FH Technikum Wien

Documentation of my research stay

at the Florida Gulf Coast University, Florida, USA

Documentation submitted in partial fulfilment of the requirements for the Marshall Plan Scholarship

MOTION ANALYSIS OF PARKINSON`S DISEASE PATIENTS AND STROKE PATIENTS

by

Mag. Hans Kainz
Max-Mell-Weg 22, A-8132 Pernegg, Austria, hans.kainz@gmx.net

Supervisor 1: Dr. James Sweeney
Supervisor 2: Dr. Kristine Csavina

FACHHOCHSCHULE
TECHNIKUM WIEN

„LIVE AND LEARN“

George Gascoigne (1539-1577)

Abstract

Background: Most people with Parkinson`s disease (PD), as well as post-stroke patients, have problems walking and showing an inefficient gait patterns. The current work is divided into two studies which focus on the following topics: (1) spinal mobilization treatment for people with PD, and (2) body weight supported treadmill training for post-stroke patients. Both studies analyzed the effect of the treatment/ training on gait recovery.

Methods: In both studies, a three-dimensional, infrared motion capture system was used to capture kinematic data. In the first study, motion parameters of 17 participants with PD before and after one session of a spinal mobilization therapy were compared to motion parameters of 13 age-matched healthy participants. In the second study, 10 post-stroke subjects were divided into two groups. One group received conventional over-ground gait training (CT) and the other group received body weight supported (BWS) treadmill gait training. After discharge from the hospital gait parameters were compared between both groups and a control group with participants without a stroke history.

Results: In the first study, significantly improved motion parameters were found immediately after participants with PD received one spinal mobilization treatment. In the second study, most gait parameters showed superior values for the BWS group. However, clinical evaluation showed a higher improvement in walking for the CT group when compared to the BWS group.

Conclusion: People with PD benefit from a spinal mobilization treatment in terms of an increased mobility and superior gait parameters. In the second study, it was not possible to make a clear conclusion regarding whether BWS training or CT has more advantages in regaining normal gait pattern in post-stroke patients due to the small number of participants.

Keywords: Parkinson`s disease; stroke; motion analysis; biomechanics.

Acknowledgements

This work would not have been possible without the support of many individuals. First and foremost I would like to thank the Marshall Plan Foundation for giving me the unique opportunity to do a research stay in the USA. Without the financial support from the Marshall Plan Foundation, this research stay would not have been possible.

My gratitude also goes to Mrs Angelina Kratschanova (FH Technikum Wien), Mrs Elaine Hozdik (FGCU), and my supervisors Dr. Kristine Csavina (FGCU) and Dr. James Sweeney (FGCU) for helping me with the organization and paperwork for my scholarship.

I would like to gratefully acknowledge all my workmates, who were involved in these projects. In particular, Dr. Kristine Csavina, Dr. Mollie Venglar, Dr. Thomas Bevins, Dr. Arie van Duijn, Alecia Popovich, Valeria Suarez, and Rachelle Yusufbekov. Without these people it would not have been possible to complete these projects, especially within the given time frame.

I am grateful to Donald Hansen, Honorary Consul General of Luxembourg, and Gerda Hansen, Honorary Consul of Austria, for their hospitality and all the interesting discussions we had.

In addition, I would also like to express my appreciation to all people that I met during my stay in the USA. It has been a pleasure spending my time with each and all of you.

Further, I would like to thank programme director Dr. Martin Reichel (FH Technikum Wien) for always having an open door and his kindness in regard to my research stay.

My final gratitude goes to my family, especially to my mother and my sister. I want to thank them for always supporting me in whatever I pursue.

Table of Contents

Preface

The primary aim of my research stay was to determine if body weight supported treadmill gait training is superior to conventional gait training in post-stroke patients as it relates to regaining normal gait pattern. Due to a lack of post-stroke subjects (only 5 participants in each group), my main duty, besides the stroke study, was to analyze if one spinal mobilization treatment increases mobility and improves gait mechanics in people with Parkinson`s disease. Both studies focused on gait biomechanics, which was the overall topic of the research assignment from the Marshall Plan Foundation.

Some findings of my work were presented as two posters at the Biomedical Engineering Society Conference in Atlanta. Both posters are attached to the annex of the current work (Annex A and B).

For the motion analyis it was necessary to create some virtual markers and several pipelines. Annex C to I show documents and pipelines which were created by me.

This work aims to provide an overview of my duties at the Florida Gulf Coast University and also includes the theoretical background of the addressed topics.

HK

1 Theoretical background

1.1 Human movement

Gait, as well as all other voluntary movements, results from a complicated process, which involves the brain, spinal cord, peripheral nerves, muscles, tendons, bones, and joints. The spinal cord, which is an extension of the brain, is a bundle of nerve fibers that is connected to the peripheral nerves. The axons of the peripheral nerves, in turn, stimulate muscle fibers resulting in a contraction of the related muscle. Tendons connect muscles and bones, whereby the contraction of a muscle leads to movement of the connected bone [1]. To understand the fundamentals of human movement it is necessary to have some knowledge about motor control and the underlying neural system, which are explained on the following pages.

1.1.1 Motor control

Motor control desribes the process by which the central nervous system (CNS) receives, integrates, and assimilates sensory information with past experiences for planning and executing appropriate motor response. All parameters of human movement and how the CNS controls them is not fully understood yet but scientists in this field developed several models (e.g. neurologic, biomechanical, and behavioral models) which assist to understand the fundamental concept of motor control. One of the major questions in motor control is: How does the brain control so many different joints and muscles with all these „degrees of freedom“? It is assumed that the brain simplifies this task to function collectively (muscle synergies or coordinate structures). Motor programs that specify fixed relationships among muscles are stored in memory and can be recalled to guide an action. If a motor program is chosen the order of muscles contraction is fixed but the absolute level of force and the duration of the program can vary. The spring model from Fel´dman explains the control of the final position of a limb by a balanced agonist and

antagonist activity. An alternate explanation to the motor program theory is that the CNS creates solutions for actions whenever they are needed. A new approach to explain changes from one action pattern to another is based on principles of open systems. Synergies emerge and are constrained by the physical characteristics of the human body and the environmental contex in which actions are performed. If one element in a chain is activated, the others will be activated in a fixed order too. By constraining the relationship between the elements a new property can emerge and create a new order. Under this assumption the change from walking to running can be explained by an increased velocity [2]. The central pattern generator consists of networks of neurons in various parts of the brain and spinal cord and produces pattern of nerve impulses which are sent to muscles. Animal studies showed that a rhythm generating system in the spinal cord exists, which is controlled by neuronal input from the brain and receives feedback from sensors in muscles, joints and skin [1], [3]. These theroies were just some approaches to explain motor control. For further information about motor control refer to Rosenbaum [4] or to Montgomery and Connolly [2].

Sensory receptors play a major role in regulating motor behavior at spinal and higher center levels. Mechanoreceptors, for example, serve to assess the internal and external environment. On the spinal level motor control occur through reflexsive activity, as well as through regulation of muscle length and force. The basal ganglia are involved in motor processing and motor learning. From the cerebral cortex a major circuit arises, projects to the basal ganglia, and returns to the cortex via the thalamus. The nuclei of the basal ganglia control specific parameters of movements including velocity, amplitude and direction of the action. The basal ganglia are involved in the initiation, executing and completing of movements. The cerebellum has several functions in motor control. It is involved in planning and executing movements, regulating postural adjustments and serving as comparator between the actual movement and feedback. The motor systems of the cerebral cortex function as modules. Development of a motor program is a shared role between the motor cortex, the premotor cortex, the supplementary motor area, the posterior parietal region, and various subcortical centers. Depending on the movement, a different module will serve as the predominant functional unit [2].

1.1.2 Gait

Neurophysiology of gait

It is assumed that the basic motor pattern for stepping is generated in the spinal cord and that various brain regions, including motor cortex, cerebellum, and brain stem, are only involve in fine control of walking. The spinal cord has Central Pattern Generators (CPGs) which are networks of nerve cells producing specific, rhythmic movements such as walking, without conscious effort. CPGs are responsible for hard wired-synergy and produce and coordinate locomotion. The motor cortex modifies these synergies in complex demands of gait related activities. Each leg has its own autonomous pattern generating networks and can therefore operate independently. Figure 1 summarizes the role of the CNS in walking.

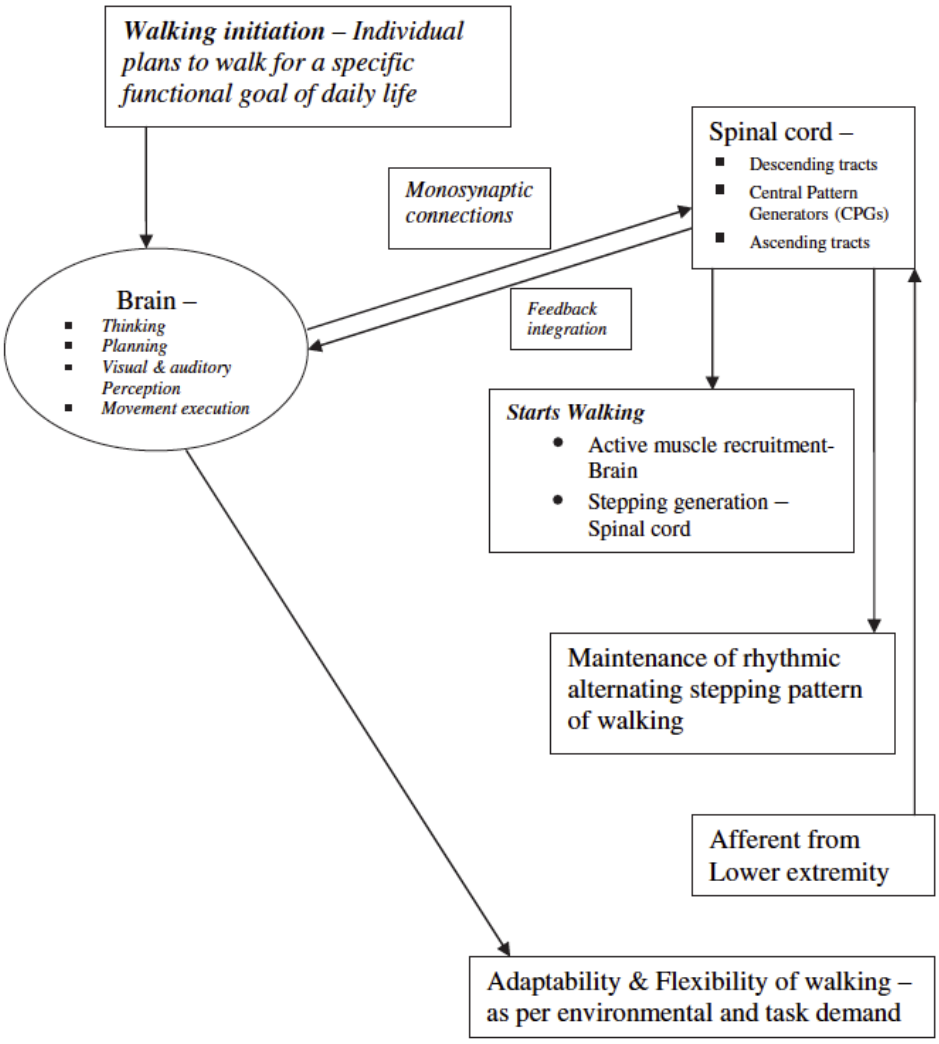


Figure 1: Role of brain and spinal cord in walking ([5], p. 16).

Phases of gait

The aim of walking is to move the body forward while maintaining stance stability. There are several ways to subdivide the gait but the most common way is the subdivison into following eight phases: initial contact, loading response, mid stance, terminal stance, pre swing, initial swing, mid swing and terminal swing [6].

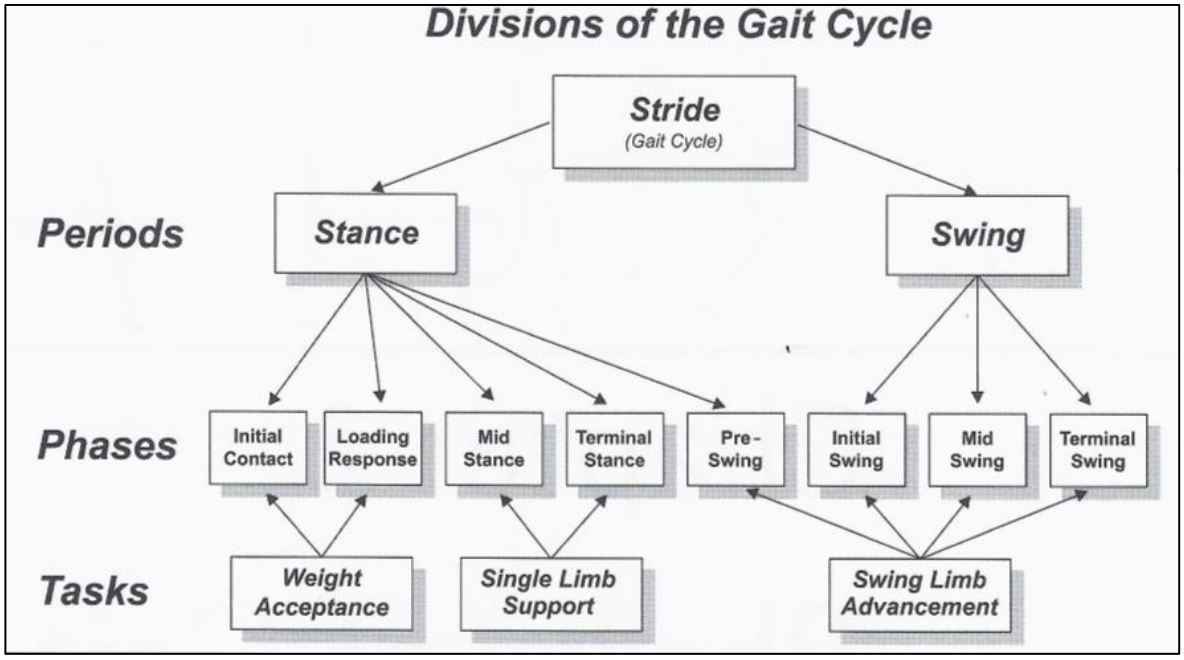


Figure 2 shows the functional division of the gait cycle.

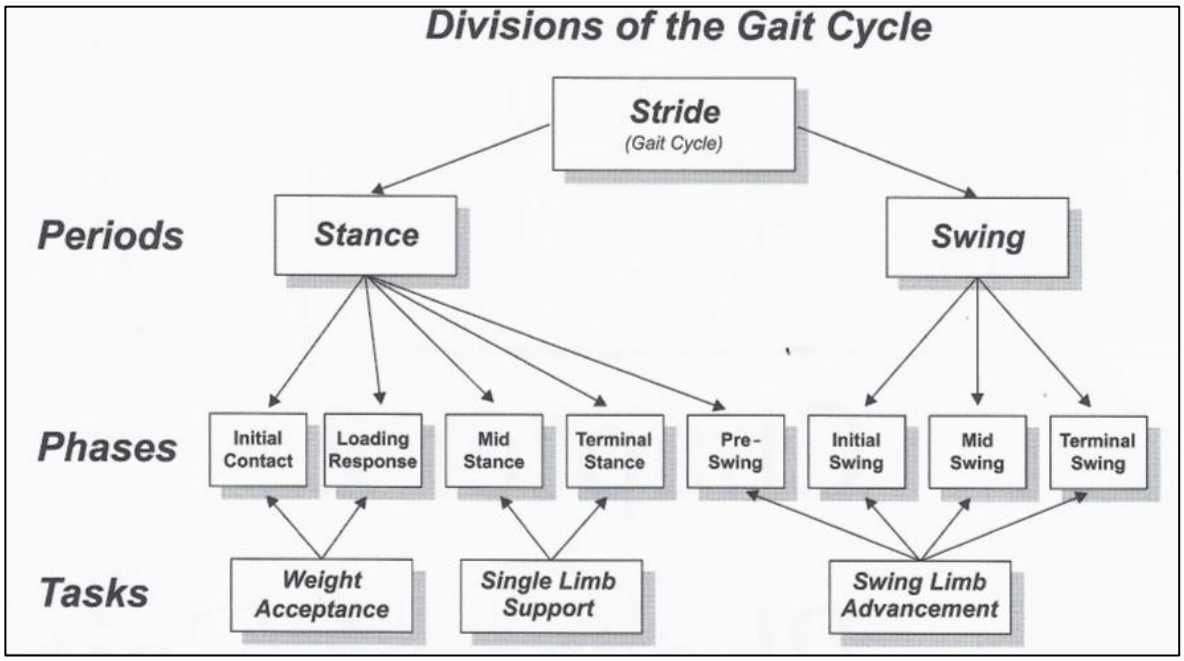


Figure 2: Functional division of the gait cycle. A stride is the functional term for the gait cycle. The periods show the basic division of the gait cycle by foot contact. Each phase is determined by limb postures. The tasks show the grouping of the phases by the functions to which they contribute ([6],p.10).

