal of Motor Behavior. 1983 Dec 1;15(4):302–30.
4. Eng JJ, Winter DA. Kinetic analysis of the lower limbs during walking: what information can be gained from a three-dimensional model? J Biomech. 1995 Jun;28(6):753–8.
5. Ishihara M, Higuchi Y, Yonetsu R, Kitajima H. Plantarflexor training affects propulsive force generation during gait in children with spastic hemiplegic cerebral palsy: a pilot study. J Phys Ther Sci. 2015 May;27(5):1283–6.
6. Ishihara M, Higuchi Y. Kinetic Relationships between the Hip and Ankle Joints during Gait in Children with Cerebral Palsy: A Pilot Study. J Phys Ther Sci. 2014 May;26(5):737–40.
7. Eek MN, Tranberg R, Beckung E. Muscle strength and kinetic gait pattern in children with bilateral spastic CP. Gait & Posture. 2011;33(3):333–7.
8. Hendrey G, Clark RA, Holland AE, Mentiplay BF, Davis C, Windfeld-Lund C, et al. Feasibility of Ballistic Strength Training in Subacute Stroke: A Randomized, Controlled, Assessor-Blinded Pilot Study. Arch Phys Med Rehabil. 2018 Dec;99(12):2430–46.
9. Williams G, Hassett L, Clark R, Bryant A, Olver J, Morris ME, et al. Improving Walking Ability in People With Neurologic Conditions: A Theoretical Framework for Biomechanics-Driven Exercise Prescription. Archives of Physical Medicine and Rehabilitation. 2019;100(6):1184–90.
10. Williams GP, Schache AG, Morris ME. Mobility after traumatic brain injury: relationships with ankle joint power generation and motor skill level. J Head Trauma Rehabil. 2013 Oct;28(5):371–8.
11. Williams G, Clark RA, Hansson J, Paterson K. Feasibility of ballistic strengthening exercises in neurologic rehabilitation. Am J Phys Med Rehabil. 2014 Sep;93(9):828–33.
12. Jessica Rose. Human walking. 2nd ed. Baltimore: Williams & Wilkins; 1994.
13. Kirtley C, Whittle MW, Jefferson RJ. Influence of walking speed on gait parameters. Journal of Biomedical Engineering. 1985 Oct 1;7(4):282–8.
14. Dicharry J. Kinematics and kinetics of gait: from lab to clinic. Clin Sports Med. 2010 Jul;29(3):347–64.
15. Perry, J. Scientific basis of rehabilitation. Instr Course Lect; 1992.
16. J Perry. Gait analysis normal and pathological function. Thorofare, NJ: Slack Incorpoated; 1992.
17. Armand S, Decoulon G, Bonnefoy-Mazure A. Gait analysis in children with cerebral palsy. EFORT Open Rev. 2016 Dec;1(12):448–60.
18. Cimolin V, Condoluci C, Costici PF, Galli M. A proposal for a kinetic summary measure: the Gait Kinetic Index. Comput Methods Biomech Biomed Engin. 2019 Jan;22(1):94–9.
19. Vuillermin C, Rodda J, Rutz E, Shore BJ, Smith K, Graham HK. Severe crouch gait in spastic diplegia can be prevented: a population-based study. The Journal of bone and joint surgery British volume. 2011;93(12):1670.
20. Lin Y-C, Gfoehler M, Pandy MG. Quantitative evaluation of the major determinants of human gait. J Biomech. 2014 Apr 11;47(6):1324–31.
21. Allard P. Three dimensional analyses of human movement. III. Champaign; 1995.

22. Ounpuu S. The biomechanics of walking and running. Clinics in sports medicine. 1994 Nov 1;13:843–63.
23. Sadeghi H. Contributions of lower-limb muscle power in gait of people without impairments. Physical therapy. 2000;80(12):1188.
24. Protokinetics Team. Understanding Phases of the Gait Cycle [Internet]. General Gait Analysis |. 2018. Available from: https://www.protokinetics.com/2018/11/28/understanding-phases-of-the-gait-cycle/
25. Whittle M. Whittle´s gait analysis. 5th ed. Philadelphia: Churchill Livingstone; 2012.
26. Cavagna GA, Kaneko M. Mechanical work and efficiency in level walking and running. J Physiol. 1977 Jun;268(2):467--81.
27. Olney SJ, Griffin MP, McBride ID. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. Phys Ther. 1994 Sep;74(9):872–85.
28. Gage JR, DeLuca PA, Renshaw TS. Gait analysis: principle and applications with emphasis on its use in cerebral palsy. Instr Course Lect. 1996;45:491–507.
29. Lewis CL, Laudicina NM, Khuu A, Loverro KL. The Human Pelvis: Variation in Structure and Function During Gait. Anat Rec (Hoboken). 2017 Apr;300(4):633–42.
30. JudgeRoy JO, Davis B III, Õunpuu S. Step Length Reductions in Advanced Age: The Role of Ankle and Hip Kinetics. The Journals of Gerontology: Series A. 1996 Nov 1;51A(6):M303–12.
31. Bejek Z, Paroczai R, Illyes A, Kiss RM. The influence of walking speed on gait parameters in healthy people and in patients with osteoarthritis. Knee Surg Sports Traumatol Arthrosc. 2006 Jul;14(7):612–22.
32. Krebs DE, Robbins CE, Lavine L, Mann RW. Hip biomechanics during gait. J Orthop Sports Phys Ther. 1998 Jul;28(1):51–9.
33. Dahl HA, Eric Rinvik. Menneskets funksjonelle anatomi: Med hovedvekt på bevegelsesapparatet. 1st ed. Cappelen akademisk; 2010.
34. Alegre LM, Jiménez F, Gonzalo-Orden JM, Martín-Acero R, Aguado X. Effects of dynamic resistance training on fascicle lengthand isometric strength. Journal of Sports Sciences. 2006 May 1;24(5):501–8.
35. Aagaard P, Andersen JL, Dyhre-Poulsen P, Leffers A, Wagner A, Magnusson SP, et al. A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. Journal of Physiology. 2001;534(2):613–23.
36. Renstrøm, R. Kraft og Bevegelse: Innføring i Mekanikk. Høyskoleforl.; 1997.
37. Lieber RL, Steinman S, Barash IA, Chambers H. Structural and functional changes in spastic skeletal muscle. Muscle & Nerve. 2004 May 1;29(5):615–27.
38. Lieber RL, Friden J. Functional and clinical significance of skeletal muscle architecture. Muscle Nerve. 2000 Nov;23(11):1647–66.
39. Aagaard P. Training-induced changes in neural function. Exerc Sport Sci Rev. 2003 Apr;31(2):61–7.
40. Stauber WT. Eccentric action of muscles: physiology, injury, and adaptation. Exerc Sport Sci Rev. 1989;17:157–85.
41. Seliktar R, Bo, L. The theory of kinetic analysis in human gait (Gait Analysis: Theory and Applications). St. Louis: Mosby; 1995.
42. Winter D. The Biomechanics and Motorcontrol of Human Gait: Normal, Elderly and Pathological. 2nd ed. Ontario, University og Waterloo; 1991.
43. Bogey RA, Barnes LA. Estimates of individual muscle power production in normal adult walking.(Report). Journal of NeuroEngineering and Rehabilitation. 2017;14(1).
44. Vladimir M. Zarsiorsky. Kinetics og Human motion. 2002.
45. Damiano DL, Martellotta TL, Quinlivan JM, Abel MF. Deficits in eccentric versus