Each phase has its own objectives and is characterized by a selective synergistic motion to achieve these goals. The main task of the first two phases (initial contact and loading response) is weight acceptance, which includes shock absorption, initial limb stability and the preservation of progression. The objectives of phase one (initial contact) are to start stance with a heel rocker and to decelerate the impact. This phase constitutes 0% to 2% of the gait cylce and is characterized by a flexed hip, an extended knee and a dorsiflexed to neutral ankle. The aim of the second phase (loading response) is the transfer of the body weight onto the forward limb. The heel thereby serves as a rocker and the knee is flexed for shock absorbtion. Loading response constitutes 2% to 12% of the gait cycle and ends at the time when the other limb is lifted for swing, which also indicates the end of the double stance period. The main task of the next two phases (mid stance and terminal stance) is the support of the single limb. The objectives of these two phases are limb and trunk stability and the progression of the body over the stationary foot (mid stance) and further beyond the supporting foot (terminal stance). Mid stance constitutes 12% to 31% of the gait cycle and ends when the body weight is aligned over the forefoot. Ankle dorsiflexion forces the limb to advance over the stationary foot while knee and hip extend. Terminal stance begins with heel rise and ends when the other foot strikes the ground. This phase constitutes 31% to 50% of the gait cycle. Heel rise and an increased hip extension serve for progression of the body. The task of the last four phases, which include pre-swing, initial swing, mid swing and terminal swing, is the advancement of the swing limb. Pre-swing constitutes 50% to 62% of the gait cycle and is the second (terminal) double stance interval in the gait cycle. This final phase of stance is characterized by an increased ankle plantarflexion, knee flexion and a reduction of hip extension. The forward “push” of the trailing extremity accelerates progression and prepares the limb for the demands of swing. Initial swing constitutes 62% to 75% of the gait cycle and begins as the foot is lifted from the ground and ends when the swinging foot is opposite the stance foot. In this first phase of swing increased knee flexion lifts the foot for toe clearance and hip flexion advances the limb. Mid swing constitutes 75% to 87% of the gait cycle and ends when the swing limb is forward and the tibia is vertical. Further hip flexion induces to an advancement of the limb anterior to the body weight line. Terminal stance

constitues 87% to 100% of the gait cycle and ends when the foot strikes the floor. Limb advancement is completed by knee extension and the limb is prepared for stance. Ankle is in a dorsiflexed to neutral position [6]. Figure 3 illustrates all phases of the gait cycle.

There are sex-related and age-related differences in the “normal” gait, whereby an appropriate “normal” standard needs to be choosen for every individual [1].

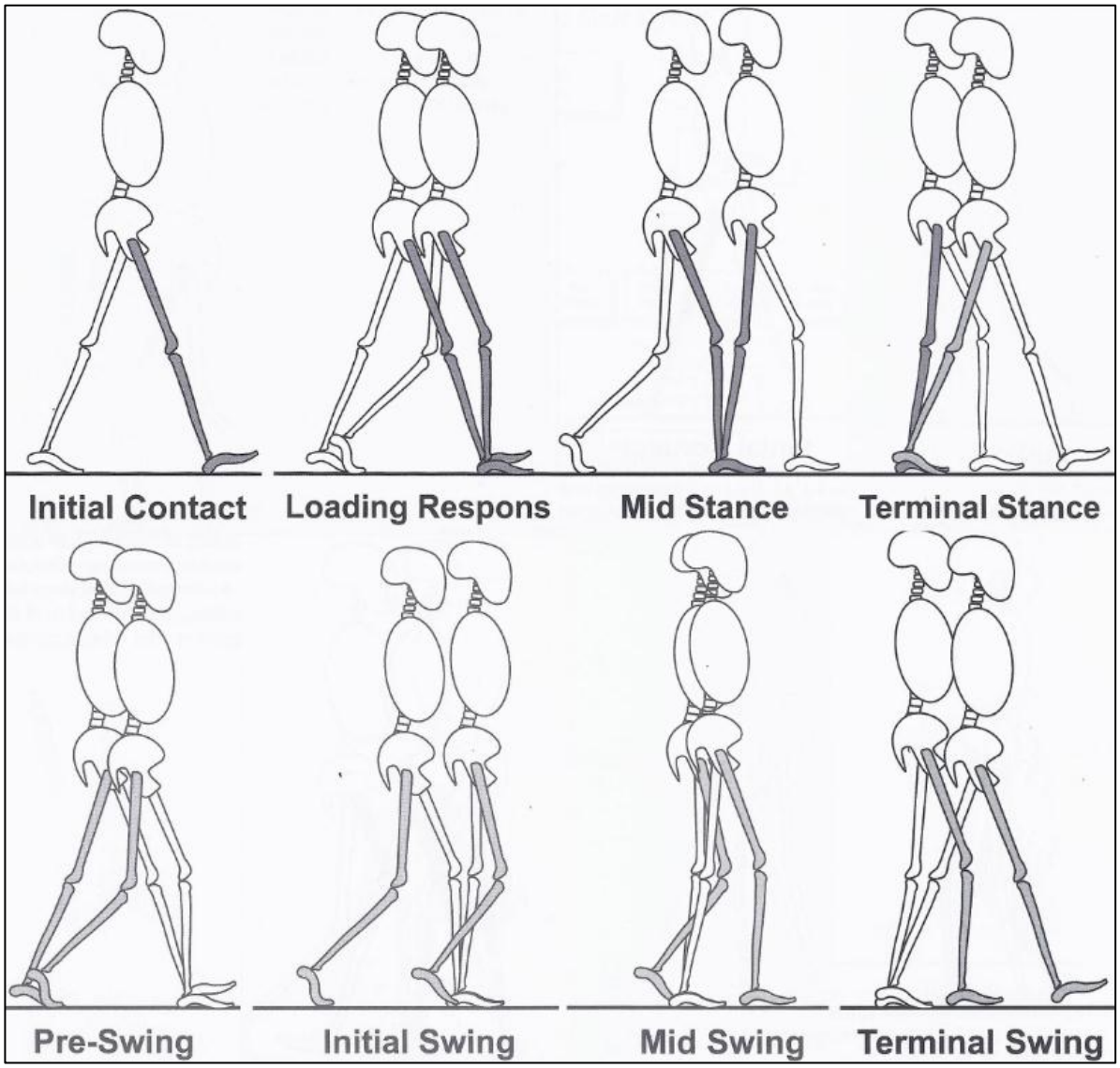


Figure 3: All eight phases of the gait cycle. Shading indicates the reference limb ([6],p.11-16 modified).

1.2 Motion analysis

Many different methods for motion analysis are available. Depending on the demands of the analyses either a simple, cheap device or a more complex and expensive system should be chosen. Videotape examination, analysis with electrogoniometers or force sensor systems, pedobarography, electromyography, energy consumption measurements, analysis with accelerometers or kinematic systems are just a few examples for the variety of methods which are available [1]. The aim of this chapter is not to explain all these different methods, but to provide a short overview of the kinematic system, which was used in the present work.

1.2.1 Motion capture system (Qualisys, Gothenburg, Sweden)

An 8-camera OQUS 300 1.3MP infrared motion capture system (Qualisys, Gothenburg, Sweden) was used to capture kinematic data. For data collection, reflective markers were placed on bony landmarks of the feet, legs, pelvis, and shoulders of each subject. Following 44 markers (Figure 4) were placed on the subject`s skin to define the gait model of the present work:

- RSHLD = Right schoulder, placed on the superior surface of the acromion.
- LSHLD = Left shoulder, placed on the superior surface of the acromion.
- RIC = Right iliac crest, placed on the superior iliac crest.
- LIC = Left iliac crest, placed on the superior iliac crest.
- RASIS = Right anterior superior iliac spine
- LASIS = Left anterior superior iliac spine
- RGT = Right greater trochanter of femur

- LGT = Left greater trochanter of femur
- RTH1 – RTH4 = Right thigh 1 – 4, four markers in a cluster placed on the thigh.
- LTH1 – LTH4 = Left thigh 1 – 4, four markers in a cluster placed on the thigh.
- RLK = Right lateral knee, placed on the lateral epicondyle.
- LLK = Left lateral knee, placed on the lateral epicondyle.
- RMK = Right medial knee, placed on the medial epicondyle.
- LMK = Left medial knee, placed on the medial epicondyle.
- RSK1 – RSK4 = Right shank 1 – 4, four markers in a cluster placed on the shank.
- LSK1 – LSK4 = Left shank 1 – 4, four markers in a cluster placed on the shank.
- RLA = Right lateral ankle, placed on the lateral malleolus.
- LLA = Left lateral ankle, placed on the lateral malleolus.
- RMA = Right medial ankle, placed on the medial malleolus.
- LMA = Left medial ankle, placed on the medial malleolus.
- RFT1 = Right foot 1, placed on the medial aspect of the 1st metatarsal head.
- LFT1 = Left foot 1, placed on the medial aspect of the 1st metatarsal head.
- RFT2 = Right foot 2, placed on the lateral aspect of the 5st metatarsal head.
- LFT2 = Left foot 2, placed on the lateral aspect of the 5st metatarsal head.
- BK1 – BK4 = Back 1 – 4, four markers in a cluster placed on the lower back.
- RPSIS = Right posterior superior iliac spine
- LPSIS = Left posterior superior iliac spine
- RHEEL = Right heel
- LHEEL = Left heel

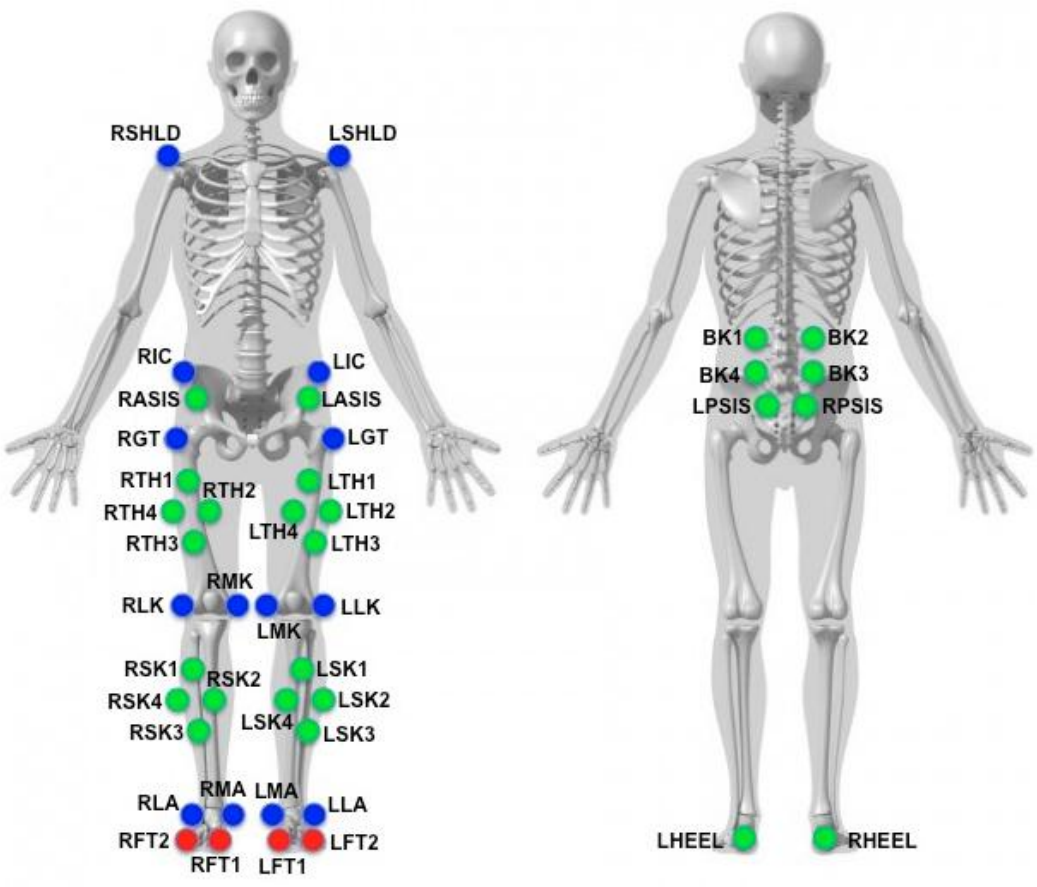


Figure 4: Marker setup. Blue markers were used for the segment definition. Green markers were only used for tracking. Red markers were used for both, the segment definition and for tracking ([7], modified).

Before capturing of the desired motion, a static trial was capured of every subject. Subjects had to stand still with their arms stretched besides for approximately 3 seconds. Afterwards, the medial knee markers and the medial ankle markers were removed and subjects were introduced to do the desired motion. The static trial was necessary for model development in Visual3D, which will be explained on the next pages. Upon data collection QTM software (Qualisys Track Manager, Qualisys, Gothenburg, Sweden) was used to evaluate if all markers were present. After all markers were labeled and gap-filled with the QTM software, data were exported as c3d files for processing in Visual3D.

1.2.2 Data analysis software (Visual3D, C-motion, Germantown, MD, USA)

Data analysis was conducted with Visual3D software (C-motion, Germantown, MD, USA). Exported c3d files from QTM were imported into Visual3D. A Visual3D gait model with a torso, pelvis, left and right thigh, left and right shank, and left an right foot segment was applied to the static trial of each subject. The torso segment was defined by the shoulder markers (right and left) and the iliac crest markers (right and left). BK1 to BK4 markers served as tracking markers for the torso. The pelvis was defined by the iliac crest markers and the greater trochanter markers. Anterior and posterior superior iliac spine markers were used for tracking the pelvis segment. Thighs were defined by the greater trochanter markers and the medial and lateral knee markers. The four markers in a cluster on each thigh (LTH1-4 and RTH1-4) served as tracking markers for the thighs. Shanks were defined by the medial and lateral knee markers and the medial and lateral ankle makers. The four clustered markers on each shank were used for tracking the shank segments. Feet were defined by both ankle markers (medial and lateral) and foot markers 1 and 2 (1^{st} and 5^{th} metatarsal head). The foot markers 1 and 2 together with the heel markers were also used for tracking the feet segments. Additional markers were placed on the right and left elbow and on the right and left hand. These additional marker were not considered in any analyses of the current work and therefore these markers are not mentioned in the text and not pictured in Figure 4. Figure 5 shows a stroke patient with all markers on his body. After the gait model was applied to the static trial, dynamic trials were imported and the model was appended to the dynamic trials. In some subjects a marker in the static or dynamic trial was missing. In such a case it was not possible to apply the gait model in a proper way and it therefore was necessary to create a virtual marker. Appendix C and D include instructions how to create a virtual marker in a static and dynamic trial. Several pipelines (some of them can be found in appendix E to I) and reports were used to calculate joint range of motion values and spatiotemporal gait data.

Figure 5: Stroke patient with all markers on his body from the front (left picture) and from behind (right picture). The belt, which the physiotherapist is holding in the right picture was used for security reasons. In the case that the participant would stumble, the physiotherapist would be able to prevent the participant from falling.

1.3 Parkinson`s disease

Parkinson`s disease (PD) is a neurological condition which afflicts more than half a million peple in the U.S. alone. James Parkinson first described this disease in 1817 [8].

Two major clinical subtypes of PD exist: A tremor-predominant form and a type known as “postural imbalance and gait disorder” (PIGD). The tremor-predominant type is often observed in younger people. The PIGD type is characterized by akinesia, rigidity, and gait and balance impairment. This type is observed in older people (70 years or above). In general, the first subtype leads to a slow decline of motor function, whereas the PIGD subtype worsens more rapidly [9].

1.3.1 Clinical characteristics of PD

PD is characterised by following four main motor disorders: [10]

- Paucity of movement
- Rigidity
- Tremor
- Postural instability

Paucity of movement is the most characteristic motor symptom of basal ganglia dysfunction in PD and consists of slowness of initiation of movement with progressive reduction in speed and amplitude of repeated movements. Paucity of movement therefore includes following three symptoms: bradykinesia (=slowness of movement), hypokinesia (reduced movement) and akinesia (inability to initiate movement). These terms are often used interchangeably. Bradykinesia is a major cause of disability in people with PD because it affects activities of daily living such as dressing, preparing meals, eating and bathing [10].

Rigidity describes the increased resistance to passive movements around a joint. It can affect any part of the body. The increase in muscle tone lead to joint movements that feel like the joint is moving through teeth of a cogwheel. Rigidity is

more evident in flexors than in extensors, which contributes to the classical flexed posture of PD patients [10].

The typical tremor in people with PD occurs at rest and disappears or decreases with action. It is fairly slow with a frequence of 4 to 6 Hz. The hands are the most prominent area of the tremor but it can also appear in the lower extremities and the chin/jaws [10].

Postural instability appears at an advanced stage of PD and signifies the transition from mild to moderate PD, as defined by the Hoehn and Yahr staging of the disease (see chapter 1.3.4). It may cause the patient to fall and therefore it is a very disabling symptom of PD. Postural instability together with disturbance of gait may be the result of non-dopaminergic degenerations [10].

Besides motor disorders, non-motor symptoms such as depression, neuropsychological dysfunctions, cognitive deficiencies with and without dementia, olfactory deficiency, sleep disturbance, fatigue, pain and other sensory phenomena, and various autonomic disturbances can occur as well [10].

The term “parkinsonism” is used for symptoms that are common in PD plus signs and symptoms that are not characteristic of PD. Parkinsonism usually describes syndroms with known etiology, such as parkinsonism due to ischemic injuries or exposure to toxin [11].

1.3.2 Epidemiology

PD is the second most common neurodegenerative disorder after Alzheimer`s disease. Overall incidence rates for PD range between 1.5 and 22 per 100,000 person-years. In older people (above 55 or 65 years) incidence rates between 410 and 529 per 100,000 person-years are reported. In the US alone, these numbers predict an approximately 59,000 new cases per year in individuals with an age of 65 years or above. The incidence of PD seems to be higher in men than in women with a ratio between 1.46 and 1.49. Overall prevalence of PD range from 167 to 5,703 per 100,000. A review estimated the PD prevalence among people 65 years or older at 950 per 100,000, which is equal to 349,000 affected individuals in the US alone. Worldwide the prevalence of PD in people above age 50 was estimated

between 4.1 and 4.6 million in 2005. By the year 2030 this number was projected to increase to a prevalence between 8.7 and 9.3 million. Patients with PD have an approximately two-fold increased mortality rate compared to the general population. [11]

1.3.3 Diagnosis of PD

PD is diagnosed entirely on clinical grounds. Various diagnostic criteria for PD are available but the United Kingdom Parkinson's Disease Society Brain Bank diagnostic criteria are generally accepted as best clinical practice. These diagnostic criteria include following three steps: [10]

- Step 1: Diagnosis of parkinsonian syndrom
  Presence of bradykinesia and at least one of the following features:
  - Muscular rigidity
  - Rest tremor (4 to 6 Hz)
  - Postural instability which is not caused by primary visual, vestibular, cerebellar or proprioceptive dysfunction

- Step 2: Exclusion criteria for PD
  History of repeated strokes, history of repeated head injury, history of definite encephalitis, strictly unilateral features after 3 years, more than one affected relative and cerebellar signs are just a few examples for exclusion criteria for PD.

- Step 3: Supportive prospective positive criteria for PD
  Three or more of the following features are required for diagnosis of definite PD:
  - Unilateral onset
  - Rest tremor present
  - Progressive disorder
  - Persistent asymmetry affecting side of onset most

- Excellent response (70 to 100%) to levodopa (see chapter 1.3.6)
- Severe levodopa-induced chorea
- Levodopa response for 5 years or more
- Clinical course of 10 years or more

Although these criteria reduced misdiagnosis of PD substantially, it does not guarantee complete accuracy. Many core symptoms of PD also occur in other diseases whereby diagnosis of PD becomes very difficult [10].

1.3.4 PD evaluation scales

A useful rating scale for PD has to meet following criteria: validity, reliability, and responsiveness. A wide variety of assessment tools for PD are available, which evaluate impairment, disability, or both. This work focus on the Hoehn and Yahr Staging Scale (HY scale) because it is a well recognized scale, and the Unified Parkinson's Disease Rating Scale (UPDRS) because it is more comprehensive than the Hoehn and Yahr Staging Scale [10], [12].

Hoehn and Yahr Staging Scale

The HY scale is a widely used clinical rating scale for PD. The advantages of this scale are that it is simple and easy to apply. The HY scale is based on the two-fold concept that the severity of PD relates to bilateral motor involvement and compromised balance/gait. The original scale consists of five stages, whereby stage 1 describes patients with only unilateral involvement, stage 2 characterizes bilateral or midline involvement without impairement of balance, stage 3 includes postural instability, stage 4 further includes loss of physical independence, and stage 5 describes patients who are wheelchair- or bed-bounded. During the 1990`s 0.5 increments were introduced to the original HY scale which lead to the development of the modified Hoehn and Yahr scale, a widely used rating scale for PD. Table 1 shows all stages of the modified HY scale. The primary index of

disease severity in the HY scale is postural instability and therefore it does not capture all impairments or disability from other motor features of PD. Further it does not give any information about nonmotor problems in PD. These are the main weaknesses of the HY scale [13].

Table 1: Modified Hoehn and Yahr Scale ([13] ,p.1021).

Stage	Characteristics
1,0	Unilateral involvement only
1,5	Unilateral and axial involvement
2,0	Bilateral involvement without impairment of balance
2,5	Mild bilateral disease with recovery on pull test
3,0	Mild to moderate bilateral disease; some postural instability; physically independent
4,0	Severe disability; still able to walk or stand unassisted
5,0	Wheelchair bound or bedridden unless aided

Unified Parkinson's Disease Rating Scale (UPDRS)

Prior to the development of the UPDRS many different rating scales for PD (Webster, Columbia, King`s College, New York University Parkinson's Disease Scale, etc.) were used, whereby it was very difficult to make comparative assessments. The UPDRS is the most widely used clinical rating scale for PD and clinimetric analyses provided its scientific and clinical credibility. The scale is divided into following four components: [14]

- Part I: Mentation, behavior and mood
- Part II: Activities of daily living
- Part III: Motor examination
- Part IV: Complications

For detailed description of the UPDRS see appendix J. Although this scale is very comprehensive (it includes 42 questions), some screening questions on several

important non-motor aspects of PD are missing. This is the main weakness of the UPDRS [14].

1.3.5 Biochemical reason for PD

PD is caused by a destruction of certain nerve cells that lie in the brains stem's sustantia nigra (pars compacta). These neurons normally help to control motion by releasing the neurotransmitter dopamine into the striatum [10].
To understand the role of dopamine it is necessary to understand the structure and function of the basal ganglia. The basal ganglia is a collection of nuclear masses deep in the brain beneath the cerebral cortex surrounding the thalamus and hypothalamus. They are crucial for modulating and facilitating various motor and cognitive programs. Following structures of the basal ganglia are important in controlling movement: the striatum (which is divided into the caudate nucleus and the putamen), the globus pallidus, the subthalamic nucleus, and the substantia nigra (pars compacta and pars reticulata). Diseases of the basal ganglia lead to a vast number of movement abnormalities ranging from hypokinesia to hyperkinesia. Most afferents to the striatum come from the cerebral cortex. This corticostriatal pathway uses glutamic acid as its neurotransmitter. The most abundant typ of neuron in the caudate and putamen is the medium spiny neuron, which uses γ–aminobutyric acid (GABA). These neurons project to the major output regions of the basal ganglia. All regions of the striatum, which receive input from the cortex, send outputs to the lateral globus pallidus (LGP), the medial globus pallidus (MGP), and the substantia nigra pars reticulata (SNR). The LGP sends inhibitory projections to the MPG and the subthalamic nucleus (STN), which also receives a direct excitatory projection from the motor cortex. The STN, in turn, sends excitatory glutamatergic projection to the LGP and additional to the MPG and SNR. The MPG and SNR, which are the main output nuclei of the basal ganglia, send major inhibitory GABAergic projections to the ventral tier nuclei of the thalamus. The excitatory thalamocortical pathway uses glutamate as its neurotransmitter and function as feedback circle between the cortical regions and the related parts of the

basal ganglia [15]. Figure 6 illustrates the structure and function of the basal ganglia.

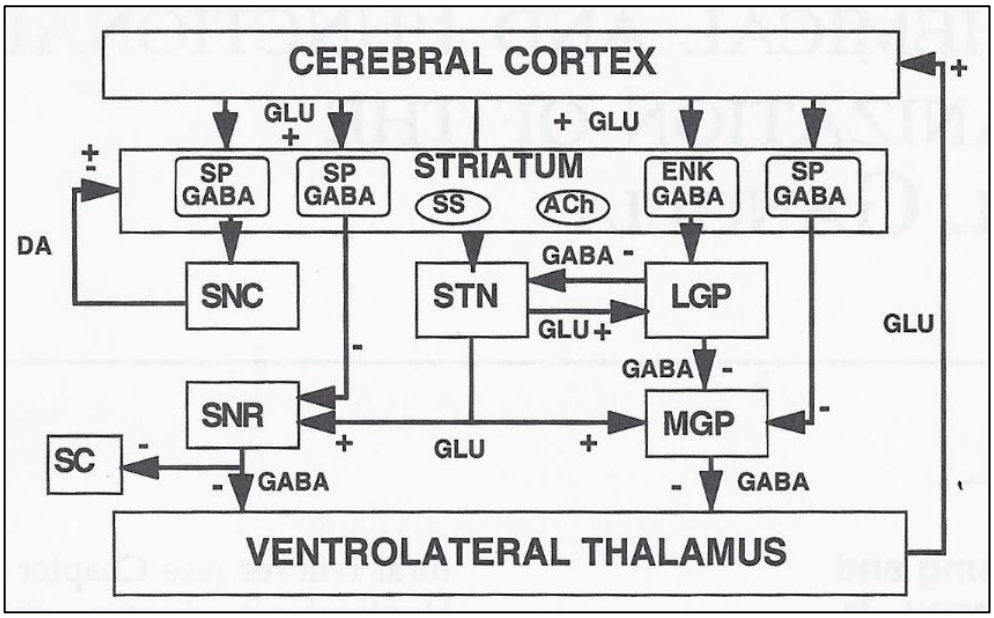


Figure 6: Simplified diagram of the basal ganglia pathways involved in motor function. Large letters indicate the nuclei; small letters indicate the neurotransmitters and neuromodulators used by the pathways. Arrowheads indicate the direction of impulse flow. Plus and minus signs indicate the excitatory or inhibitory nature of the neurotransmitter of the pathway. The dopamine pathway from substantia nigra pars compacta to the striatum excites striatal substance P output cells and inhibits enkephalin output cells. ACh=acetylcholine; DA=dopamine; ENK=enkephalin; GABA=γ-aminobutyric acid; GLU=glutamate; LGP= lateral globus pallidus; MGP=medial globus pallidus; SC=superior colliculus; SNC=substantia nigra pars compacta; SNR=substantia nigra pars reticulata; SP=substance P; SS=somatostatin; STN=subthalamic nucleus ([15], p. 2).

Cortical inputs excite two separate but parallel striatal output pathways: [15]

- In the first pathway, which is called the direct motor pathway, cortical inputs excite striatal GABAergic neurons. These neurons in turn project to and inhibit the MGP and SNR, which project to the thalamus. When a certain motor program is selected, the appropriate thalamic neurons are disinhibited, whereby the motor program can be facilitated. The striatal neurons subserving this pathway bear dopamine D1 receptors on their surfaces. These neurons therefore are excited by dopaminergic input from the substantia nigra pars compacta (SNC). Figure 7 shows the direct motor pathway. Difficulties in facilating and/or maintaining motor programs in PD patients are caused by a

loss of dopaminergic input to this pathway. This fact lead to bradykinesia, one of the main clinical motor disorders in PD [15].

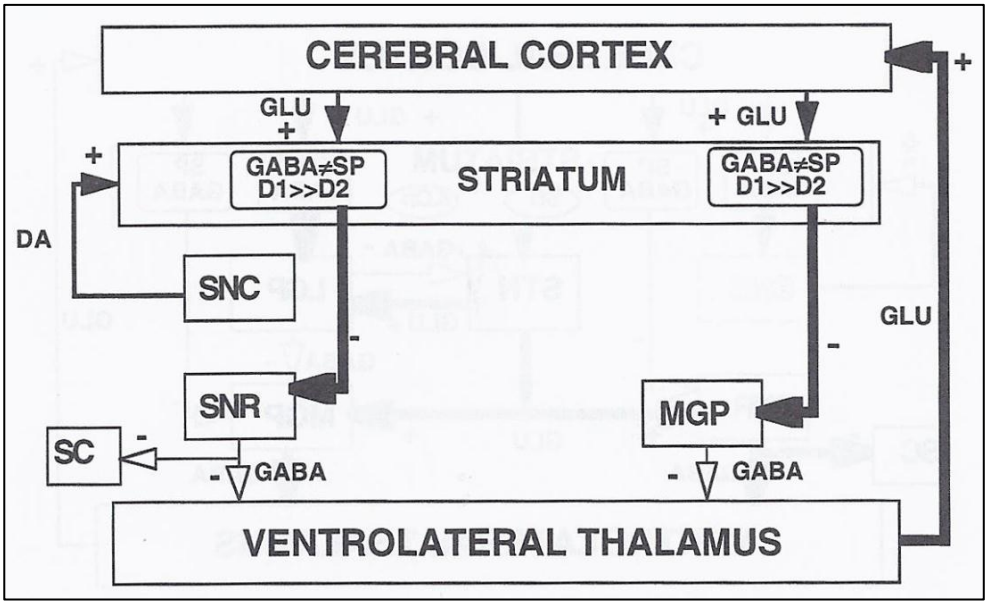


Figure 7: The direct motor circuit through the basal ganglia. Activated pathways are shown large; inhibited pathways are shown smaller. Plus and minus signs indicate the excitatory or inhibitory nature of the neurotransmitter of the pathway. In this pathway excitatory cortical output stimulates the striatal GABA/SP neurons that project to SNR and MPG. The SNR and MPG are inhibited, and the ventrolateral thalamus is released (disinhibited) from the tonic inhibition it received from SNR and MPG. The thalamus is therefore free to provide excitatory feedback to the cortex. This pathway is used to sustain an ongoing pattern of motor behavior. It becomes dysfunctional in PD, causing slowness of movement and inability to sustain an effort. DA=dopamine; GABA=γ-aminobutyric acid; GLU=glutamate; MGP=medial globus pallidus; SC=superior colliculus; SNC=substantia nigra pars compacta; SNR=substantia nigra pars reticulata; SP=substance P ([15], p. 7).

- In the second pathway (Figure 8), which is called the indirect pathway excitatory cortical output stimulates GABAergic inhibitory outputs to the LGP, which in turn inhibit the STN, MGP, and SNR. STN are excitatory and project to MGP and SNR, whereby the activity of certain MGP and SNR neurons are increased. These neurons inhibit the thalamic neurons that would otherwise facilitate unwanted motor programs. The striatal neurons subserving this pathway bear dopamine D2 receptors. These neurons therefore are inhibited by dopaminergic input from the SNC. In people with PD decreased inhibitation of the indirect pathway causes excessive subthalamic activity that results in excessive suppression of unwanted movements [15].

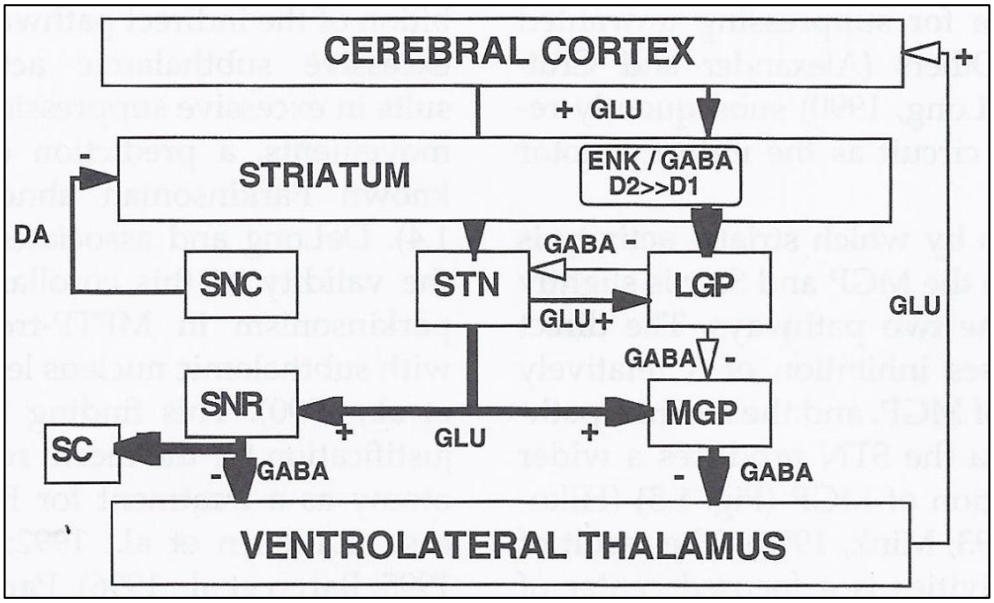


Figure 8: The indirect motor circuit through the basal ganglia. In this pathway excitatory cortical output stimulates striatal ENK and GABA neurons that project to LPG. The LPG is inhibited, so the STN is disinhibited. The excitatory STN drives the SNR and MPG to inhibit the thalamus. The STN can also be activated directly by the cortex. Plus and minus signs indicate the excitatory or inhibitory nature of the neurotransmitter of the pathway. This pathway is used to suppress inappropriate motor behaviors. It becomes hyperactive in PD, leading to inability to switch to new motor behaviors (akinesia). DA=dopamine; ENK=enkephalin; GABA=γ-aminobutyric acid; GLU=glutamate; LGP=lateral globus pallidus; MGP=medial globus pallidus; SC=superior colliculus; SNC=substantia nigra pars compacta; SNR=substantia nigra pars reticulata; STN=subthalamic nucleus ([15], p. 7).