concentric torque in children with spastic cerebral palsy. Med Sci Sports Exerc. 2001 Jan;33(1):117–22.
46. Riad J, Haglund-Akerlind Y, Miller F. Power generation in children with spastic hemiplegic cerebral palsy. Gait Posture. 2008 May;27(4):641–7.
47. Brockett CL, Chapman GJ. Biomechanics of the ankle. Orthop Trauma. 2016 Jun;30(3):232–8.
48. Hof AL, Zandwijk JPV, Bobbert MF. Mechanics of human triceps surae muscle in walking, running and jumping. Acta Physiologica Scandinavica. 2002;174(1):17–30.
49. Stewart C, Postans N, Schwartz MH, Rozumalski A, Roberts A. An exploration of the function of the triceps surae during normal gait using functional electrical stimulation. Gait & Posture. 2007 Oct 1;26(4):482–8.
50. Hunt MA, Hatfield GL. Ankle and knee biomechanics during normal walking following ankle plantarflexor fatigue. Journal of Electromyography and Kinesiology. 2017 Aug 1;35:24–9.
51. Sawicki S Gregory, Lewis L Cara, Ferris P Daniel. It Pays to Have a Spring in Your Step. Exercise and Sport Sciences Reviews. 2009;37(3):130–8.
52. Zelik KE, Huang T-WP, Adamczyk PG, Kuo AD. The role of series ankle elasticity in bipedal walking. Journal of Theoretical Biology. 2014 Apr 7;346:75–85.
53. Lythgo N. RELATIONSHIP BETWEEN ANKLE PLANTAR FLEXOR POWER AND EMG MUSCLE ACTIVITY DURING GAIT. 2012.
54. Fickey SN, Browne MG, Franz JR. Biomechanical effects of augmented ankle power output during human walking. J Exp Biol. 2018 Nov 16;221(Pt 22).
55. Lenhart RL, Francis CA, Lenz AL, Thelen DG. Empirical evaluation of gastrocnemius and soleus function during walking. Journal of Biomechanics. 2014;47(12):2969–74.
56. Francis CA, Lenz AL, Lenhart RL, Thelen DG. The modulation of forward propulsion, vertical support, and center of pressure by the plantarflexors during human walking. Gait & Posture. 2013 Sep 1;38(4):993–7.
57. DeVita P, Helseth J, Hortobagyi T. Muscles do more positive than negative work in human locomotion. J Exp Biol. 2007 Oct 1;210(19):3361.
58. Zelik KE, Adamczyk PG. A unified perspective on ankle push-off in human walking. J Exp Biol. 2016 Dec 1;219(Pt 23):3676–83.
59. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. Clinical Biomechanics. 1999 Feb 1;14(2):125–35.
60. Farris DJ, Sawicki GS. The mechanics and energetics of human walking and running: a joint level perspective. Journal of the Royal Society Interface. 2012;9(66):110–8.
61. Neptune RR, Kautz SA, Zajac FE. Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. Journal of Biomechanics. 2001 Nov 1;34(11):1387–98.
62. Neptune RR, Clark DJ, Kautz SA. Modular control of human walking: A simulation study. Journal of Biomechanics. 2009 Jun 19;42(9):1282–7.
63. Farris DJ, Hampton A, Lewek MD, Sawicki GS. Revisiting the mechanics and energetics of walking in individuals with chronic hemiparesis following stroke: from individual limbs to lower limb joints. Journal of NeuroEngineering and Rehabilitation. 2015 Feb 27;12(1):24.
64. Lunn DE, Lampropoulos A, Stewart TD. Basic biomechanics of the hip. Orthopaedics and Trauma. 2016 Jun 1;30(3):239–46.
65. DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. J Appl Physiol (1985). 2000 May;88(5):1804–11.
66. Mcgibbon C, Krebs D. Discriminating age and disability effects in locomotion:

Neuromuscular adaptations in musculoskeletal pathology. Journal of Applied Physiology. 2004;96(1):149–60.
67. Neptune RR, Sasaki K, Kautz SA. The effect of walking speed on muscle function and mechanical energetics. Gait Posture. 2008 Jul;28(1):135–43.
68. Requião LF, Nadeau S, Milot MH, Gravel D, Bourbonnais D, Gagnon D. Quantification of level of effort at the plantarflexors and hip extensors and flexor muscles in healthy subjects walking at different cadences. Journal of Electromyography and Kinesiology. 2005 Aug 1;15(4):393–405.
69. Goldberg SR, Anderson FC, Pandy MG, Delp SL. Muscles that influence knee flexion velocity in double support: implications for stiff-knee gait. Journal of Biomechanics. 2004;37(8):1189–96.
70. Allard P, Lachance R, Aissaoui R, Duhaime M. Simultaneous bilateral 3-D able-bodied gait. Human Movement Science. 1996;15(3):327–46.
71. Neptune RR, Zajac FE, Kautz SA. Muscle force redistributes segmental power for body progression during walking. Gait & Posture. 2004 Apr 1;19(2):194–205.
72. Lewis CL, Ferris DP. Walking with increased ankle pushoff decreases hip muscle moments. Journal of Biomechanics. 2008;41(10):2082–9.
73. Tateuchi H, Tsukagoshi R, Fukumoto Y, Oda S, Ichihashi N. Immediate effects of different ankle pushoff instructions during walking exercise on hip kinematics and kinetics in individuals with total hip arthroplasty. Gait & Posture. 2011 Apr 1;33(4):609–14.
74. Opheim A, Jahnsen R, Olsson E, Stanghelle JK. Walking function, pain, and fatigue in adults with cerebral palsy: a 7-year follow-up study. Dev Med Child Neurol. 2009 May;51(5):381–8.
75. Williams G, Kahn M, Randall A. Strength training for walking in neurologic rehabilitation is not task specific: a focused review. Am J Phys Med Rehabil. 2014 Jun;93(6):511–22.
76. Moreau NG, Bodkin AW, Bjornson K, Hobbs A, Soileau M, Lahasky K. Effectiveness of Rehabilitation Interventions to Improve Gait Speed in Children With Cerebral Palsy: Systematic Review and Meta-analysis. Phys Ther. 2016 Dec;96(12):1938–54.
77. Scianni A, Butler JM, Ada L, Teixeira-Salmela LF. Muscle strengthening is not effective in children and adolescents with cerebral palsy: a systematic review. Australian Journal of Physiotherapy. 2009 Jan 1;55(2):81–7.
78. Scholtes VA, Becher JG, Comuth A, Dekkers H, Van Dijk L, Dallmeijer AJ. Effectiveness of functional progressive resistance exercise strength training on muscle strength and mobility in children with cerebral palsy: a randomized controlled trial. Dev Med Child Neurol. 2010 Jun;52(6):e107-113.
79. Winter DA. Energy generation and absorption at the ankle and knee during fast, natural, and slow cadences. Clin Orthop Relat Res. 1983 May;(175):147–54.
80. Moreau NG, Holthaus K, Marlow N. Differential adaptations of muscle architecture to high-velocity versus traditional strength training in cerebral palsy. Neurorehabil Neural Repair. 2013 May;27(4):325–34.
81. Romkes J, Brunner R. Comparison of a dynamic and a hinged ankle-foot orthosis by gait analysis in patients with hemiplegic cerebral palsy. Gait Posture. 2002 Feb;15(1):18–24.
82. Crenshaw S, Herzog R, Castagno P, Richards J, Miller F, Michaloski G, et al. The efficacy of tone-reducing features in orthotics on the gait of children with spastic diplegic cerebral palsy. J Pediatr Orthop. 2000 Apr;20(2):210–6.
83. Naumann M, Boo LM, Ackerman AH, Gallagher CJ. Immunogenicity of botulinum toxins. J Neural Transm (Vienna). 2013 Feb;120(2):275–90.
84. Zurcher AW, Molenaers G, Desloovere K, Fabry G. Kinematic and kinetic evaluation of the ankle after intramuscular injection of botulinum toxin A in children with cerebral palsy.

Acta Orthop Belg. 2001 Dec;67(5):475–80.
85. Lee JH, Sung IY, Yoo JY, Park EH, Park SR. Effects of different dilutions of botulinum toxin type A treatment for children with cerebral palsy with spastic ankle plantarflexor: A randomized controlled trial. Effects of different dilutions of botulinum toxin type A treatment for children with cerebral palsy with spastic ankle plantarflexor: A randomized controlled trial. 2009;41(9).
86. Gannotti ME, Gorton GE, Nahorniak MT, Masso PD. Walking abilities of young adults with cerebral palsy: Changes after multilevel surgery and adolescence. Gait & Posture. 2010 May 1;32(1):46–52.
87. Martins E, Cordovil R, Oliveira R, Letras S, Lourenço S, Pereira I, et al. Efficacy of suit therapy on functioning in children and adolescents with cerebral palsy: a systematic review and meta-analysis. Developmental Medicine & Child Neurology. 2016 Apr 1;58(4):348–60.
88. Lopopolo RB, Greco M, Sullivan D, Craik RL, Mangione KK. Effect of therapeutic exercise on gait speed in community-dwelling elderly people: a meta-analysis. Phys Ther. 2006 Apr;86(4):520–40.
89. Dorsch S, Ada L, Alloggia D. Progressive resistance training increases strength after stroke but this may not carry over to activity: a systematic review. Journal of Physiotherapy. 2018 Apr 1;64(2):84–90.
90. Newton RU, Kraemer WJ. Developing Explosive Muscular Power: Implications for a Mixed Methods Training Strategy. Strength and Conditioning. 1994;16(5):20.
91. Wiley ME, Damiano DL. Lower-Extremity strength profiles in spastic cerebral palsy. Developmental Medicine & Child Neurology. 1998 Feb 1;40(2):100–7.
92. Fukunaga T, Kubo K, Kawakami Y, Fukashiro S, Kanehisa H, Maganaris CN. In vivo behaviour of human muscle tendon during walking. Proc Biol Sci. 2001 Feb 7;268(1464):229–33.
93. Ratamess NA, Alvar BA, Evetoch TK, Housh TJ, Kibler WB, Kraemer WJ, et al. Progression models in resistance training for healthy adults.(Special Communications)(Report). Medicine and Science in Sports and Exercise. 2009;41(3):687–708.
94. Damiano D, Quinlivan J, Owen B, Shaffrey M, Abel M. Spasticity versus strength in cerebral palsy: relationships among involuntary resistance, voluntary torque, and motor function. Eur J Neurol. 2001;8:40–9.
95. Rose J, Mcgill KC. Neuromuscular activation and motor-unit firing characteristics in cerebral palsy. Developmental Medicine & Child Neurology. 2005;47(5):329–36.
96. Rose J, Haskell WL, Gamble JG, Hamilton RL, Brown DA, Rinsky L. Muscle pathology and clinical measures of disability in children with cerebral palsy. J Orthop Res. 1994 Nov;12(6):758–68.
97. Nogueira W, Gentil P, Mello SNM, Oliveira RJ, Bezerra AJC, Bottaro M. Effects of power training on muscle thickness of older men. Int J Sports Med. 2009 Mar;30(3):200–4.
98. Farthing JP, Chilibeck PD. The effects of eccentric and concentric training at different velocities on muscle hypertrophy. European Journal of Applied Physiology. 2003 Aug 1;89(6):578–86.
99. van Vulpen LF, de Groot S, Rameckers EAA, Becher JG, Dallmeijer AJ. Effectiveness of Functional Power Training on Walking Ability in Young Children With Cerebral Palsy: Study Protocol of a Double-Baseline Trial. Pediatr Phys Ther. 2017 Jul;29(3):275–82.
100. van Vulpen LF, de Groot S, Rameckers E, Becher JG, Dallmeijer AJ. Improved Walking Capacity and Muscle Strength After Functional Power-Training in Young Children With Cerebral Palsy. Neurorehabil Neural Repair. 2017 Sep;31(9):827–41.

101. Jonkers I, Delp S, Patten C. Capacity to increase walking speed is limited by impaired hip and ankle power generation in lower functioning persons post-stroke. Gait & Posture. 2009;29(1):129–37.
102. Esquenazi A. Evaluation and Management of Spastic Gait in Patients With Traumatic Brain Injury. Journal of Head Trauma Rehabilitation. 2004;19(2):109–18.
103. Perry, J. The use of gait analysis for surgical recommendations in traumatic brain injury. J Head Trauma Rehabil. 1999 Apr;14(2):116–35.
104. Williams G, Schache AG. The distribution of positive work and power generation amongst the lower-limb joints during walking normalises following recovery from traumatic brain injury. Gait Posture. 2016 Jan;43:265–9.
105. Ballaz L, Plamondon S, Lemay M. Ankle range of motion is key to gait efficiency in adolescents with cerebral palsy. Clin Biomech. 2010;25(9):944–8.
106. Andersen GL, Irgens LM, Haagaas I, Skranes JS, Meberg AE, Vik T. Cerebral palsy in Norway: Prevalence, subtypes and severity. European Journal of Paediatric Neurology. 2008 Jan 1;12(1):4–13.
107. Bell J Katharine, Õunpuu A Sylvia, Deluca J Peter, Romness J Mark. Natural Progression of Gait in Children With Cerebral Palsy. Journal of Pediatric Orthopaedics. 2002;22(5):677–82.
108. Gjesdal BE, Jahnsen R, Morgan P, Opheim A, Mæland S. Walking through life with cerebral palsy: reflections on daily walking by adults with cerebral palsy. International Journal of Qualitative Studies on Health and Well-being. 2020 Jan 1;15(1):1746577.
109. Morgan P, McGinley J. Gait function and decline in adults with cerebral palsy: a systematic review. Disability and Rehabilitation. 2014 Jan 1;36(1):1–9.
110. Tonmukayakul U, Shih STF, Bourke-Taylor H, Imms C, Reddihough D, Cox L, et al. Systematic review of the economic impact of cerebral palsy. Research in Developmental Disabilities. 2018;80:93–101.
111. Jack R. Engsberg, Sandy A. Ross, Tae Sung Park. Changes in ankle spasticity and strength following selective dorsal rhizotomy and physical therapy for spastic cerebral palsy. Journal of Neurosurgery. 1999;91(5):727–32.
112. Jack R. Engsberg, Kenneth S. Olree, Sandy A. Ross, T. S. Park. Spasticity and strength changes as a function of selective dorsal rhizotomy. Journal of Neurosurgery. 1998;88(6):1020–6.
113. Knutsson E, Mårtensson A, Gransberg L. Influences of muscle stretch reflexes on voluntary, velocity-controlled movements in spastic paraparesis. Brain. 1997 Sep 1;120(9):1621–33.
114. Kent-Braun JA, Ng AV, Young K. Skeletal muscle contractile and noncontractile components in young and older women and men. J Appl Physiol (1985). 2000 Feb;88(2):662–8.
115. Narici MV, Maffulli N. Sarcopenia: characteristics, mechanisms and functional significance. Br Med Bull. 2010;95:139–59.
116. Jahnsen R, Villien L, Egeland T, Stanghelle JK. Locomotion skills in adults with cerebral palsy. Clin Rehabil. 2004 May 1;18(3):309–16.
117. Jahnsen R, Villien L, Stanghelle JK, Holm I. Fatigue in adults with cerebral palsy in Norway compared with the general population. Developmental Medicine & Child Neurology. 2003 May 1;45(5):296–303.
118. Bottos M, Feliciangeli A, Sciuto L, Gericke C, Vianello A. Functional status of adults with cerebral palsy and implications for treatment of children. Dev Med Child Neurol. 2001 Aug;43(8):516–28.
119. Livingston MH, Rosenbaum PL, Russell DJ, Palisano RJ. Quality of life among adolescents with cerebral palsy: what does the literature tell us? Developmental Medicine &

Child Neurology. 2007 Mar 1;49(3):225–31.
120. Stewart DA, Lawless JJ, Shimmell LJ, Palisano RJ, Freeman M, Rosenbaum PL, et al. Social Participation of Adolescents with Cerebral Palsy: Trade-offs and Choices. Physical & Occupational Therapy In Pediatrics. 2012 Mar 22;32(2):167–79.
121. Karlsson J 1953-, Bahr R 1957-. Aktivitetshåndboken: fysisk aktivitet i forebygging og behandling. [Oslo]: Helsedirektoratet; 2008. 624 s. ill.
122. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. Developmental Medicine & Child Neurology. 1997 Apr 1;39(4):214–23.
123. Moissenet F, Armand S. Qualitative and quantitative methods of assessing gait disorders. In 2015. p. 215–40.
124. Jahnsen R, Aamodt G, Rosenbaum P. Gross Motor Function Classification System used in adults with cerebral palsy: agreement of self-reported versus professional rating. Dev Med Child Neurol. 2006 Sep;48(9):734–8.
125. Himuro N, Mishima R, Seshimo T, Morishima T, Kosaki K, Ibe S, et al. Change in mobility function and its causes in adults with cerebral palsy by Gross Motor Function Classification System level: A cross-sectional questionnaire study. NeuroRehabilitation. 2018;42(4):383–90.
126. Noorkoiv M, Lavelle G, Theis N, Korff T, Kilbride C, Baltzopoulos V, et al. Predictors of Walking Efficiency in Children With Cerebral Palsy: Lower-Body Joint Angles, Moments, and Power. Phys Ther. 2019 Jun 1;99(6):711–20.
127. Mueller MJ, Sinacore DR, Hoogstrate S, Daly L. Hip and ankle walking strategies: effect on peak plantar pressures and implications for neuropathic ulceration. Archives of physical medicine and rehabilitation. 1994;75(11):1196.
128. Whitney DG, Hurvitz EA, Devlin MJ, Caird MS, French ZP, Ellenberg EC, et al. Age trajectories of musculoskeletal morbidities in adults with cerebral palsy. Bone. 2018 Sep 1;114:285–91.
129. Zelik KE, Honert EC. Ankle and foot power in gait analysis: Implications for science, technology and clinical assessment. Journal of Biomechanics. 2018;75:1–12.
130. Winters TFJ, Gage JR, Hicks R. Gait patterns in spastic hemiplegia in children and young adults. J Bone Joint Surg Am. 1987 Mar;69(3):437–41.
131. Rodda J, Graham HK. Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm. European Journal of Neurology. 2001;8(s5):98–108.
132. Brandstater ME, de Bruin H, Gowland C, Clark BM. Hemiplegic gait: analysis of temporal variables. Archives of physical medicine and rehabilitation. 1983;64(12):583.
133. Peat M, Dubo HI, Winter DA, Quanbury AO, Steinke T, Grahame R. Electromyographic temporal analysis of gait: hemiplegic locomotion. Arch Phys Med Rehabil. 1976 Sep;57(9):421–5.
134. Wall JC, Turnbull GI. Gait asymmetries in residual hemiplegia. Archives of physical medicine and rehabilitation. 1986;67(8):550.
135. Braendvik SM, Roeleveld K, Andersen GL, Raftemo AER, Ramstad K, Majkic-Tajsic J, et al. The WE-Study: does botulinum toxin A make walking easier in children with cerebral palsy?: Study protocol for a randomized controlled trial. Trials. 2017 Feb 6;18(1):58.
136. Dobson F, Morris M, Baker R, Wolfe R, Graham H. Clinician agreement on gait pattern ratings in children with spastic hemiplegia. Dev Med Child Neurol. 2006;48(6):429–35.
137. Riad J, Modlesky CM, Gutierrez-Farewik EM, Brostrom E. Are muscle volume differences related to concentric muscle work during walking in spastic hemiplegic cerebral palsy? Clin Orthop Relat Res. 2012 May;470(5):1278–85.