The way how striatal activity is transmitted to the MGP and SNR is slightly different in both pathways. The direct pathway produces inhibition of a relatively small region of MPG. The indirect pathway causes a wider area of excitation of MPG. In the thalamus these two activities lead to a disinhibited center that facilitates desired activity with a surrounding periphery where undesired activites are suppressed. [15] The dopamine pathway from the substantia nigra pars compacta to the striatum modulates basal ganglia function by controlling the response of caudate and putamen neurons to cortical inputs [15]. Figure 9 shows the basal ganglia-thalamocortical circuitry for healthy people and people with PD (without functioning of the dopamine pathway).

Besides the pathological hallmark of a dopaminergic neuronal loss within the substantia nigra pars compacta, the presence of Lewy bodies is a second hallmark of PD. Lewy bodies are neuronal inclusions which appear in the area of neuronal degeneration [10].

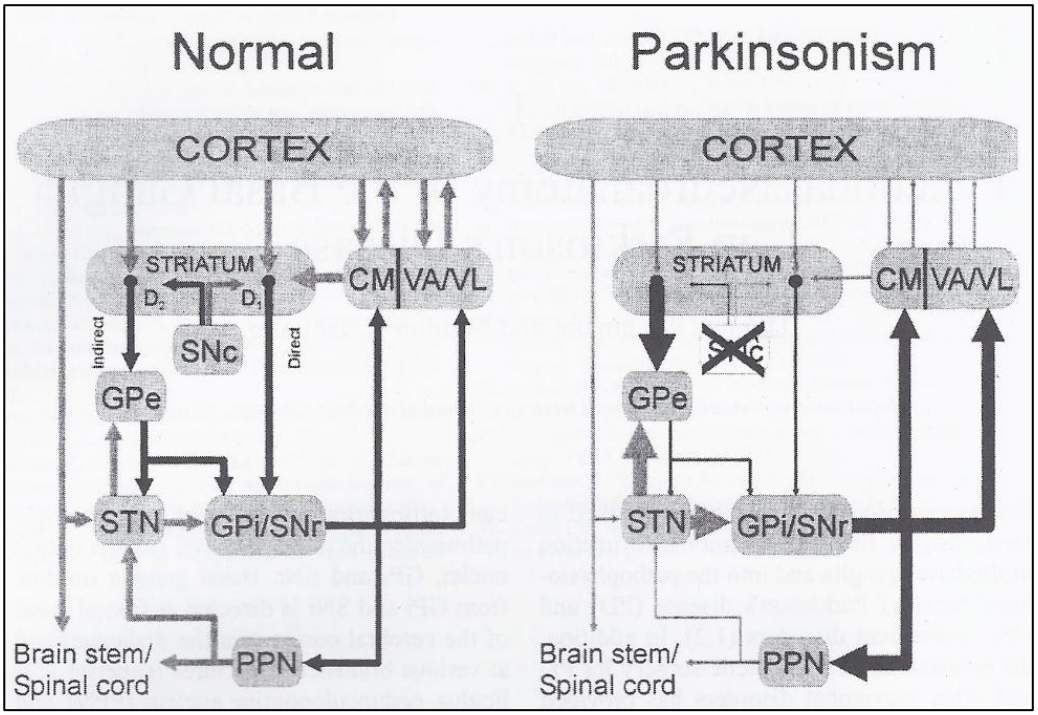


Figure 9: Simplified schematic diagram of the basal ganglia-thalamocortical circuitry under normal and parkinsonian conditions. Inhibitory connections (solid arrows) and excitatory connections (open arrows) are shown. The principal input nuclei of the basal ganglia, the striatum, and the subthalamic nucleus (STN) are connected to the output nuclei, the internal segment of the globus pallidus (GPi, also known as medial globus pallidus) and the substantia nigra pars reticulata (SNr). Basal ganglia output is directed at several thalamic nuclei (the ventroanterior (VA)/ventrolateral (VL) nucleus and the centromedian (CM)) and at brainstem nuclei (the pedunculopontine nucleus (PPN) and others). In addition to the changes in the rate of neuronal discharge (shown here as changes in the width of the connection arrows), there are prominent alterations in discharge patterns. GPe=external segment of the globus pallidus (also known as lateral globus pallidus); SNc=substantia nigra pars compacta ([16], p.10).

It is still unkown how the various neurons of the substantia nigra pars compacta become injured. As a part of normal aging about four percent of our original complement of dopamine-producing neurons disappears during each decade of adulthood. Symptoms of PD occur after approximately 70 percent of the neurons have been destroyed [8].

Although the etiology of PD is not well understood, it is likely to involve both genetic and environmental factors. Eleven genes and an additional three genetic loci have already been associated with PD. Smoking, coffee and uric acid seems to be associated with lower PD risk. Some pesticides may increase the risk for PD but further research in this area is needed to identify specific compounds that may play a causal role [11].

1.3.6 Treatment of PD

Over the last decades tremendous progress has been made in the development of treatments for PD. In general, therapies for PD can be divided into drug treatments and surgery treatments [10], [17].

Drug treatment of PD

Drugs for PD can be categorized into following subgroups: [17]

- Drugs that replace dopamine (Levodopa therapy)
- Dopamine agonists
- Drugs that prevent the breakdown of dopamine/ levodopa (COMT inhibitors)
- Other antiparkinsonian medication including anticholinergics and amantadine

The discovery of levodopa-replacement therapy was one of the most important events in the history of therapy for PD. It is extremly useful in treating the major motor symptoms of PD. Dopamine itself is not orally active, is rapidly broken down and is unable to cross the blood-brain barrier. However, levodopa is an amino acid, which is orally active and able to cross the blood-brain barrier. In the brain levodopa is converted to dopamine by the enzyme dopa-decarboxylase. Levodopa therapy is the most effective treatment for PD since more than 30 years. It has increased the quality of life of virtually all PD patients. The main disadvantage of levodopa is that it does not stop the neurodegenerative process. Nonmotor symptoms such as dementia or psychiatric disturbances and motor complications such as dyskinesia and motor fluctuations often appear over time which are further disadvantages of the levodopa therapy [10], [17].

The occurrence of complications after chronic levodopa therapy led to the investigation of new therapeutic strategies for the treatment of PD. One of these strategies is the use of dopamine agonists as an adjunct to levodopa. Due to dopamine agonists it is possible to reduce levodopa-induced motor complications by lowering the dose of levodopa. Dopamine agonists act directly on pre- and

postsynaptic dopamine receptors and a conversion to dopamine is therefore not necessary. Dopamine agonists have a longer half-life than levodopa which results in a more continuous dopamine receptor stimulation. Further advantages of dopamine agonists are that they are independent of the degenerating neurons and that no free radicals or induction of oxidative stress are generated. Neuropsychiatric complications, sleep disturbance and postural hypotension are the main side effects wich can occur due to the use of dopamine agonists. Bromocriptine, pergolide, pramixpexole, ropinirole and cabergoline are some of the common oral used dopamine agonists. Apomorphine is an additional dopamine agonist but it can only be administered by subcutaneous injection [10], [17].

Catechol-*O*-methyltransferase (COMT) is an enzyme that is responsible for the central and peripheral metabolism of levodopa. Normally it converts much of the ingested levodopa dose to 3-*O*-methyldopa, which is a non-toxic metabolite. By inhibiting COMT, an increased amount of levodopa can enter the brain and a lower doses of levodopa can therefore be used without any loss of treatment benefits. Tolacapone and entacapone are the most common COMT inhibitors, whereby tolacapone has been suspended in Canada and the European Union because of reports of hepatic failure [10], [17].

There are some more antiparkinsonian medications available. Anticholinergics and amantadine are two of them. Anticholinergics block interstriatal cholinergic transmission which helps to restore the balance between the cholinergic and dopaminergic systems. Anticholinergic drugs are more effective at treating tremor than treating rigidity or akinesia. Neuropsychiatric side effects and parasympathomimetic side effects are the main disadvantages of anticholinergics. Amantadine is a noncompetitive antagonist of the *N*-Methyl-D-aspartate receptor and it can increase dopamine release and inhibit dopamine uptake. It improves all cardinal symptoms of PD, especially dyskinesias. The incidence of significant side effects is low if amantadine is administered in a low dosis (200 to 300 mg/d) [10], [17].

Surgical treatment of PD

Ablative surgery or the implantation of a deep brain simulation (DBS) electrode are the two main options for surgerical treatments of PD. The target sites for surgical treatments of PD are the thalamus, globus pallidus internal segment (GPi) and subthalamic nucleus. Each site may be destroyed (thalamotomy, pallidotomy and subthalamotomy) or stimulated (thalamic DBS, globus pallidal DBS and subthalamic nucleus DBS). Each surgery can be performed unilaterally or bilaterally. Thalamotomy is an effective treatment for tremor-predominant PD. However, most physicians prefer GPi and STN as targets for the treatment of PD whereby thalamotomy has become increasingly rare. Pallidotomy is a safe and effective procedure for the amelioration of medication-related dyskinesia, as well as rigidity, bradykinesia, and tremor. In bilateral pallidotomy an increased incidence of speech, swollowing, and behavioral disturbance have been reported, whereby this procedure cannot be recommended. Subthalamotomy leads to an inprovement of all cardinal signs of PD on the side contralateral to the surgery. Bilateral subthalamotomy improves axial motor features and reduces daily levodopa doses, and dyskinesia. Hemichorea/ballism is the most serious complication which can occur due to subthalamic lesioning. Thalamic DBS is for patients with disabling tremor-dominant PD with stable or very slow progressive akinesia. It is an effective and safe treament for tremor in PD but in patients with more progressive PD, either globus pallidal DBS or subthalamic nucleus DBS may be more appropriate. Globus pallidal DBS is an effective treatment for PD, although the ideal target remains uncertain. Subthalamic nucleus DBS is used in patients with moderate to advanced PD and its benefits last for several years. The advantages of DBS are that bilateral surgeries are possible without increased risk of complications and that the procedure is reversible because no permanent lesion is created. The disadvantages are that DBS are very expensive compared to ablative surgeries, batteries have to be replaced after three to seven years, and a regular follow-up and reprogramming is necessary [10], [17].

1.4 Stroke

The word ‘stroke’ was originally short for ‘stroke of apoplexy’ and ‘apoplexy’ is derived from the Greek word ‘apoplexia’, which means a sudden loss of feeling and motion, as if struck by a thunderbolt. A stroke is characterised by an acute loss of focal brain function lasting more than 24 hours or leading to death. There are two main syndromes, which lead to a stroke: either hemorrhage into or over the brain substance (=hemorrhagic stroke) or inadequate blood supply to a part of the brain (=ischemic stroke) can cause a stroke. Approximately 80% of all strokes are ischemic strokes. Hemorrhagic strokes account for the remaining 20%. After coronary heart disease and cancer, stroke is the third leading cause of mortality in the United States. The incidence rate of first-ever stroke in the Caucasian populations is approximately 200 per 100,000 per year. Stroke incidence rate increases with age. Figure 10 shows the context between stroke incidence rate and age among 10 different communities. The prevalence of stroke is about 1% of the population but depends on the age and gender structure of the population. In women and men with an age between 65 and 74 years, the prevalence of a stroke is 25 and 50 per 1,000 respectively [18], [19].

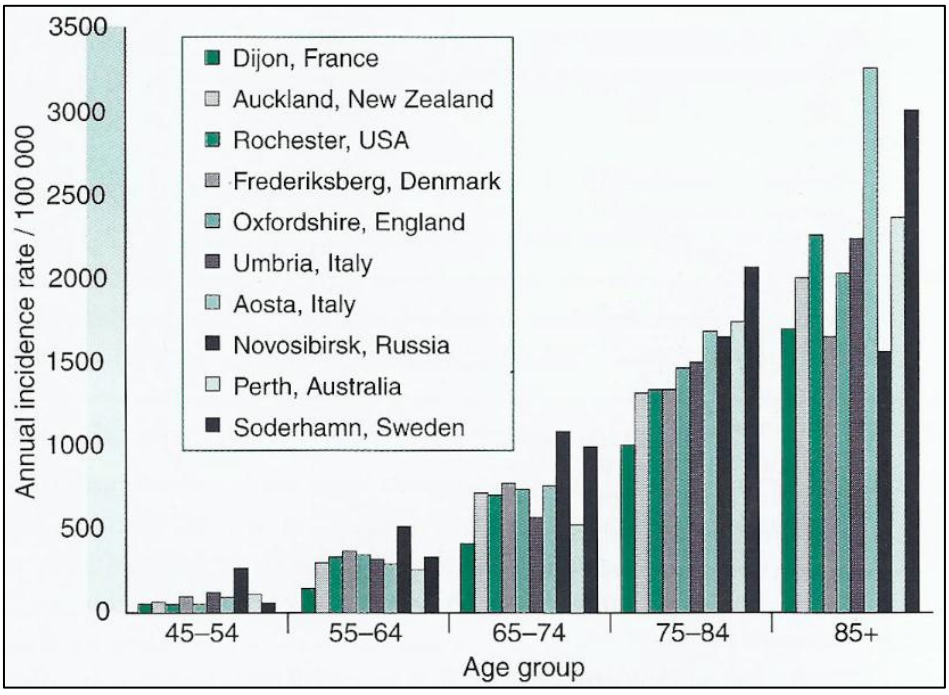


Figure 10: Incidence of stroke (ischemic and hemorrhagic combined) among 10 different communities in age groups 45 years and older ([18], p.9).

There are several known risk factors, which increase the likelyhood of a stroke. Hypertension, cardiac disease, atrial fibrillation, diabetes mellitus, cigarette smoking, alcohol abuse and hyperlipidemia are modifiable risk factors. Nonmodifiable risk factors include age, gender, race and heredity. The likelyhood to suffer a stroke is higher in elderely people, men and black persons compared to young generations, women and white persons [19].

1.4.1 Pathogenesis and pathology of stroke

The two internal carotid and two vertebral arteries serve to supply the brain with blood. These arteries anastomose at the base of the brain and form the circle of Willis, which is shown in Figure 11. The vertebrobasilar arterial system supplies the posterior third of the brain and the carotid artery system supplies the anterior two-thirds of the brain [19].

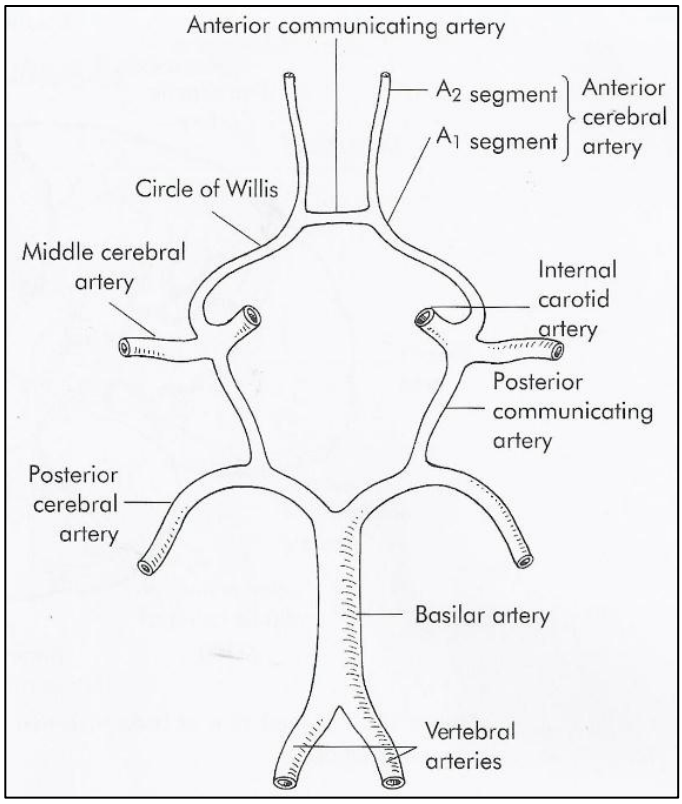


Figure 11: Circle of Willis and cerebral circulation ([19], p. 7).

Depending on the cause of the stroke, there are several different stroke subtypes. The major subgroups incluse embolic stroke, thrombolic stroke and hemorrhagic stroke. An ischemic stroke, the most common form of stroke, is caused by a cessation of cerebral blood flow, which leads to tissue anoxia. Such a cessation of cerebral blood flow can be caused by an embolus or a thrombus. A cerebral embolic stroke is the most common form of ischemic stroke, and it is usually characterized by an abrupt onset. There are many different sources (cardiac, vascular, etc) for an emboli. However, most embolic strokes of known cause are the result of a cardiac emboli. The source of about 40% of embolic strokes is still unknown. Most causes of thrombotic strokes are related to the development of abnormalities in the arterial vessel wall. Atherosclerosis, arteritis, dissections, and external compression of the vessels are some examples for causes which can lead to thrombotic stroke. In many patients thrombosis and embolism are both present. Most causes of hemorrhagic strokes include deep hypertensive intracerebral hemorrhages, ruptured saccular aneurysms, bleeding from an arteriovenous malformation, and spontaneous lobar hemorrhages. The difference between an ischemic stroke and a transient ischemic attack (TIA) is that a TIA is a reversible defect because no cerebral infarction ensues. By definition, the defect of TIAs must resolve within 24 hours. TIA can be caused by an embolus, a thrombus or it could also be the result from a cerebral vasospasm [19].

1.4.2 Gait dysfunction in stroke

Post-stroke patients walk is characterized by synergistic mass patterns of the affected lower limb rather than selective control of individual joint movements. These synergistic patterns include quadriceps and gluteus maximus contractions cause a mass extension pattern during stance phase, and hip flexors, knee flexors and ankle dorsiflexors contractions cause a mass flexion pattern during swing phase. Post-stroke patient show more co-contractions of agonist and antagonist muscles at the ankle and knee joints during stance phase, which may lead to a safer and more stable gait pattern. Balance dysfunction is also very common in post-stroke patients. It is caused by disturbance in various physiological systems,

which are responsible for postural control. In post-stroke patients poor single limb support and uncontrolled forward movement lead to gait asymmetry, which is comprised of decreased stance time and prolonged swing period of the affected side. Step length of the paretic limb has been reported to be either longer or shorter. The reason for this fact is still unknown. Further, post-stroke patients gait is characterized by a slower walking speed, shorter stride length and cycle duration, and a longer duration of double-limb support. Inefficient energy expenditure, falls, abnormal joint loading, joint damage, deformity and pain are some negative effects, which may be caused by these asymmetries. Temporal symmetry ratio (TSR) and step length ratio (SLR) are two ways to quantify these asymmetries. TSR and SLR can be calculated with following equations [5]:

$$TSR = \frac{paretic\ swing\ time}{non{-}paretic\ swing\ time} \quad \text{or} \quad TSR = \frac{paretic\ stance\ time}{non{-}paretic\ stance\ time}$$

$$SLR = \frac{paretic\ step\ length}{non - paretic\ step\ length}$$

Dysfunctions of gait and balance in post-stroke patients increase their risk of falls four times and their risk of hip fractures ten times compared to healthy people [5].

2 Part I: Parkinson`s disease study

2.1 Introduction

Parkinson`s disease (PD) is the second most common neurodegenerative disorder after Alzheimer`s disease. In individuals 65 years or above the median incidence rate is 160 per 100,000 person-years. The cardinal signs of PD are resting tremor, bradykinesia, rigidity and postural reflex impairment [11]. Gait disorders in PD include hypokinesia, freezing of gait, postural instability, and can lead to falls and reduced quality of life. These gait impairments are associated with lower limb muscle weakness and reduced joint range of motion (ROM) [20], [21]. Spinal mobilization of the lumbar spine is a painless and non-invasive manual therapy technique for certain patient populations. It is used to reduce pain and disability in patients with mobility deficits and back pain. Spinal mobilization can also be used to improve spine and hip mobility [22]. However, spinal mobilization is not a prescribed therapy for people living with PD. The purpose of this study was to compare gait parameters of people with PD before and after one session of a spinal mobilization therapy. The hypothesis was that people with PD will benefit from the spinal mobilization therapy in terms of improved gait parameters.

2.2 Material and methods

17 subjects (mean BMI 25.6±3.5) with a modified Hoehn and Yahr stage between 2 and 3 and 13 age-matched healthy subjects (mean BMI 25.6±3.9) were enrolled in this study. The data from these groups (PD group and normal group) represent a subcomponent of a larger investigation. The study was approved by the Florida Gulf Coast University Institutional Review Board and was conducted in the Arthrex Biomechatronic lab in the U.A. Whitaker College of Engineering (Florida Gulf Coast University, Fort Myers, FL, USA).

2.2.1 Motion capturing

For data collection, 26 reflective markers were placed on bony landmarks of the legs, pelvis, and shoulders of each participant. An 8-camera OQUS 300 1.3MP infrared motion capture system (Qualisys, Gothenburg, Sweden) was used to capture kinematic data. The torso segment was defined by the shoulder markers (right and left) and the iliac crest markers (right and left). Four clustered markers on the lower back served as tracking markers for the torso. The pelvis was defined by the iliac crest markers and the greater trochanter markers. Anterior and posterior superior iliac spine markers were used for tracking the pelvis. Thighs were defined by the greater trochanter markers and the medial and lateral knee markers. The four markers in a cluster on each thigh served as tracking markers for the thighs. These marker positions were chosen according to the guidelines from Visual3D (C-motion, Germantown, MD, USA), which was also the used software for data analysis. Figure 12 illustrates the marker positions.

2.2.2 Test procedure

Participants with PD performed two tests, including normal walking and a Multi-Directional Reach Test (MDRT), before and after the spinal mobilization treatment. The participants were instructed to walk at a self-selected paced for the walking trials. Afterwards the participants were instructed to stand on both feet and reach with the outstretched arm in each direction (forward, right, left, and lean backward) as far as they felt comfortable without moving their feet or bending their knees [23]. After the initial test, each subject lay on his/her side on the therapy treatment table and received a spinal mobilization treatment performed by a licensed physical therapist certified in manual therapy and a professor at FGCU. After the final testing participants with PD were asked the following questions:

- Question 1: How do you feel now? (after mobilization)
- Question 2: Do you notice a difference in how you walk? If yes, what is the differece?
- Question 3: Do you feel any differently about your balance? If yes, what is the difference?

Normal participants only performed a walking test and did not get any treatment.

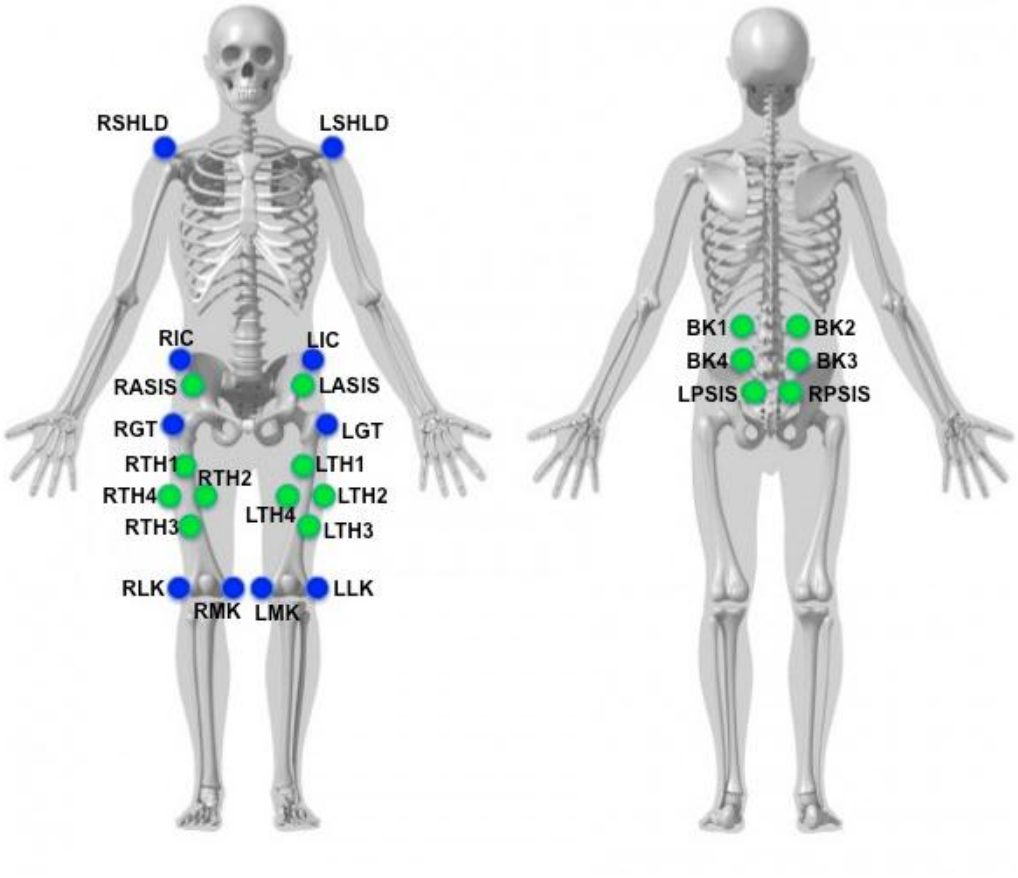


Figure 12: Schematic illustration of the marker postion. Blue markers were used for the segment definition. Green markers were only used for tracking. RSHLD/ LSHLD=right/ left shoulder, placed on the superior surface of the acromion; RIC/ LIC=right/ left iliac crest, placed on the superior iliac crest; RASIS/ LASIS=right/ left anterior superior iliac spine; RGT/ LGT= right/ left greater trochanter of femur; RTH1-4/ LTH1-4= right/ left thigh, four markers in a cluster placed on the thigh on each side; RLK/ LLK=right/ left lateral knee, placed on the lateral epicondyle; RMK/ LMK=right/ left medial knee, placed on the medial epicondyle; BK1-4= Back, four markers in a cluster placed on the lower back; RPSIS/ LPSIS=right/ left posterior superior iliac spine ([7], modified).

2.2.3 Spinal mobilizaion treatment

After the initial test (gait and MDRT) every participant with PD received a lumbar rotation mobilization. The purpose of this treatment is to manipulate specific lumbar segments (L1-L2 through L5-S1) into rotation. The patient is positioned side lying with the bottom leg at approximately 30 degrees of hip and knee flexion. The therapist uses one hand to stabilize a certain lumbar segment. The spine with all segments above the targeted segment is rotated but the targeted segment is

maintained in neutral. For detailed description of the treatment procedure refer to Olsen [24].

2.2.4 Data collection

For motion analysis, ROM values from the hip in reference to the pelvis, from the pelvis in space, and from the torso in reference to the pelvis were calculated for the gait and MDRT trials. ROM data for all three planes (sagittal, frontal, and trasverse) were considered in gait trials. For the MDRT analysis ROM data only in the sagittal and frontal plane were examined because this test is primary executed in these two planes. Additional recorded gait parameters include step length, step time, stance time, swing time, speed, stride length, cycle time and double limb support (DLS) time. These were also used to evaluate the effect of the intervention.

2.2.5 Statistical methods

Averages and standard deviations were computed for all values. Statistical comparison between the pre and post test was performed with SPSS Statistics software (version 11.5), using a paired Student's t-test. A one-way analysis of variance (ANOVA) and post hoc analysis with Bonferroni adjustment was used to compare PD participants data before and after the intervention with the data from the normal (healthy) participants. P-values of <0.05 were considered as statistically significant.

2.3 Results

2.3.1 Evaluation of questionnaires

Question 1 – How do you feel now?
13 (76%) subjects recognized an improvement. 10 out of these 13 subjects who recognized an improvement said that they felt looser, 1 felt more fluid, 1 felt lighter and his/her hips felt better and 1 mentioned that it was easier to do the test. 4 (24%) subjects did not recognize any change.

Question 2 – Do you notice a difference in how you walk? If yes, what is the difference?
10 (59% out of all 17 subjects, 67% of subjects who answered this question) subjects recognized an improvement. 3 out of these 10 subjects said that it was easier to walk, 1 subject recognized more bounce and more spring in steps, 1 patient felt more fluid, 1 person could swing her/his arms better and felt more comfortable, 1 subject felt more confident, 1 patient felt smoother, 1 person felt lighter and 1 felt more relaxed and more free. 5 (29%) subjects did not recognize any change. 2 (12%) subjects did not answer the question.

Question 3 - Do you feel any differently about your balance? If yes, what is the difference?
8 (47% out of all 17 subjects, 53% of subjects who answered this question) subjects recognized an improvement. 4 out of these 8 subjects mentioned that they could reach further, 2 patients said that they had a better balance, 1 person felt more balanced and 1 individual felt more flexible and more comfortable. 7 (41%) subjects reported no change. 2 (12%) subjects did not answer the question.

<u>Follow-up:</u>
If subjects recognized an improvement a follow-up survey one week after the intervention was done.
Question 1 – How do you feel now?
5 out of 13 (38%) subjects answered the follow-up question 1. 4 patients reported no difference and 1 subject mentioned that he felt a little better.

Question 2 – Do you notice a difference in how you walk? If yes, what is the difference?

4 out of 10 (40%) subjects answered this question. All 4 subject reported no change of their walk.

Question 3 - Do you feel any differently about your balance? If yes, what is the difference?

3 out of 8 (38%) subjects answered this question. 2 patients reported no change of their balance and 1 person mentioned that he felt a little different.

Summary of the questionaries:

Question 1 - general	• 76% of all subjects felt better after the spinal mobilization therapy
Question 2 - walk	• 67% of subjects who answered question 2 noticed an improvement in their walk
Question 3 - balance	• 53% of subjects who answered question 3 recognized an improvement in their balance

2.3.2 Gait analysis

Hip ROM

Figure 13 shows the averages of hip ROM for PD subjects and normal subjects in all three planes. The comparison between the pre and post PD hip data, which was done with a paired stundents t-test, showed only in the right hip ROM in the sagittal plane (flexion – extension movement) a significant difference. ROM of the right hip in the sagittal plane significantly increased ($p=0.0001$) from 33.4±6.8° to 34.9±6.4° due to the intervention. Comparison between pre/ post PD hip data and the hip data of normal (healthy) subjects was done with an analysis of variances (ANOVA) and showed a significant difference in ROM of the left hip in the sagittal ($p=0.039$) and frontal ($p=0.001$) plane. Bonforroni post hoc test only led to a significant difference in the frontal plane. In this plane pre PD subjects ROM of the left hip (8.4±2.8°) was significant smaller ($p=0.001$) than the ROM of normal subjects (11.8±1.6°). Post PD subjects ROM of the left hip in the frontal plane (8.5±2.6°)

was significant smaller (p=0.002) than the ROM of normal subjects (11.8±1.6°) as well. The post hoc test for the sagittal plane did not show any significant difference. However, a almost significant difference (p=0.052) between the pre PD ROM data of the left hip (34.6±6.8°) and normal ROM data of the left hip (40.2±5.3°) was noticed in the sagittal plane. Table 2 shows average hip ROM values for all groups.