138. Galli M, Cimolin V, Rigoldi C, Tenore N, Albertini G. Gait patterns in hemiplegic children with Cerebral Palsy: comparison of right and left hemiplegia. Res Dev Disabil. 2010 Dec;31(6):1340–5.
139. Opheim A, McGinley JL, Olsson E, Stanghelle JK, Jahnsen R. Walking deterioration and gait analysis in adults with spastic bilateral cerebral palsy. Gait & Posture. 2013 Feb 1;37(2):165–71.
140. Williams G, Morris ME, Schache A, McCrory PR. People Preferentially Increase Hip Joint Power Generation to Walk Faster Following Traumatic Brain Injury. Neurorehabil Neural Repair. 2010 Jan 19;24(6):550–8.
141. Winter DA, Patla AE, Frank JS, Walt SE. Biomechanical Walking Pattern Changes in the Fit and Healthy Elderly. Physical Therapy. 1990 Jun 1;70(6):340–7.
142. Wren TAL, Cheatwood AP, Rethlefsen SA, Hara R, Perez FJ, Kay RM. Achilles tendon length and medial gastrocnemius architecture in children with cerebral palsy and equinus gait. J Pediatr Orthop. 2010 Aug;30(5):479–84.
143. Gao F, Zhao H, Gaebler-Spira D, Zhang L-Q. In vivo evaluations of morphologic changes of gastrocnemius muscle fascicles and achilles tendon in children with cerebral palsy. Am J Phys Med Rehabil. 2011 May;90(5):364–71.
144. Martin Lorenzo T, Rocon E, Martinez Caballero I, Lerma Lara S. Medial gastrocnemius structure and gait kinetics in spastic cerebral palsy and typically developing children: A cross-sectional study. Medicine (Baltimore). 2018 May;97(21):e10776.
145. Coggan AR, Spina RJ, King DS, Rogers MA, Brown M, Nemeth PM, et al. Histochemical and enzymatic comparison of the gastrocnemius muscle of young and elderly men and women. J Gerontol. 1992 May;47(3):B71-76.
146. Gajdosik RL, Vander Linden DW, McNair PJ, Riggin TJ, Albertson JS, Mattick DJ, et al. Viscoelastic properties of short calf muscle-tendon units of older women: effects of slow and fast passive dorsiflexion stretches in vivo. Eur J Appl Physiol. 2005 Oct;95(2–3):131–9.
147. Bottinelli R, Pellegrino MA, Canepari M, Rossi R, Reggiani C. Specific contributions of various muscle fibre types to human muscle performance: an in vitro study. Journal of Electromyography and Kinesiology. 1999;9(2):87–95.
148. Hortobagyi T, Devita P. Mechanisms responsible for the age-associated increase in coactivation of antagonist muscles. Exerc Sport Sci Rev. 2006 Jan;34(1):29–35.
149. Andersson C, Mattsson E. Adults with cerebral palsy: a survey describing problems, needs, and resources, with special emphasis on locomotion. Dev Med Child Neurol. 2001 Feb;43(2):76–82.
150. Williams G, Schache AG. The distribution of positive work and power generation amongst the lower-limb joints during walking normalises following recovery from traumatic brain injury. Gait Posture. 2016 Jan;43:265–9.
151. Silder A, Heiderscheit B, Thelen DG. Active and passive contributions to joint kinetics during walking in older adults. J Biomech. 2008;41(7):1520–7.
152. Pouliot-Laforte A, Parent A, Ballaz L. Walking efficiency in children with cerebral palsy: relation to muscular strength and gait parameters. Computer Methods in Biomechanics and Biomedical Engineering. 2014;17(1):104–5.
153. Browne MG, Franz JR. More push from your push-off: Joint-level modifications to modulate propulsive forces in old age. PLoS One. 2018;13(8):e0201407.
154. Milot M-H, Nadeau S, Gravel D, Bourbonnais D. Effect of increases in plantarflexor and hip flexor muscle strength on the levels of effort during gait in individuals with hemiparesis. Clin Biomech (Bristol, Avon). 2008 May;23(4):415–23.
155. Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: Characteristics. Gait & Posture. 1996;4(2):136–48.
156. Fry NR, Gough M, Shortland AP. Three-dimensional realisation of muscle

morphology and architecture using ultrasound. Gait & Posture. 2004;20(2):177–82.
157. Olney SJ, Griffin MP, Monga TN, Mcbride ID. Work and power in gait of stroke patients. Archives of physical medicine and rehabilitation. 1991;72(5):309.
158. Grimes DA, Schulz KF. Descriptive studies: what they can and cannot do. The Lancet. 2002;359(9301):145–9.
159. Brighton B, Bhandari M, Tornetta P 3rd, Felson DT. Hierarchy of evidence: from case reports to randomized controlled trials. Clin Orthop Relat Res. 2003 Aug;(413):19–24.
160. Audigé L, Hanson B, Kopjar B. Issues in the planning and conduct of non-randomised studies. Injury. 2006 Apr 1;37(4):340–8.
161. Polit DF, Beck CT. Nursing Research: Generating and assessing Ecidence for Nursing Practice. Tenth. Lippincott Williams and Wilkins, Wolters Kluwer; 2016.
162. Thornquist E. Vitenskapsfilosofi og vitenskapsteori : for helsefag. 2. utg. Bergen: Fagbokforl.; 2018.
163. Dallan O. Metode og oppgaveskriving: Oslo: Gyldendal Norsk Forlag AS; 2012.
164. Fosang AL, Galea MP, Mccoy AT, Reddihough DS, Story I. Measures of muscle and joint performance in the lower limb of children with cerebral palsy. Developmental Medicine & Child Neurology. 2003;45(10):664–70.
165. Baker R, Leboeuf F, Reay J, Sangeux M. The Conventional Gait Model : The Success and Limitations. In 2017. p. 1–19.
166. McGinley JL, Baker R, Wolfe R, Morris ME. The reliability of three-dimensional kinematic gait measurements: A systematic review. Gait & Posture. 2009 Apr 1;29(3):360–9.
167. Julie Pallant. SPSS Survival Manual. Open University Press; 2016.
168. Kooistra B, Dijkman B, Einhorn TA, Bhandari M. How to design a good case series. J Bone Joint Surg Am. 2009 May;91 Suppl 3:21–6.
169. Ross SA, Engsberg JR. Relationships between spasticity, strength, gait, and the GMFM-66 in persons with spastic diplegia cerebral palsy. Arch Phys Med Rehabil. 2007 Sep;88(9):1114–20.
170. Balaban B, Tok F, Tan AK, Matthews DJ. Botulinum toxin a treatment in children with cerebral palsy: its effects on walking and energy expenditure. Am J Phys Med Rehabil. 2012 Jan;91(1):53–64.
171. Mutlu A, Livanelioglu A, Gunel MK. Reliability of Ashworth and Modified Ashworth Scales in Children with Spastic Cerebral Palsy. BMC Musculoskeletal Disorders. 2008 Apr 10;9(1):44.
172. Numanoglu A, Gunel MK. Intraobserver reliability of modified Ashworth scale and modified Tardieu scale in the assessment of spasticity in children with cerebral palsy. Acta Orthop Traumatol Turc. 2012;46(3):196–200.
173. Wedege P, Steffen K, Strøm V, Opheim AI. Reliability of three-dimensional kinematic gait data in adults with spinal cord injury. J Rehabil Assist Technol Eng. 2017 Sep 14;4:2055668317729992–2055668317729992.
174. Borhani M, McGregor AH, Bull AMJ. An alternative technical marker set for the pelvis is more repeatable than the standard pelvic marker set. Gait Posture. 2013/06/19. 2013 Sep;38(4):1032–7.
175. Lerner Z, Board W, Browning R. Effects of an Obesity-Specific Marker Set on Estimated Muscle and Joint Forces in Walking. Medicine and science in sports and exercise. 2014 Feb 10;46.
176. Schwartz MH, Rozumalski A, Trost JP. The effect of walking speed on the gait of typically developing children. J Biomech. 2008;41(8):1639–50.
177. DeLuca PA. The use of gait analysis and dynamic EMG in the assessment of the child with cerebral palsy. Human Movement Science. 1991 Oct 1;10(5):543–54.
178. Hakkinen K, Komi PV, Alen M. Effect of explosive type strength training on

isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. Acta Physiol Scand. 1985 Dec;125(4):587–600.
179. Mentiplay BF, Williams G, Tan D, Adair B, Pua Y-H, Bok CW, et al. Gait velocity and joint power generation after stroke: contribution of strength and balance. Am J Phys Med Rehabil. 2018 Dec 28;
180. Sugisaki N, Kanehisa H, Kawakami Y, Fukunaga T. Behavior of Fascicle and Tendinous Tissue of Medial Gastrocnemius Muscle during Rebound Exercise of Ankle Joint. International Journal of Sport and Health Science. 2005 Jan 1;3:100–9.
181. Schache AG, Dorn TW, Williams GP, Brown NAT, Pandy MG. Lower-Limb Muscular Strategies for Increasing Running Speed. J Orthop Sports Phys Ther. 2014 Aug 7;44(10):813–24.