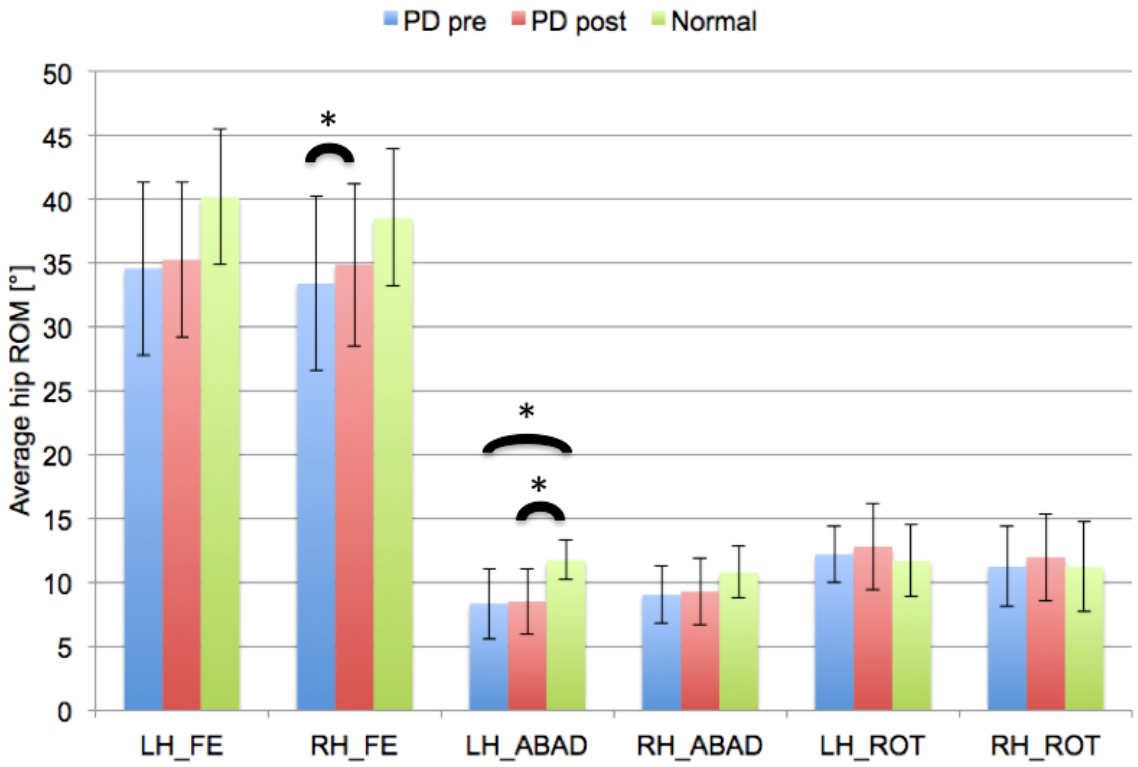


Figure 13: Averages and standard deviations in hip ROM for all three planes. The blue columns represent averages of ROM for PD subjects before the intervention, the red columns represent averages for PD subjects after the intervention and the green columns represent average for normal (healthy) subjects. The black curves with the stars mark significant differences. LH_FE/ RH_FE=left/ right hip_flexion-extension movement; LH_ABAD/ RH_ABAD=left/ right hip_abduction-adduction movement; LH_ROT/ RH_ROT=left/ right hip_rotation movement.

Table 2: Hip ROM values (averages and standard diviations) for PD subjects and normal subjects. PD pre=PD subjects before the intervention; PD post=PD subjects after the intervention; Normal= normal (healthy) subjects.

ROM	Sagittal plane		Frontal plane		Transversal plane	
	Left Hip	Right hip	Left Hip	Right hip	Left Hip	Right hip
PD pre	34.6±6.8°	33.4±6.8°	8.4±2.8°	9.1±2.2°	12.2±2.2°	11.3±3.1°
PD post	35.3±6.0°	34.9±6.4°	8.5±2.6°	9.3±2.6°	12.8±3.3°	12.0±3.4°
Normal	40.2±5.3°	38.6±5.4°	11.8±1.6°	10.8±2.0°	11.7±2.8°	11.3±3.5°

Figure 14 shows differences in average ROM between the left and right hip for PD subjects before and after the intervention. Average difference between right and left hip ROM in the sagittal plane decreased from 2.7±2.4° before the intervention to 2.5±2.3° after the intervention. In the frontal plane average differences between the right and left hip ROM increased from 1.6±1.4° to 1.9±1,7°. Differences between the right and left hip ROM in the transversal plane decreased from 2.3±1.1° to 1.9±1.6°. None of these changes were significant.

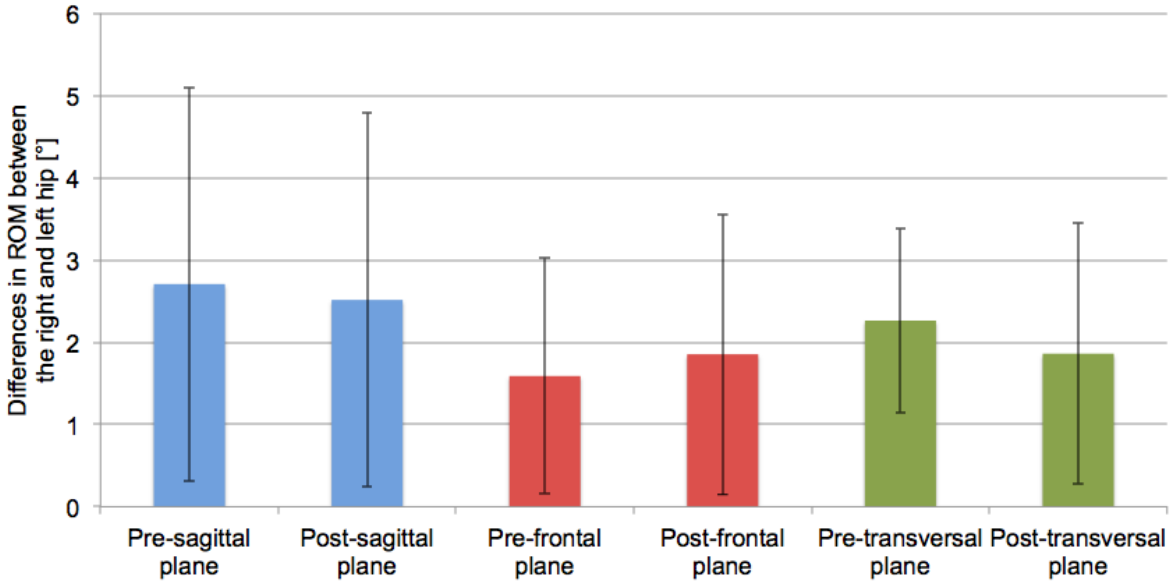


Figure 14: Difference in ROM (averages±standard deviations) between the right and left hip. Blue columns represent pre and post PD hip ROM in the sagittal plane, red columns represent pre and post PD hip ROM in the frontal plane and green columns represent PD hip ROM in the transversal plane.

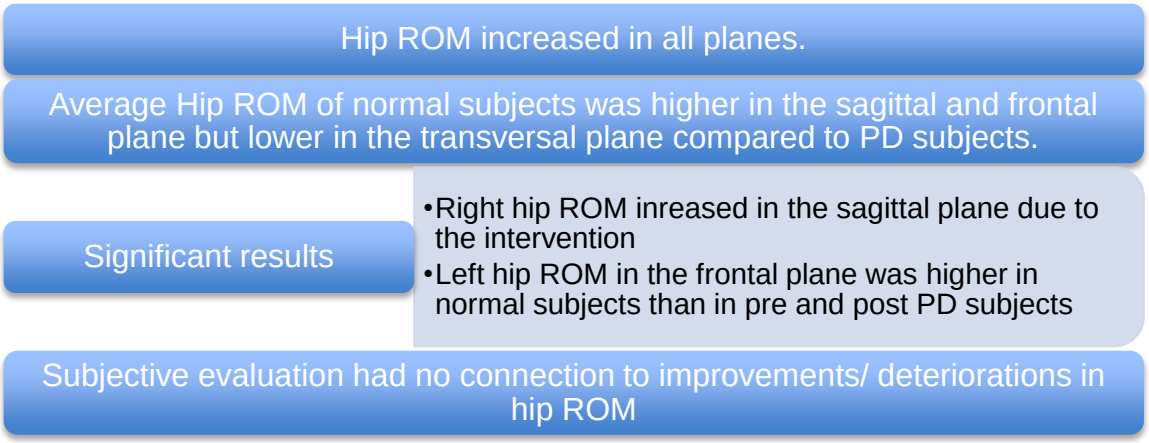
Table 3 shows the percentage change in hip ROM in the connection with the results of question two from the questionary. In the sagittal plane 60% of subjects who reported an improvement in their gait had an improvement of ROM on both hip sides. In subjects who did not report any change only 40% had an improved hip ROM on both sides. In the frontal plane hip ROM showed no clear difference between subjects who reported an improvement in their gait and subject who did not report any change. In the transversal plane the same amount of subjects (20%) who recognized an improved gait and who did not recognize any change had an improved hip ROM on both sides. However, deterioriations in hip ROM on both sides in the transversal plane occurred four times as often in subjects who did not

recognize any change (40%) compared to subject who recognized an improvement in their gait (10%). 80% of subjects who did not recognize any change had PD stage 2.

Table 3: Percentage changes in hip ROM between the pre and post test for PD patients who recognized an improvement in their gait (improvement rows) and for PD patient who did not recognize any change in their gait (no change rows). Green values represent improvements in ROM (closer to the data of normal subjects) and red values represent deteriorations of ROM. Left-sag/ Right-sag=left/ right hip ROM in the sagittal plane; Left-fro/ Right-fro=left/ right hip ROM in the frontal plane; Left-tra/ Right-tra=left/ right hip ROM in the transversal plane.

Q 2	PD-patient	PD-Stage	Left-sag	Right-sag	Left-fro	Right-fro	Left-tra	Right-tra
improvement	PD07	2	7.6%	8.1%	-29.0%	-5.8%	36.4%	29.1%
	PD11	2	-4.7%	1.7%	6.9%	22.5%	-7.5%	17.0%
	PD23	2	4.0%	8.7%	3.3%	0.4%	14.4%	-4.2%
	PD26	2	0.2%	4.0%	-3.0%	-7.5%	10.4%	1.2%
	PD20	2.5	5.7%	6.4%	24.0%	-4.5%	2.9%	-0.4%
	PD22	2.5	4.6%	2.8%	6.7%	-13.3%	19.2%	-0.4%
	PD04	3	0.7%	10.5%	20.5%	-9.2%	-15.1%	2.6%
	PD14	3	2.7%	-2.2%	1.3%	1.8%	7.9%	-5.4%
	PD21	3	-1.9%	0.2%	2.9%	7.3%	-7.2%	5.2%
	PD27	3	4.8%	9.2%	22.8%	16.4%	8.3%	-5.4%
no change	PD12	2	-0.8%	3.2%	10.5%	14.9%	-24.5%	16.4%
	PD17	2	4.8%	9.5%	-2.2%	20.2%	9.2%	25.6%
	PD18	2	-1.7%	1.5%	9.1%	9.2%	6.7%	23.6%
	PD28	2	-0.9%	0.7%	-2.0%	-3.3%	5.0%	2.3%
	PD15	3	-2.1%	1.1%	-10.0%	-2.5%	-7.1%	13.6%

Summary of the hip ROM findings:



Pelvis ROM

Figure 15 shows the averages of pelvis ROM for PD subjects and normal subjects in all three planes. The comparison between the pre and post PD pelvis ROM data showed a significant difference in the transversal plane (rotation movement). ROM of the pelvis in the transversal plane significantly decreased (p=0.024) from 8.6±2.4° before the intervention to 8.0±2.5° after the intervention. Comparison between pre/ post PD pelvis ROM data and the pelvis ROM data of normal (healthy) subjects showed no significant differences. Table 4 shows average pelvis ROM values for all groups.

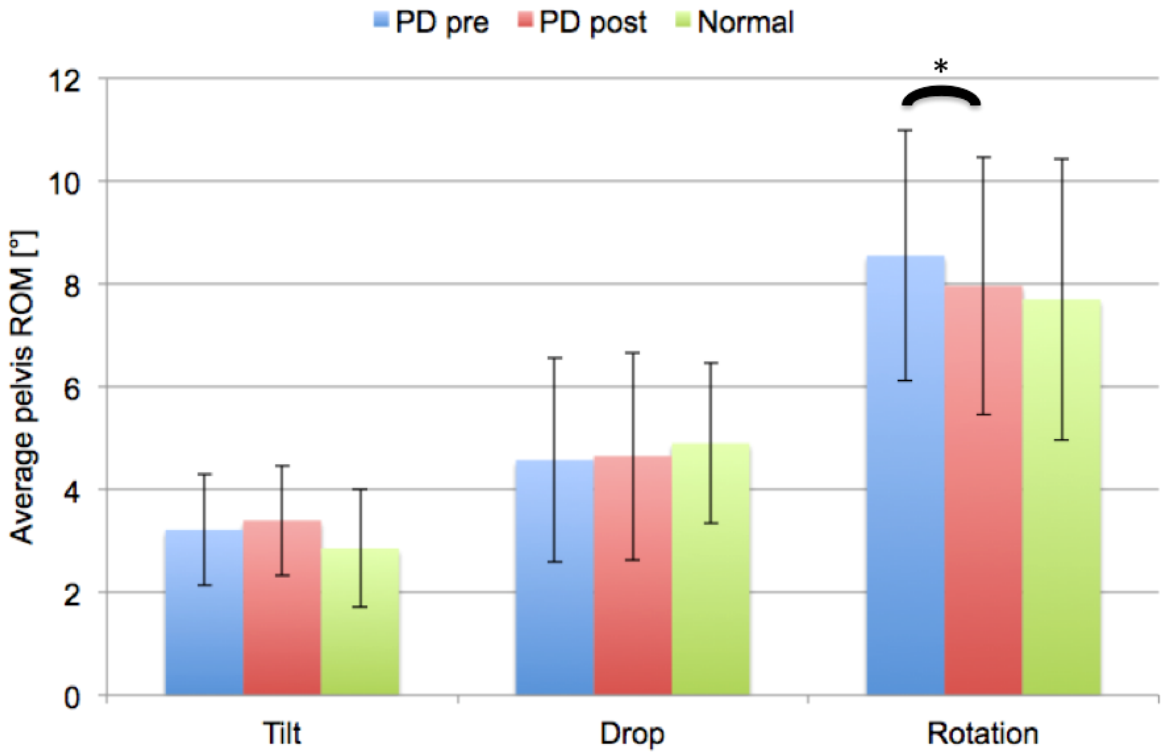


Figure 15: Averages and standard deviations in pelvis ROM for all three planes. The blue columns represent averages of ROM for PD subjects before the intervention, the red columns represent averages for PD subjects after the intervention and the green columns represent average for normal (healthy) subjects. The black curve with the star mark the significant difference between the pre and post PD ROM data in the transversal plane. Tilt=movement in the sagittal plane; Drop=movement in the frontal plane; Rotation=movement in the transversal plane.

Table 4: Pelvis ROM values (averages and standard diviations) for PD subjects and normal subjects. PD pre=PD subjects before the intervention; PD post=PD subjects after the intervention; Normal= normal (healthy) subjects.

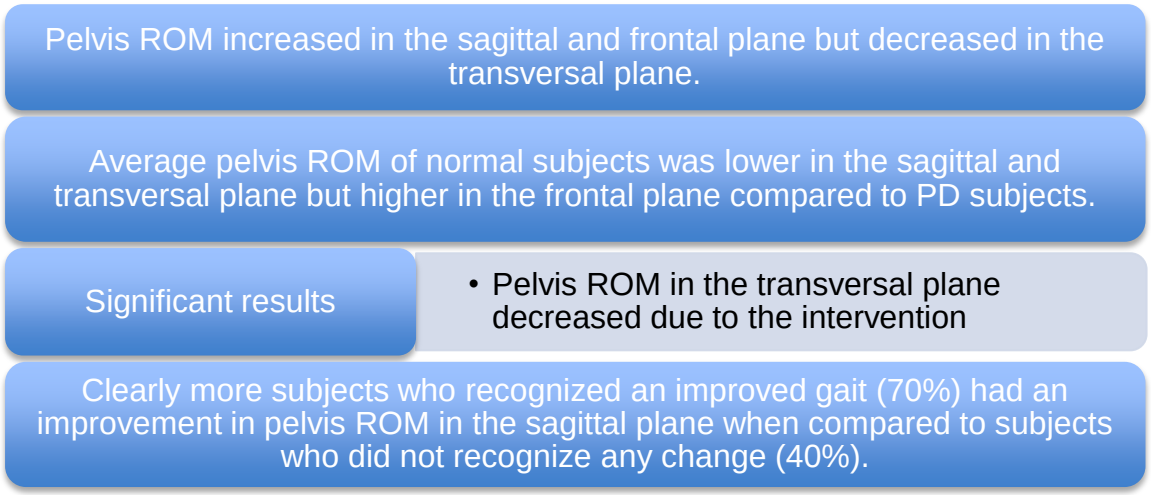
ROM	Sagittal plane	Frontal plane	Transversal plane
PD pre	3.2±1.1°	4.6±2.0°	8.6±2.4°
PD post	3.4±1.1°	4.7±2.0°	8.0±2.5°
Normal	2.9±1.1°	4.9±1.6°	7.7±2.7°

Table 5 shows the percentage change in pelvis ROM in the connection with the results of question two from the questionary. In the sagittal plane as well as in the frontal and transversal plane 70% of subjects who recognized an improvement in their gait had an improved pelvis ROM (closer to normal). 40% of subjects who did not recognize any change in their gait had an improved pelvis ROM in the sagittal plane and 60% of the same subjects had an improvement in the frontal and transversal plane.

Table 5: Percentage changes in pelvis ROM between the pre and post test for PD patients who recognized an improvement in their gait (improvement rows) and for PD patient who did not recognize any change in their gait (no change rows). Green values represent improvements in ROM (closer to the data of normal subjects) and red values represent deteriorations of ROM. Tilt=movement in the sagittal plane; Drop=movement in the frontal plane; Rotation=movement in the transversal plane.

Q 2	PD-patient	PD stage	Tilt	Drop	Rotation
improvement	PD07	2	3.2%	-55.2%	-39.3%
	PD11	2	-7.0%	19.2%	1.5%
	PD23	2	-9.3%	17.9%	-10.8%
	PD26	2	31.5%	12.4%	5.1%
	PD20	2.5	13.0%	41.2%	2.1%
	PD22	2.5	-14.7%	4.4%	-3.9%
	PD04	3	-24.4%	13.9%	-14.2%
	PD14	3	8.5%	-27.4%	-17.4%
	PD21	3	3.5%	7.2%	9.4%
	PD27	3	2.3%	13.5%	6.6%
no change	PD12	2	12.0%	2.8%	-7.8%
	PD17	2	-10.1%	2.9%	-16.7%
	PD18	2	36.8%	20.7%	-18.7%
	PD28	2	136.1%	14.8%	-19.5%
	PD15	3	-13.1%	-13.5%	-1.0%

Summary of the pelvis ROM findings:



Torso ROM

Figure 16 illustrates the averages of torso ROM for PD subjects and normal subjects in all three planes. The comparison between the pre and post PD torso ROM data as well as the comparison between pre/ post PD torso ROM data and the torso ROM data of normal subjects showed no significant differences. Torso ROM values in the sagittal plane of normal subjects were higher than torso ROM values of PD subjects in the same plane. In the frontal and transversal plane torso ROM values were lower in normal subjects than in PD subjects. Table 6 shows average torso ROM values for all groups (Pre PD, Post PD, and Normal).

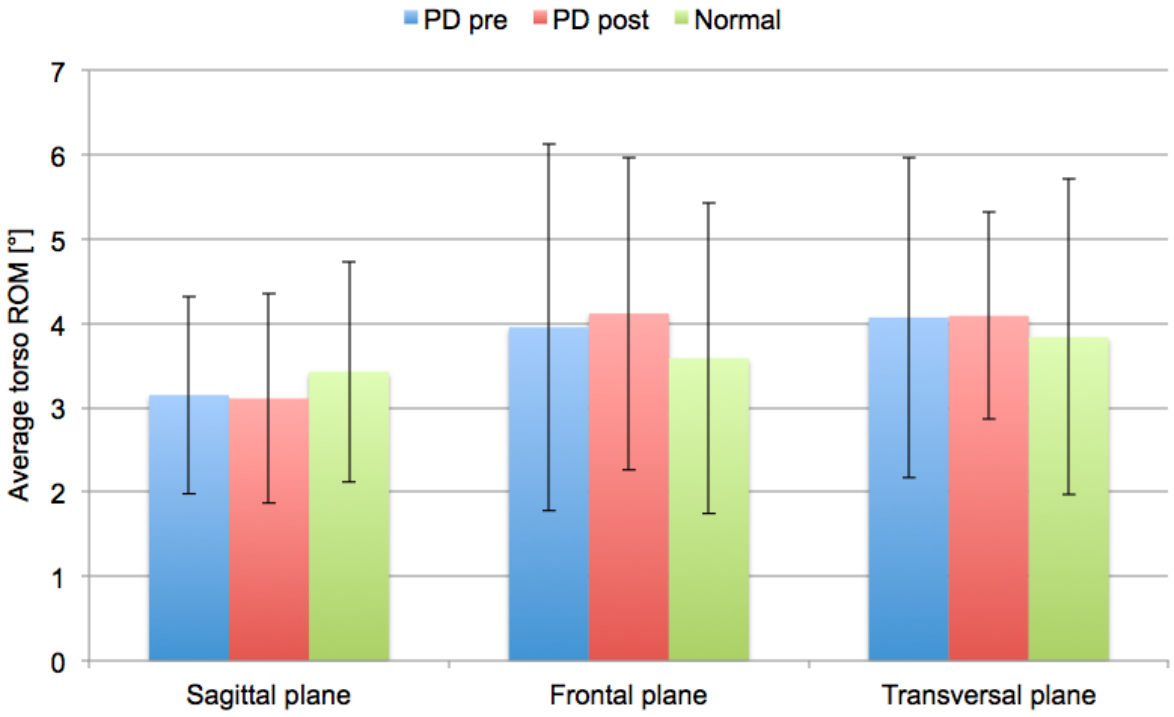


Figure 16: Averages and standard deviations in torso ROM for all three planes. The blue columns represent averages of ROM for PD subjects before the intervention, the red columns represent averages for PD subjects after the intervention and the green columns represent average for normal (healthy) subjects.

Table 6: Torso ROM values (averages and standard diviations) for PD subjects and normal subjects. PD pre=PD subjects before the intervention; PD post=PD subjects after the intervention; Normal= normal (healthy) subjects.

ROM	Sagittal plane	Frontal plane	Transverse plane
PD pre	3.2±1.2°	4.0±2.2°	4.1±1.9°
PD post	3.1±1.2°	4.1±1.9°	4.1±1.2°
Normal	3.4±1.3°	3.6±1.8°	3.8±1.9°

Table 7 shows the percentage change in torso ROM in the connection with the results of question two from the questionary. In the sagittal plane the same amount of subjects (60%) who reported an improvement in their gait and who did not recognize any change had an improvement of torso ROM (closer to average ROM of normal subjects). In the frontal plane 40% of subjects who reported an improvement and 60% of subjects who did not report any change had an improved torso ROM. In the transverse plane 50% of subjects who reported an improvement in their gait had an improvement in torso ROM after the intervention. 20% of

subjects who did not recognize any difference had an improved torso ROM in the transverse plane.

Table 7: Percentage changes in torso ROM between the pre and post test for PD patients who recognized an improvement in their gait (improvement rows) and for PD patient who did not recognize any change in their gait (no change rows). Green values represent improvements in ROM (closer to the data of normal subjects), red values represent deteriorations of ROM, and blue values represent subjects in which the pre and post value had the same difference to the average value of normal subjects.

Q 2	PD-patient	PD stage	Sagittal plane	Frontal plane	Transverse plane
improvement	PD07	2	-33.3%	-50.9%	-48.1%
	PD11	2	-19.8%	52.0%	-35.4%
	PD23	2	-25.4%	10.0%	56.0%
	PD26	2	8.6%	35.0%	21.8%
	PD20	2.5	-13.3%	87.9%	42.5%
	PD22	2.5	-24.5%	-21.8%	-13.3%
	PD04	3	-34.7%	28.0%	22.5%
	PD14	3	-15.8%	-32.2%	-47.6%
	PD21	3	40.0%	-5.1%	26.4%
	PD27	3	-11.8%	30.4%	1.3%
no change	PD12	2	-3.1%	171.9%	51.3%
	PD17	2	26.5%	-17.4%	-7.0%
	PD18	2	38.8%	-13.1%	39.8%
	PD28	2	77.4%	22.5%	-20.0%
	PD15	3	-4.7%	-11.8%	9.8%

Summary of the torso ROM findings:

Torso ROM decreased in the sagittal, increased in the frontal plane, and did not change in the transverse plane.

Average torso ROM of normal subjects was higher in the sagittal plane and lower in the frontal and transverse plane when compared to PD subjects.

There were no significant results.

Clearly more subjects who recognized an improved gait (50%) had an improvement in torso ROM in the transverse plane when compared to subjects who did not recognize any change (20%).

Additional gait parameters

Speed, stride wide and length, cycle time, step length and step time for the left and right leg, stance and swing time for the left and right leg, and DLS (= double limb support) time were additional to ROM data recorded and analyzed. All of these parameters except for swing time (no change) improved following the spinal mobilization. Figure 17 and Figure 18 shows averages gait parameter values for PD subjects (pre and post data) and normal subjects. Speed was higher in normal subjects than in PD subjects. Average stride wide of normal subjects was lower than average stride wide of PD subjects before the intervention. After the intervention average stride wide of PD subjects and normal subjects were equal. Average stride length was longer in normal subjects than in PD subjects. Average cycle time was lower in normal subjects than in PD subjects. Average step length was longer in normal subjects than in PD subjects. Average step time of the left leg was lower in normal subjects than in PD subjects before the intervention. After the spinal mobilization average step time of the left leg was equal between normal and PD subjects. Average step time of the right leg was equal between normal and PD subjects. Average stance time was shorter in normal subjects than in PD subjects before the intervention. After the intervention average stance time values between PD and normal subjects were equal. In normal and PD subjects average swing time of the left leg were the same. Average swing time of the right leg was lower in normal subjects than in PD subjects. DLS time of normal subjects was lower compared to PD subjects before the intervention but higher compared to PD subjects after the intervention. Table 8 shows averages and standard deviations of all additional gait parameters for PD subjects (pre and post data) and normal subjects.

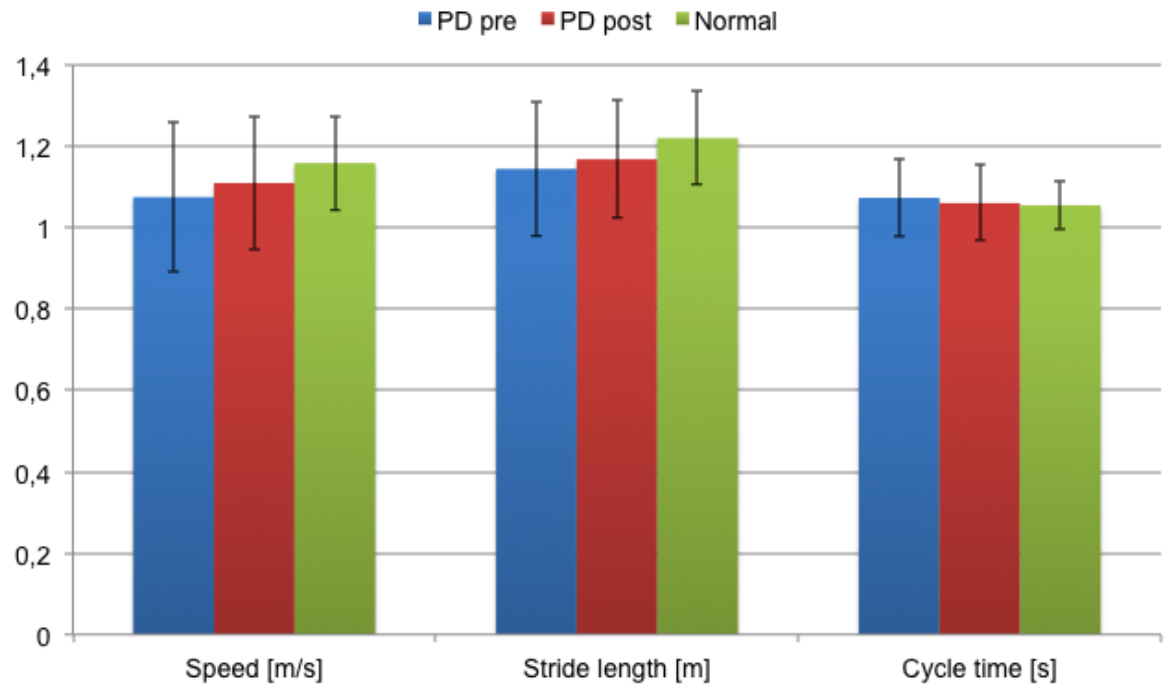


Figure 17: Averages and standard deviations for gait speed, stride length and cycle time. The blue columns represent averages for PD subjects before the intervention, the red columns represent averages for PD subjects after the intervention and the green columns represent average for normal (healthy) subjects.

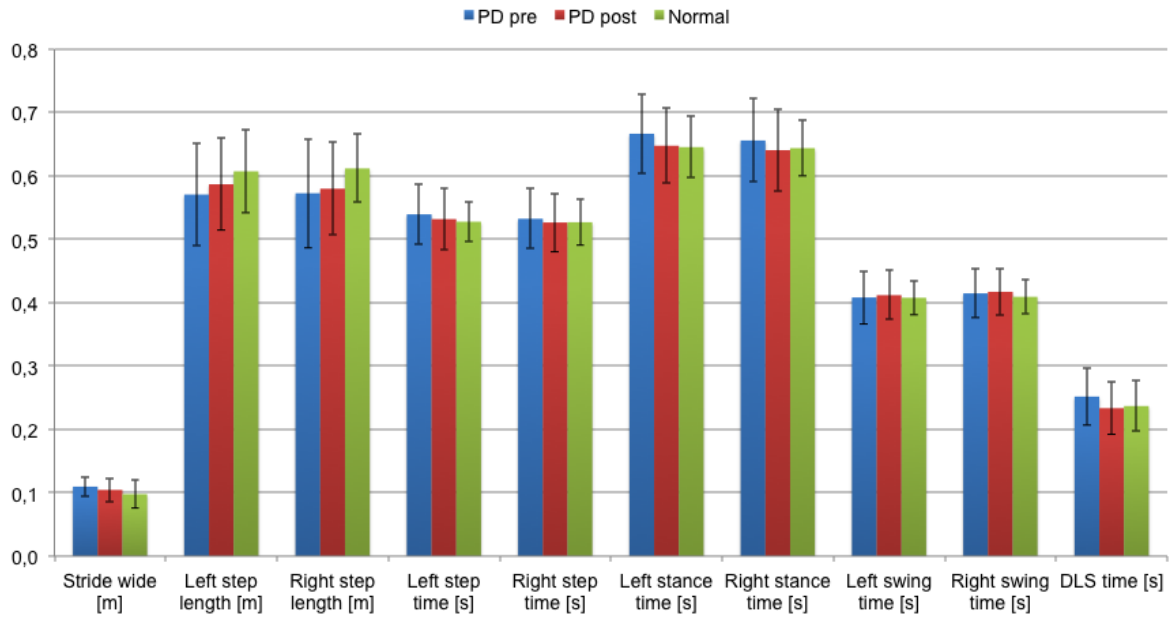


Figure 18: Averages and standard deviations for stride wide, step length, step time, stance time, swing time and DLS (= double limb support) time. The blue columns represent averages for PD subjects before the intervention, the red columns represent averages for PD subjects after the intervention and the green columns represent average for normal (healthy) subjects.

The comparison between pre PD data and post PD data (paired students t-test) led to following significant differencess:

- Speed significantly increased (p=0.025)
- Stride wide significantly decreased (p=0.01)
- Stride length significantly increased (p=0.021)
- Left step length significantly increased (p=0.006)
- Left (p=0.021) and right stance time (p=0.021) significantly decreased
- DLS time significantly decreased (p=0.011)

Comparison between pre/ post PD data and normal data (ANOVA) did not show any significant differences.

Table 8: Additional gait parameters (averages and standard diviations) for PD subjects and normal subjects. PD pre=PD subjects before the intervention; PD post=PD subjects after the intervention; Normal= normal (healthy) subjects; DLS= double limb support. Green values represent improvements (closer to the data of normal subjects), and blue values represent subjects in which the pre and post values were equal.

Gait parameters	PD pre	PD post	Normal
Speed [m/s]	1.07±0.18	1.11±0.16	1.16±0.12
Stride length [m]	1.14±0.17	1.17±0.14	1.22±0.11
Cycle time [s]	1.07±0.09	1.06±0.09	1.05±0.06
Stride wide [m]	0.11±0.02	0.10±0.02	0.10±0.02
Left step length [m]	0.57±0.08	0.59±0.07	0.61±0.07
Right step length [m]	0.57±0.09	0.58±0.07	0.61±0.05
Left step time [s]	0.54±0.05	0.53±0.05	0.53±0.03
Right step time [s]	0.53±0.05	0.53±0.05	0.53±0.04
Left stance time [s]	0.67±0.06	0.65±0.06	0.65±0.05
Right stance time [s]	0.66±0.07	0.64±0.06	0.64±0.04
Left swing time [s]	0.41±0.04	0.41±0.04	0.41±0.03
Right swing time [s]	0.42±0.04	0.42±0.04	0.41±0.03
DLS time [s]	0.25±0.05	0.23±0.04	0.24±0.04

Table 9 and **Table 10** show the percentage change of all additional gait parameters in the connection with the results of question two from the questionary. Speed

increased in 60% of subjects who recognized an improvement in their gait and in 40% of subjects who did not recognize any change. Stride wide decreased in 80% and stride length increased in 70% of subjects who reported an improved gait. In subjects who did not report any change 60% had a decreased stride wide and 60% had an increased stride length. 40% of PD subjects who recognized an improved gait and 20% of PD subjects who did not recognize any change had a decreased cycle time. Step length and step time did not show any clear differences (not more than 10%) between subjects who reported an improved gait and subjects who did not report any change. Stance time of the left foot decreased in 60% and stance time of the right foot decreased in 70% of subjects who recognized an improvement in their gait. In subjects who did not recognize any change stance time of the left foot decreased in 20% and stance time of the right foot decreased in 40%. 70% of subjects who reported an improved gait and 40% of subjects who did not report any change had a decreased DLS time. Twice (40%) as many subjects who did not recognize any change had deteriorations in swing time compared to subjects who recognized an improvement (20%).