Chapter 9: Appendices

Appendix 1: Approval – REK

REK
REGIONALE KOMITEER FOR MEDISINSK OG HELSEFAGLIG FORSKNINGSETIKK

Region:	Saksbehandler:	Telefon:	Vår dato:	Vår referanse:
REK vest	Jessica Svärd	55978497	04.02.2019	2018/2390/REK vest
			Deres dato:	Deres referanse:
			11.12.2018	

Vår referanse må oppgis ved alle henvendelser

Silje Mæland
Fakultet for helse- og sosialvitskap

2018/2390 Trening av funksjonell kapasitet for gange hos voksne med cerebral parese

Forskningsansvarlig: Høgskulen på Vestlandet
Prosjektleder: Silje Mæland

Vi viser til søknad om forhåndsgodkjenning av ovennevnte forskningsprosjekt. Søknaden ble behandlet av Regional komité for medisinsk og helsefaglig forskningsetikk (REK vest) i møtet 16.01.2019. Vurderingen er gjort med hjemmel i helseforskningsloven (hforsknl) § 10.

Prosjektomtale
De fleste personer med cerebral parese (CP) har i oppveksten hatt mye fysioterapi med fokus på gangtrening. Ingen studier har til nå vist å kunne anbefale en treningsintervensjon over en annen for å påvirke gangfunksjonen. En kritisk review av Williams et al. (2014) poengterer at trening for å påvirke gange hos neurologiske pasienter ikke er oppgavespesifikk. Flere studier har vist at eksentrisk styrketrening påvirker muskelens kapasitet for hurtig kraftutvikling, ballistisk styrketrening er oppgavespesifikk for hurtig kraftutvikling. I denne studien vil halvparten av deltakerne trene eksentrisk styrketrening og den andre halvparten trene ballistisk styrketrening. Vi vil måle endring basalt, funksjonelt og selvopplevd.

Vurdering

Forsvarlighet
Studien har liten risiko med potensiell nytte for deltakerne. Prosjektet har beredskap slik at treningen ikke blir for hard. Komiteen diskuterte om studien kan klare å se forskjeller på to typer trening med få deltakere som kan ha store forskjeller i motorisk funksjonsnivå. Komiteen finner dog at studien har liten risiko og at den er forsvarlig å gjennomføre.

Forskningsansvarlig institusjon
Komiteen diskuterte om UiB også burde være forskningsansvarlig institusjon ettersom prosjektet er del av en doktorgrad ved UiB. Hvis UiB skal være forskningsansvarlig institusjon så må det søkes om i en prosjektendring.

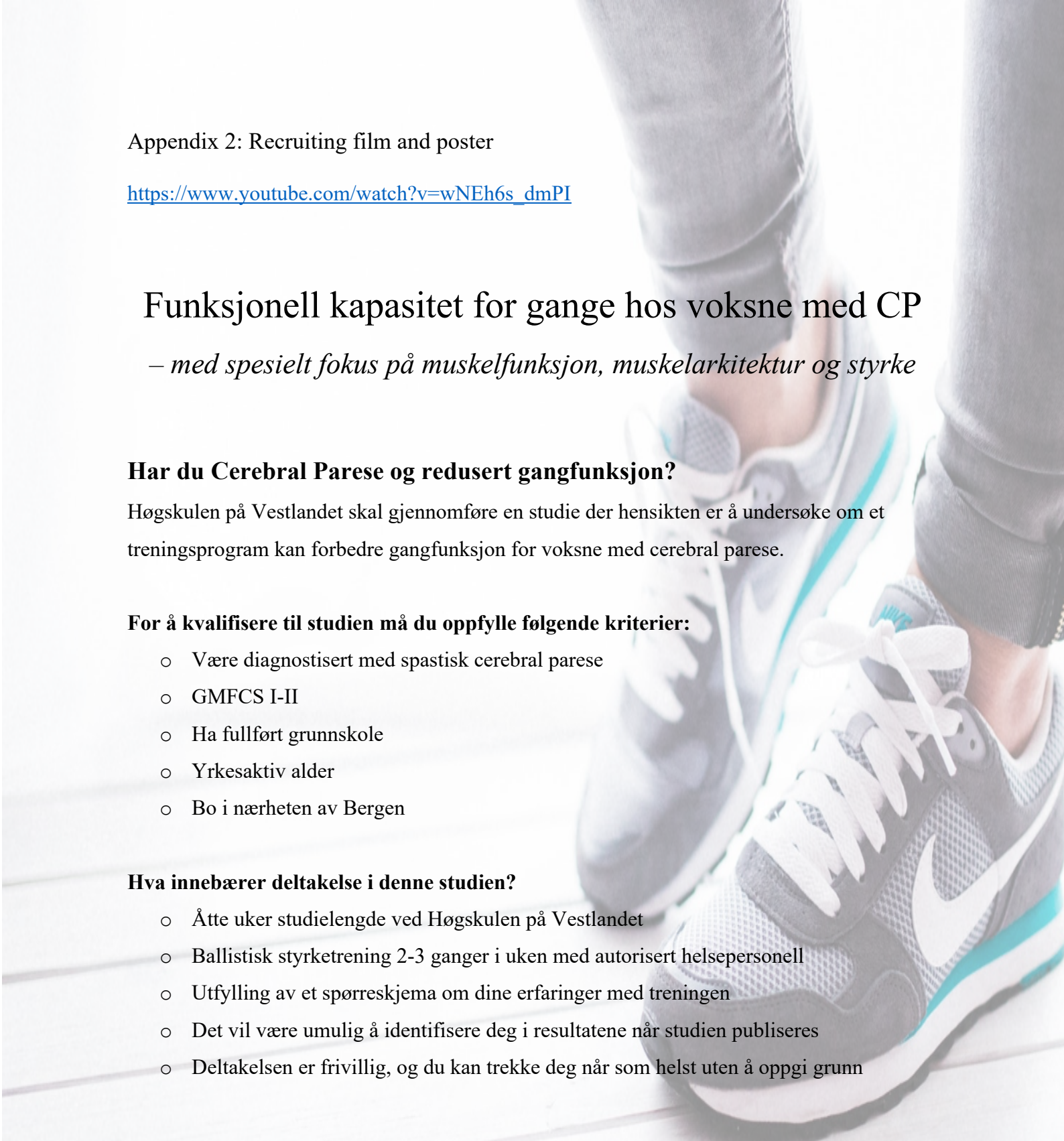
Humant biologisk materiale og biobank
Fullblod skal tas og serum og plasma skal lagres i en tidligere godkjent generell forskningsbiobank Navn på biobanken: Idrett, Helse og Funksjon: Biomarkører, ref. 2016/787. Navn på ansvarshavende: Elisabeth Ersvær, HVL. Det må informeres om at prøvene lagres i en generell biobank også i informasjonsskrivet om studien.

Besøksadresse:
Armauer Hansens Hus (AHH), Tverrfløy Nord, 2 etasje. Rom 281. Haukelandsveien 28

Telefon: 55975000
E-post: post@helseforskning.etikkom.no
Web: http://helseforskning.etikkom.no/

Appendix 2: Recruiting film and poster

https://www.youtube.com/watch?v=wNEh6s_dmPI



Funksjonell kapasitet for gange hos voksne med CP

– med spesielt fokus på muskelfunksjon, muskelarkitektur og styrke

Har du Cerebral Parese og redusert gangfunksjon?

Høgskulen på Vestlandet skal gjennomføre en studie der hensikten er å undersøke om et treningsprogram kan forbedre gangfunksjon for voksne med cerebral parese.

For å kvalifisere til studien må du oppfylle følgende kriterier:

- Være diagnostisert med spastisk cerebral parese
- GMFCS I-II
- Ha fullført grunnskole
- Yrkesaktiv alder
- Bo i nærheten av Bergen

Hva innebærer deltakelse i denne studien?

- Åtte uker studielengde ved Høgskulen på Vestlandet
- Ballistisk styrketrening 2-3 ganger i uken med autorisert helsepersonell
- Utfylling av et spørreskjema om dine erfaringer med treningen
- Det vil være umulig å identifisere deg i resultatene når studien publiseres
- Deltakelsen er frivillig, og du kan trekke deg når som helst uten å oppgi grunn

Ta gjerne kontakt dersom du ønsker mer informasjon eller ønsker å delta i prosjektet.

Kontakt person: Beate Gjesdal Eltarvåg
Stilling: Doktorgradsstipendiat, Høgskulen på Vestlandet
Telefon: +47 952 72 894
Epost: Beate.Eltarvag.Gjesdal@hvl.no

Appendix 3: Information letter and consent

Høgskulen på Vestlandet

FORESPØRSEL OM DELTAKELSE I FORSKNINGSPROSJEKTET

TRENING MED MÅL OM Å FORBEDRE GANGFUNKSJON HOS PERSONER MED CEREBRAL PARESE

Dette er en forespørsel til deg om å delta i et forskningsprosjekt der formålet er å undersøke om det er mulig å trene kraftutvikling i leggmuskulaturen som er relevant for fremdrift i gange. Forespørselen rettes til deg fordi du er voksen i yrkesaktiv alder (trenger ikke å være i arbeid), og har cerebral parese (CP). Du må ha fullført grunnskole for å kunne delta. Studien er en del av et doktorgradsprosjekt ved Universitetet i Bergen og Høgskulen på Vestlandet (HVL).

HVA INNEBÆRER PROSJEKTET?

Som deltager i denne treningsstudien vil du trene styrketrening i en periode som går over åtte uker. Det blir gjennomført tester før, undervegs og etter treningsperioden for å undersøke om treningen forbedrer gangfunksjonen din.

Når du er inkludert i studien vil du i en ukes tid før prosjektet starter gå med aktivitetsmåler. Dette er sensor som registrerer bevegelse. Denne sensoren skal du gå med en uke i strekk før, under og etter treningsperioden. I uken før treningsperioden starter skal du også gjennom omfattende testing som varer i ca. fire timer, testene utføres på Kronstad, Høgskulen på Vestlandet. Disse testene kartlegger en rekke fysiske parametere i muskelen som påvirkes med trening. Alle testene gjennomføres før og etter treningen, mens noen tester også vil bli brukt undervegs. Testene har til hensikt å undersøke egenskaper ved muskulaturen og bruk av muskulatur under gange og det blir brukt utstyr som ultralyd, tredimensjonal ganganalyse, elektromyografi og utstyr for å måle kraftutvikling.

Ballistisk styrketrening er trening med hurtig kraftutvikling som har til hensikt å bedre kraftutvikling under gange. For å undersøke at muskelen tåler denne typen trening vil vi etter første styrkeøkt ta blodprøver 30 min før økt, og 5 min, 1 time, 2 timer, 3 timer, 24 timer og 48 timer etter økten. Dette gjør vi for å se at muskulaturen tåler denne typen trening og når den er restituert til å tåle ny trening. Vi vil også måle når kraftutviklingen er tilbake til utgangskraften. Dette blir bare gjort etter første økt. I tillegg til fysiske tester og selvrapporterte spørreskjema vil vi også intervjue deg etter treningsperioden for dine erfaringer tilknyttet deltakelse.

De første fire ukene trener du to ganger i uken, og det økes til tre ganger i uken fra uke fem, eller individuelt tilpasset progresjon. To ganger i uken må du til Kronstad, Høgskulen på Vestlandet, for å trene. Estimert tidsbruk til hver økt er satt til 30 min.

MULIGE FORDELER OG ULEMPER

Deltakelse i prosjektet vil kreve tid og oppmerksomhet, og det kreves at du som deltager er tilstede på treninger og testdager. Vi har lagt opp disse dagene slik at de er tidseffektive og vil derfor ikke ta mer tid enn nødvendig.

Treningene er harde og kan oppleves ubehagelige. Det er alltid en viss sjanse for skader og overbelastning i forbindelse med styrketrening. For å forsikre oss om at treningen ikke fører til unødvendig skaderisiko vil den gradvis økes i belastning og mengde. Du kan oppleve forbigående muskelstølhet i etterkant av treningen.

De fysiske testene som utføres vil kreve maksimal innsats, og vil oppleves anstrengende. Dette kan oppleves som ubehagelig for noen, og det vil alltid være en viss risiko for skade under testen og følelse av stølhet i muskulaturen kan forekomme i etterkant.

I dette prosjektet vil det blir tatt blodprøve. Det er vanligvis ikke forbundet med smerte, men det kan oppleves ubehagelig. Du kan oppleve en lett til moderat ømhet i området hvor prøven er tatt, men dette vil kun vare 1-2 døgn og vil ikke hindre deg i å bevege eller bruke armen som før.

Om du skulle oppleve ubehag eller andre ting som du tror kan ha sammenheng med studien, kan du når som helst nå oss på telefon (telefonnummer finnes under).

FRIVILLIG DELTAKELSE OG MULIGHET FOR Å TREKKE SITT SAMTYKKE

Det er frivillig å delta i prosjektet. Dersom du ønsker å delta, undertegner du informert samtykkeerklæring på siste side. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke. Dersom du trekker deg fra prosjektet, kan du kreve å få slettet innsamlede opplysninger. Dersom du har spørsmål til prosjektet, kan du kontakte doktorgradsstipendiat Beate Eltarvåg Gjesdal, mobil 95 27 28 94, begj@hvl.no.

HVA SKJER MED INFORMASJONEN OG PRØVENE SOM BLIR TATT AV DEG?

Dataene og informasjonen som registres under testingen, skal brukes i henhold til formålet og hensikten med studien. Alle opplysningene vil bli behandlet uten direkte gjenkjennende opplysninger, som navn og fødselsnummer. Du vil ved forsøksstart få utdelt et forsøkspersonnummer (ID-nummer) som skal brukes under studien og det er bare dette nummeret som vil være tilknyttet til dine data. Det betyr at alle data vil bli behandlet konfidensielt og det vil ikke være mulig å identifisere deg i resultatene. Blodprøvene som tas av deg skal oppbevares i en forskningsbiobank lokalisert i Bergen. Ansvarshavende for biobanken (*Idrett, Helse og Funksjon: Biomarkører, ref. 2016/787*) er Elisabeth Ersvær.

Underveis i forsøket vil vi oppbevare en kodeliste med navn og forsøkspersonnummer. Denne kodelisten vil fysisk være låst inne, slik at det er kun forskerne tilknyttet studien som har adgang til den. Alle som får innsyn i informasjon om deg har taushetsplikt. Kodelisten lagres i 5 år etter prosjektslutt, og destrueres etter dette. Biobanken opphører ved prosjektslutt.

Prosjektet skal etter planen avsluttes 31.12.2023. Ved prosjektslutt vil transkriberte intervjuer anonymiseres og lydopptak slettes. I tillegg vil anonymiserte intervjuer, i tråd med Nasjonal strategi for tilgjengeliggjøring og deling av forskningsdata, bli offentlig tilgjengeliggjort i et egnet forskningsdataarkiv. Gjenbruk av

forskningsdata bidrar til vitenskapelige fremskritt, økt etterprøvbarhet og bedre kvalitetssikring av tidligere forskningsfunn.

Prosjektleder, Silje Mæland, har ansvar for den daglige driften av forskningsprosjektet og at opplysninger om deg blir behandlet på en sikker måte.

ØKONOMI

Reisegodtgjørelse med billigste kollektivtransport blir refundert.

GODKJENNING

Regional komité for medisinsk og helsefaglig forskningsetikk har vurdert prosjektet, og har gitt forhåndsgodkjenning (REK Vest 2018/2390-05)

Etter ny personopplysningslov har dataansvarlig, prorektor for forskning ved HVL Gro Anita Fonnes Flaten og prosjektleder Silje Mæland et selvstendig ansvar for å sikre at behandlingen av dine opplysninger har et lovlig grunnlag. Dette prosjektet har rettslig grunnlag i EUs personvernforordning artikkel 6a og artikkel 9 nr. 2 og ditt samtykke.

Du har rett til å klage på behandlingen av dine opplysninger til Datatilsynet.

SAMTYKKE TIL DELTAKELSE I PROSJEKTET

Hvis du har lest informasjonsskrivet og ønsker å være med som forsøksperson i prosjektet, ber vi deg undertegne nedenfor, og returnere skjemaet til en av personene oppgitt nedenfor. Du bekrefter samtidig at du har fått kopi av og lest denne informasjonen.

Det er frivillig å delta og du kan når som helst trekke deg fra prosjektet uten videre begrunnelse. Alle data vil, som nevnt ovenfor, bli avidentifisert før de blir lagt inn i en database, og senere anonymisert.

Med vennlig hilsen,

Beate Eltarvåg Gjesdal, PhD student, begj@hvl.no (mob. 95 27 28 94)

Silje Mæland, Førsteamanuensis, prosjektleder, smel@hvl.no (mob. 92 40 33 14)

JEG ER VILLIG TIL Å DELTA I PROSJEKTET

Sted og dato

Deltakers signatur

Deltakers navn med trykte bokstaver

Deltakers mobilnummer

Deltakers e-post

Appendix 4: Ballistic strength exercises

Frekvens:	2-3 ganger i uken
Intensitet:	Intensitetsnivået er individualisert og avgjøres av det maksimale nivået en deltaker kan utføre øvelsen med rett teknikk
Tidsaspekt:	8-ukers styrketreningsprogram
Dose:	Hver øvelse utføres i 5 minutter, der deltaker kan ta pause etter eget behov. Treningsinstruktør kan initiere i pauser dersom teknikken eller koordinasjonen forverres i løpet av økten. Denne tiden noteres.
Progresjon:	Øk hastighet når deltaker har kontroll på øvelsen og riktig muskulatur anvendes. Helningen på sleden økes individuelt. Helningen noteres.
Kommentar:	Eventuelle spesielle hendelser skal noteres. Dette innebærer smerte, lokasjon, fatigue, muskeltrøtthet eller andre symptomer.
Referanse:	Treningsprogrammet er inspirert av artikkelen: *«Feasibility of Ballistic Strength Training in Subacute Stroke: A Randomized, Controlled, Assesor-Blinded Pilot Study»* publisert av Hendrey et al. (8).