Table 9: Percentage changes in gait speed, stride wide, stride length, cycle time, and step length (left and right) between the pre and post test for PD patients who recognized an improvement in their gait (improvement rows) and for PD patient who did not recognize any change in their gait (no change rows). Green values represent improvements, red values represent deteriorations, and blue values represent subjects in which the pre and post values were equal.

Q2	Patient	PD stage	Speed [m/s]	Stride wide [m]	Stride length [m]	Cycle time [s]	Left step length [m]	Right step length [m]
improvement	PD07	2	10.3%	2.8%	8.8%	-2.0%	11.4%	6.3%
	PD11	2	1.9%	-8.7%	0.8%	-1.6%	1.2%	0.6%
	PD23	2	1.8%	-3.4%	1.0%	0.0%	2.8%	-0.3%
	PD26	2	-1.7%	-2.6%	-0.8%	1.0%	-0.3%	-1.0%
	PD20	2.5	15.9%	5.9%	4.7%	-8.6%	0.8%	7.3%
	PD22	2.5	-3.1%	-9.1%	-1.0%	2.9%	2.3%	-4.3%
	PD04	3	8.3%	-14.9%	2.9%	-6.3%	4.3%	2.9%
	PD14	3	-2.4%	-7.6%	-1.5%	0.9%	-2.6%	0.0%
	PD21	3	0.0%	-15.0%	0.6%	0.0%	-0.2%	1.3%
	PD27	3	6.9%	-11.2%	7.4%	1.0%	9.0%	6.3%
no change	PD12	2	4.7%	-8.6%	3.6%	-1.2%	6.1%	0.3%
	PD17	2	2.2%	0.9%	3.8%	1.8%	5.0%	2.2%
	PD18	2	0.0%	8.2%	1.1%	0.9%	0.9%	0.8%
	PD28	2	-2.5%	-3.5%	-2.8%	0.0%	-1.9%	-4.1%
	PD15	3	-2.2%	-13.6%	-2.2%	0.0%	0.3%	-4.7%

Table 10: Percentage changes in step time (left and right), stance time (left and right), swing time (left and right), and DLS time between the pre and post test for PD patients who recognized an improvement in their gait (improvement rows) and for PD patient who did not recognize any change in their gait (no change rows). Green values represent improvements, red values represent deteriorations, and blue values represent subjects in which the pre and post values were equal.

Q2	Patient	PD stage	Left Step Time [s]	Right Step Time [s]	Left Stance Time [s]	Right Stance Time [s]	Left Swing Time [s]	Right Swing Time [s]	DLS Time [s]
improvement	PD07	2	-2.0%	-4.0%	-6.2%	-1.6%	0.0%	2.8%	-14.3%
	PD11	2	1.6%	-4.8%	-5.2%	-2.6%	4.3%	0.0%	-13.3%
	PD23	2	0.0%	-1.7%	-1.4%	-1.5%	0.0%	2.2%	-4.0%
	PD26	2	2.0%	0.0%	0.0%	0.0%	2.6%	2.6%	0.0%
	PD20	2.5	-11.7%	-5.4%	-8.1%	-12.0%	-11.6%	-4.9%	-12.5%
	PD22	2.5	0.0%	4.0%	-1.5%	-1.5%	11.4%	8.3%	-12.5%
	PD04	3	-5.5%	-5.5%	-8.7%	-6.1%	-4.7%	-4.5%	-16.0%
	PD14	3	0.0%	3.8%	1.5%	1.5%	0.0%	2.3%	4.2%
	PD21	3	1.6%	0.0%	0.0%	0.0%	0.0%	2.1%	3.7%
	PD27	3	0.0%	0.0%	0.0%	-1.6%	2.5%	2.5%	-9.1%
no change	PD12	2	-2.3%	-2.3%	-2.0%	-6.1%	2.9%	5.6%	-21.4%
	PD17	2	1.9%	1.8%	1.5%	0.0%	4.8%	0.0%	-8.0%
	PD18	2	0.0%	3.8%	0.0%	-1.4%	0.0%	4.8%	4.3%
	PD28	2	2.1%	0.0%	1.6%	1.6%	-2.7%	-2.7%	8.0%
	PD15	3	1.9%	-1.9%	1.6%	1.6%	-2.4%	-2.3%	10.0%

Summary of additional gait parameters findings:

All parameters except for swing time improved due to the spinal mobilization.

Significant results

- Speed increased
- Stride wide decreased
- Stride length increased
- Left step length increased
- Left and right stance time decreased
- DLS time decreased

Clearly more subjects who recognized an improved gait (at least 30% more) had an improvement in stance time and DLS time when compared to subjects who did not recognize any change.

2.3.3 Reach test analysis

All PD subjects had to perform a MDRT before and after the intervention. Figure 19 shows characteristical MDRT torso (in reference to the pelvis) angle curves in the sagittal and frontal plane. It is clearly to see where the typical MDRT movements to the front, to the right, to the left, and to the back happened. The comparison between pre and post hip ROM data (Figure 20), as well as pre and post pelvis ROM data (Figure 21), did not show any significant differences. Torso ROM values in the sagittal plane increased from 17.6±9.8° before the spinal mobilization to 20.2±10.8° after the spinal mobilization (not significant). In the frontal plane torso ROM significantly increased (p=0.015) from 10.1±6.3° before the intervention to 12.5±6.6° after the intervention (Figure 21). Table 11 shows average hip, pelvis and torso ROM values for the sagittal and frontal plane.

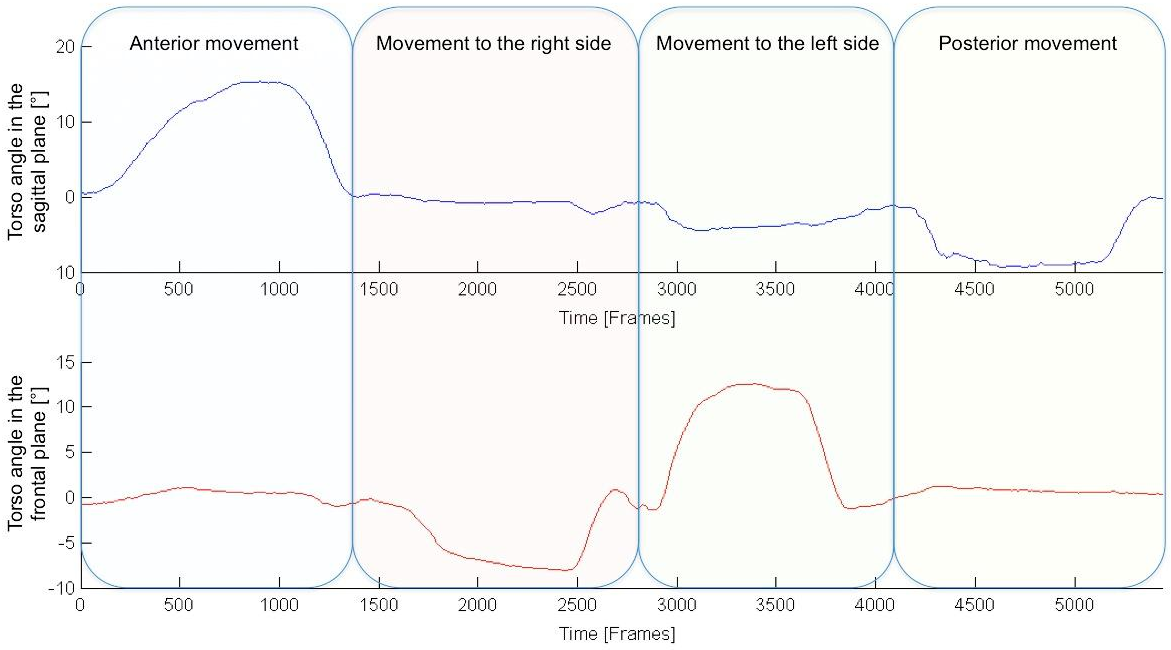


Figure 19: Characteristical MDRT torso angle curves (torso in reference to the pelvis) of one PD subject.

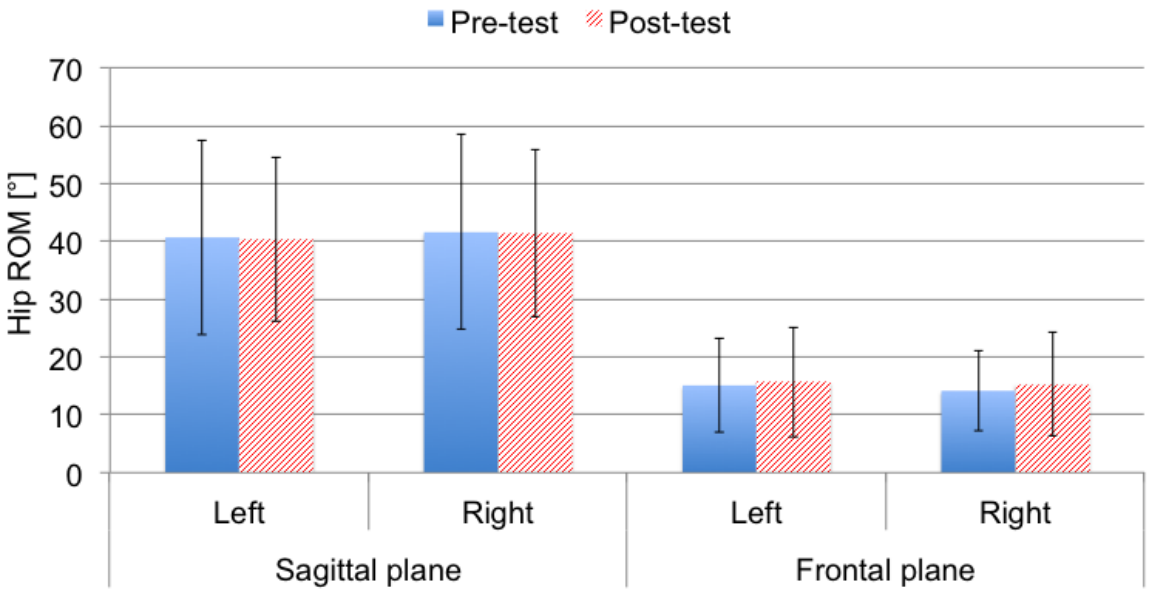


Figure 20: MDRT hip ROM values (averages and standard deviations) before and after the spinal mobilization.

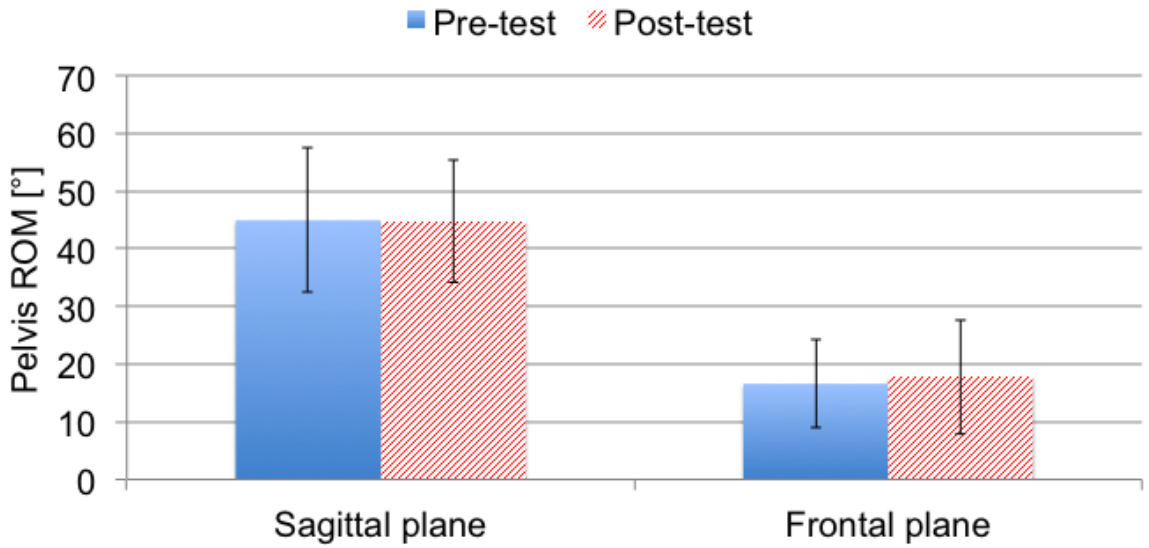


Figure 21: MDRT pelvis ROM values (averages and standard deviations) before and after the spinal mobilization.

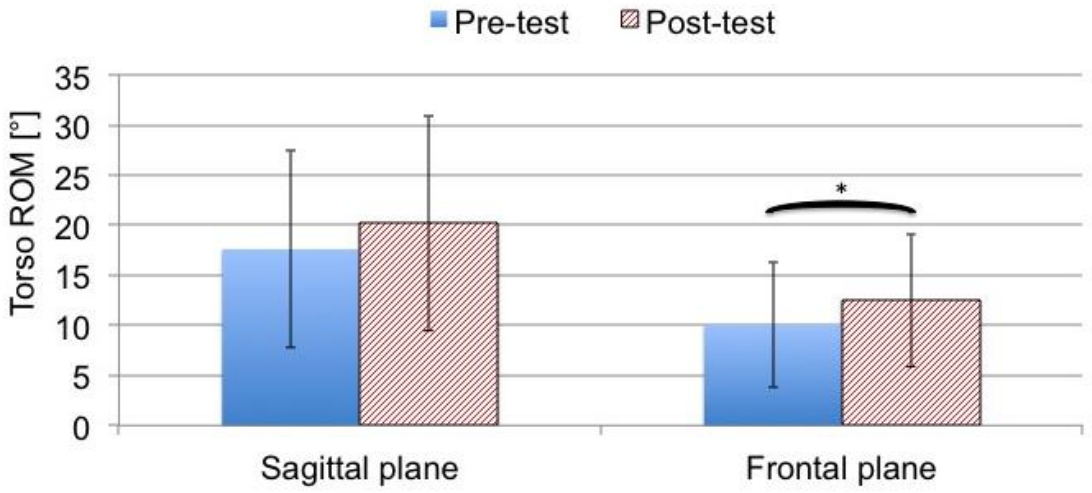


Figure 22: MDRT torso ROM values (averages and standard deviations) before and after the spinal mobilization.

Table 11: MDRT ROM values (averages ± standard deviations) before and after the intervention.

ROM		Sagittal plane		Frontal plane	
		Left	Right	Left	Right
Hip	Pre-test	40.7±16.8°	41.6±16.8°	15.1±8.1°	14.2±6.9°
	Post-test	40.3±14.1°	41.4±14.4°	15.7±9.5°	15.3±9.0°
Pelvis	Pre-test	44.9±12.5°		16.6±7.7°	
	Post-test	44.7±10.5°		17.8±9.9°	
Torso	Pre-test	17.6±9.8°		10.1±6.3°	
	Post-test	20.2±10.8°		12.5±6.6°	

There was no clear connection between the deterioration of ROM of one joint and the improvement of ROM of another joint. Most subjects who had an improved ROM after the spinal mobilization had an improvement in most ROM parameters (for example: hip, pelvis and torso). No clear trend towards the compensation of a deteriorated ROM of one joint through an improvement of ROM of another joint could be observed. Figure 32 and Figure 33 in annex N illustrated percentage changes of hip, pelvis, and torso ROM due to the spinal mobilization.

Table 12 show the percentage change of hip, pelvis, and torso ROM data in the connection with the results of question three from the questionary. No clear trend in subjects who recognized an improvement in their balance was visible.

Table 12: Percentage changes in hip, pelvis and torso reach test ROM between the pre and post test for PD patients who recognized an improvement in their gait (improvement rows) and for PD patient who did not recognize any change in their gait (no change rows). Green values represent improvements and red values represent deteriorations.

Q3	Patient	PD-Stage	Hip				Pelvis		Torso	
			Sagittal plane		Frontal plane		Sagittal plane	Frontal plane	Sagittal plane	Frontal plane
			Left	Right	Left	Right				
improvment	PD04	3	