ØVELSE 1: JUMP SQUATS

Formål: Å trene kraftutviklingen i ankel plantarfleksorer og hofteekstensorer ved hovedfokus på hastighet og høyden på hoppet

Utstyr: Sleden (Power Tower), stoppeklokke, hodepute

Utgangsstilling: Deltaker ligger supinert med hofter i 30^{o} fleksjon og fotbladene plassert på platen. Armer plasseres i kryss over brystes, alternativt kan deltaker holde i sleden. Påse at fingrene ikke blir klemt Øvelsen illustreres i bildene nedenfor

Helning: Sledens helning bestemmes ut ifra i den grad deltakeren kan vedlikeholde bilateral ankel plantarfleksjon og kneekstensjon ved fraspark

Assistent: Manuell støtte til ankelen ved å gi mediolateral stabilitet og/eller støtte i kneet er tillat

Instruksjon: «Forestill at du hopper på stedet. Spark fra med tærne og rett ut knærne i hoppet. Du skal lande på tærne igjen. Utfør bevegelsen med maksimal hastighet, så høyt du kan. Pauser er tillatt når du ønsker»

Progresjon: Øk helningen med en enhet markert om gangen. Antall grader noteres.

Bilde: Illustrasjon av «Jump Squasts»

ØVELSE 2: SINGLE LEG HOPPING ON THE PARETIC LEG

Formål: Å isolere plantarfleksorer med fokus på hastighet og hopphøyde

Utstyr: Sleden (Power Tower), stoppeklokke, hodepute

Utgangsstilling: Deltaker ligger supinert med paretisk ben ekstendert mot fotplaten. Funksjonsfrisk fot plasseres på sleden. Armer plasseres i kryss over brystes, alternativt kan deltaker holde i sleden. Påse at fingrene ikke blir klemt. Øvelsen illustreres i bildet nedenfor

Helning: Sledens helning bestemmes ut ifra i den grad deltakeren kan oppnå «fly-fasen» ideelt

Assistent: Sørg for at aktiviteten isoleres til leggmuskulaturen ved å sikre at hofte og kne er ekstendert. Manuell støtte til ankelen ved å gi mediolateral stabilitet og/eller støtte i kneet er tillat

Instruksjon: «Hopp så høyt og fort du kan ved å sparke fra med forfoten. Pass på at kneet er strakt. Pauser er tillatt når du ønsker»

Progresjon: Øk helningen med en enhet markert om gangen. Antall grader noteres.

Bilde: Illustrasjon av «Single leg hopping on paratic leg»

ØVELSE 3: JOGGING ON ALTERING LEGS

Formål: Å koordinere kraftutvikling i hoftefleksorer, hoftekstensorer og plantarfleksorer ved hurtig jogging

Utstyr: Sleden (Power Tower), stoppeklokke, hodepute

Utgangsstilling: Deltaker ligger supinert med hofter i 30º fleksjon. Armer plasseres i kryss over brystes, alternativt kan deltaker holde i sleden. Påse at fingrene ikke blir klemt Illustrert i bildene nedenfor

Helning: Sledens helning bestemmes ut ifra i den grad deltakeren kan koordinere «fly-fasen» bilateralt med god kontroll.

Assistent: Manuell støtte til ankelen ved å gi mediolateral stabilitet og/eller støtte i kneet er tillat

Instruksjon: «Forestill deg at du jogger lett på tå. Fokuser på raske skift mellom hver gang tåen treffer platen. Pauser er tillatt når du ønsker»

Progresjon: Øk helningen med en enhet markert om gangen. Antall grader noteres.

Bilde: Illustrasjon av «Jogging on altering legs»

Appendix 5: Borg Scale 6-20

Borgs skala

- *Subjektiv følelse av anstrengelse*

Nivå	Følt anstrengelse
6	Hvile
7	Svært lett
8	
9	Meget lett
10	
11	Ganske lett
12	
13	Litt anstrengende
14	
15	Hardt
16	
17	Meget hardt
18	
19	Ekstremt hardt!
20	Maksimalt anstrengende!

Appendix 6: Training diary

Økt	FP1	FP2	FP3	FP5	FP6	FP7	FP9	FP10
1			løping mølle		Fysio			
2		basisball, Zumba	40 min roing			Jogge		
3		Roing og styrke	1 økt					Lett tur
4			Løping		Fysio	joggetur, tur		Fløyen, trapper, tur
5		Step/styrke, Zumba	Løping, gåtur			Tur		Fløyen
6		Svømming, styrke	Elipse maskin, styrke, Løping, skitur, gåturer		2 timers tur	Trente	Ulrikken	
7		Bootcamp, Zumba øvelser			tur		Turer til fots	
8			Gått mye		egentrening	rolig jogging, spinning	Fjelltur tirsdag	
9				Svømme	Egentrening		Svømmezumba, styrke	
10		3 økter siden sist	Ellipse		egentrening			
11		Basisball, step/styrke, øvinger fra Beate	Løping		egentrening		Basistrening	
12			fjelltur, løpetur	basseng		hjemmetrening	Crossfit	
13		Svømming, step/styrke, tilsendte øvelser	5 km løp				svømme basisball aerobic, zumba, styrke, egentrening	Uteaktiviteter
14		Basisball, tur, egentrening	fjelltur	Basseng, egentrening i vann	jogging, egentrening			byfjellene
15		Svømming, egentrening i basseng	løping			Fløyen	Basiasball, egentrening, fjelltur	
16							Styrke, crossfit	

Appendix 7: Organization of test days

Uke 14/15 Pretest

F317	Oppdatert samtykke Ultralyd	
F317/F318	Kliniske tester	
F327	EMG Akselerometer Markører Ganganalyse	
F338	6MWT Con-Trex HiMAT	

Uke 14 – Trening

Bruker EMG og kraftdata under den andre treningen. EMG markører på m. gastrocnemius mediale, bilateralt. Kraftdata er på platen.

Uke 15 Blodprøver

Blodprøve 1 + trening	Blodprøve 30 min før økt Contrex Trening Blodprøve 5 min etter test Blodprøve 1h etter test	Contrex: Trening:
Blodprøve 2 + con-trex	Blodprøve Con-trex	Con-trex:
Blodprøve 3 + trening	Blodprøve 48h Con-trex Trening	Con-trex: Trening:

Uke 17 Ultralyd + trening

Først ultralyd på F318, så trening på F327.

Blodprøve 1 + trening	Blodprøve 30 min før økt Contrex Trening Blodprøve 5 min etter test Blodprøve 1h etter test	Contrex: Trening:

Uke 18 6MWT + trening

6MWT + trening	6MWT: Akselerometer + EMG Trening med kraftdata	6MWT: Trening:

Uke 19 3D + trening

3D + trening	3D: EMG + markører Trening	3D: Trening:

Uke 21 – Trening

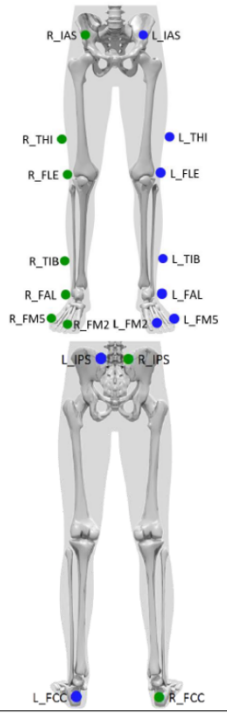
Bruker EMG og kraftdata under den andre treningen. EMG markører på m. gastrocnemius mediale, bilateralt. Kraftdata er på platen.

Uke 22 Posttest

F317	Ultralyd	
F317/F318	Kliniske tester	
F327	EMG Akselerometer Markører Ganganalyse	
F318	6MWT Con-Trex HiMAT	

Appendix 8: CGM lower body marker set

Qualisys PAF package: CGM lower body marker set



Name	Ref. [1]	Location	Static (18)	Dyn. (18)
L_IAS	IAS	Anterior superior iliac spine	X	X
L_IPS	IPS	Posterior superior iliac spine	X	X
R_IPS	IPS	Posterior superior iliac spine	X	X
R_IAS	IAS	Anterior superior iliac spine	X	X
L_THI		Lower lateral 1/3 surface of the thigh, the height is not critical	X	X
L_FLE	FLE	Lateral epicondyle	X	X
L_TIB		Lower lateral 1/3 surface of the shank, the height is not critical	X	X
L_FAL	FAL	Lateral prominence of the lateral malleolus	X	X
L_FCC	FCC	Aspect of the Achilles tendon insertion on the calcaneus	X	X
L_FM2	FM2	Dorsal aspect of the second metatarsal head	X	X
L_FM5	FM5	Dorsal margin of the fifth metatarsal head	X	X
R_THI		Lower lateral 1/3 surface of the thigh, the height is not critical	X	X
R_FLE	FLE	Lateral epicondyle	X	X
R_TIB		Lower lateral 1/3 surface of the shank, the height is not critical	X	X
R_FAL	FAL	Lateral prominence of the lateral malleolus	X	X
R_FCC	FCC	Aspect of the Achilles tendon insertion on the calcaneus	X	X
R_FM2	FM2	Dorsal aspect of the second metatarsal head	X	X
R_FM5	FM5	Dorsal margin of the fifth metatarsal head	X	X

[1] Sint Jan, S. Van (2007). Color Atlas of Skeletal Landmark Definitions. Guidelines for Reproducible Manual and Virtual Palpations. Edinburgh: Churchill Livingstone.

Appendix 9: Gait analysis protocol

Denne ganganalysen skal utføres med EMG og markører.

Før personen kommer må utstyret kalibreres.

1) Kalibrere utstyr
2) Åpne QTM

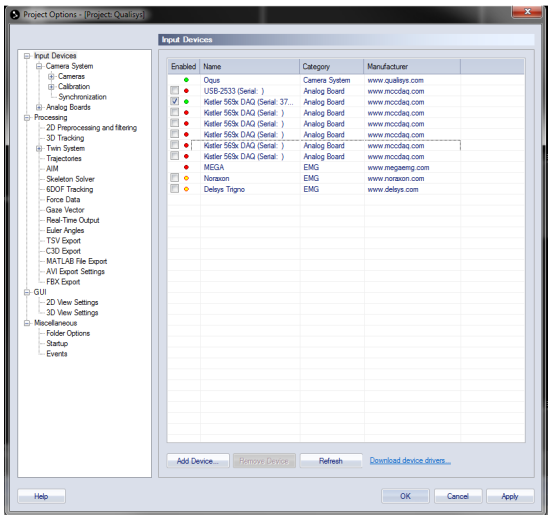
Kameraplassering

1) Kontroller i Qualisys Track Manager (QTM) at kamerarekkefølgen stemmer og korriger om nødvendig. (Ctrl+N, velg «2D» i menyen på venstre side)

Kraftplate + EMG

1) Sjekk i innstillinger at kraftplaten (Kistler 569x DAQ) er lagt til (Innstillinger - Input Devices).

Noraxon

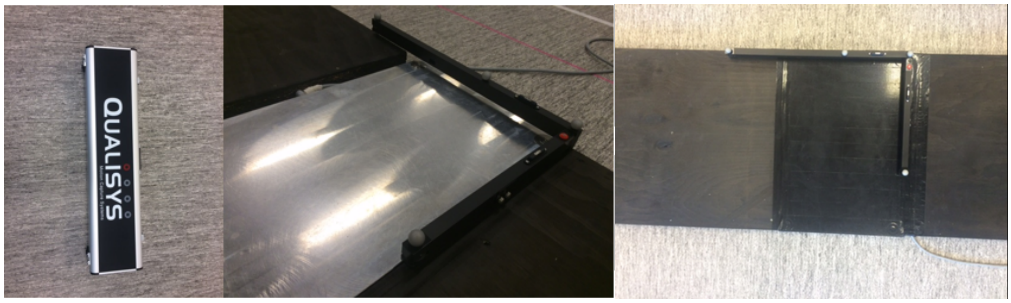


Ctrl + D

Sjekk Kistler og EMG

Kalibrering ved bruk av kraftplate i gangbane

L-ramme og kalibreringsstav ligger i Qualisyskassen. Påse at du ikke har på deg klær eller sko med tydelige reflekser. Dette kan kontrolleres ved å gå i opptaksvolumet og observere i QTM.



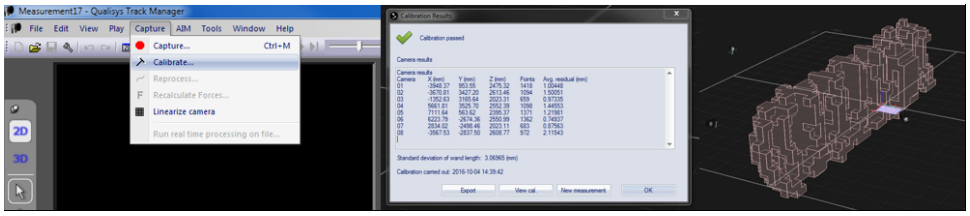
Venstre: Qualisyskassen. Høyre: Plassering av L-ramme på gangbanen

L-ramme plasseres med den korte armen i retning mot vinduene og den lange armen i retning vekk fra kontrollbordet. Armene settes på kraftplaten, i flukt med X og Y-retning. Kontroller at alle åtte kameraene registrerer de fire markørene på L-rammen.

Gjennomfør et kalibreringsopptak på 45 sek (Lengre om nødvendig for å dekke ønsket opptaksvolum). Kalibreringsstaven føres rundt i hele det ønskede opptaksvolumet.

For ganganalyse: Ved gange på gangbanen er det nok å dekke området mellom de to hvite merkene på siden (ca. 1,5m fra kraftplaten i hver retning). Pass på å få med så nært bakken som mulig, men ikke la markørene skrape borti gulvet.

Etter 60 sek kommer «Calibration results» opp på skjermen. «Average Residual» bør ligge under 1.0 mm på alle kameraene. Deretter skal kalibreringsvolumet kontrolleres visuelt. Om «Average Residual» eller kalibreringsvolumet ikke er tilfredsstillende må kalibrering gjennomføres på nytt.



Venstre: Her startes kalibrering i QTM. Midten: Calibration results og Average Residual. Høyre: Visuelt kalibrert volum.

2.6 Kalibrering av kraftplate - kun når kraftplate ikke er lokalisert i origo

Ettersom kraftplaten er mobil må lokaliseringen kalibreres før bruk. Det gjøres på følgende måte:

1. *Plasser en markør i hvert hjørne av kraftplaten.*
2. *Gjør et kort opptak i QTM (1-2 sek)*
3. *Label markørene 1, 2, 3 og 4*
4. *Lagre opptak*
5. *Gå til "Project Option - Force Data – Force-plate 1"*
6. *Trykk på "Generate". Om denne ikke lar seg trykke på, kontroller punkt 1-3.*
7. *QTM finner to mulige løsninger. Trykk OK og velg den som stemmer overens med markører*

1.1 Personer det blir utført målinger på (heretter testperson) skal på forhånd være informert om korrekt bekledning (shorts og evt sports-BH uten reflekser) og være gjort kjent med at opptak kan bli brukt i senere studier. Alle testpersoner skal signere skjemaet "Testing i rehabiliteringslab", i tillegg til evt. eget skjema for spesifikt prosjekt.

Før opptak:

- ❒ Når opptaksvolumet er kalibrert - sjekke at kraftdata og EMG signal registreres – se labens protokoll
- ❒ Finn frem EMG elektroder – 12 stk
- ❒ Gjøre klare refleksmarkører

3.1 Antropometriske mål

Følgende mål må registreres og legges inn som notat i testpersonens mappe i prosjektet;

Vekt

Høyde

Benlengde (V og H) målt fra SIAS til laterale maleol.

Knebredde (V og H) målt på det bredeste uten press mot bløtvev

Ankelbredde (V og H) målt på det bredeste (maleoler

Appendix 10: 3D capture – Registration form

FP_____ Dato:_____ Test: T_0 / T_1 / T_2

Tid:	
Utført av:	

ASIS, bendlengde, knebredde og ankelbredde måles på de kliniske testene.

Kjønn				
Alder			Venstre	Høyre
Vekt		Benlengde		
Høyde		Knebredde		
ASIS		Ankelbredde		

Kommentar/Notat;

Opptaksnotat:

NB! Hælslipp før ganganalyse – skriv inn tidspunkt på akselerometer-arket

Statisk opptak

❒ Utgangsposisjon

- Instruks: tær pekende rett frem, hoftebreddes avstand, vær obs på hyperekstenderte knær.

Navngigning: FUNCAP_FPx_T0_Stat / FUNCAP_FPx_T0_Dyn_x

Dynamisk opptak – 3 godkjente høyretreff og 3 godkjente venstretreff

❒ Foretrukket ganghastighet

Opptak	L	R
1		
2		
3		
4		
5		
6		
7		
8		
9		
10		
11		
12		
13		
14		
15		
16		
17		
18		
19		
20		

Opptak	L	R
21		
22		
23		
24		
25		
26		
27		
28		
29		
30		
31		
32		
33		
34		
35		
36		
37		
38		
39		
40		

Opptak	L	R
41		
42		
43		
44		
45		
46		
47		
48		
49		
50		
51		
52		
53		
54		
55		
56		
57		
58		
59		
60		

NB!! Hælslipp etter ganganalyse – skriv inn tidspunkt på akselerometer-arket

Opptaksnotat: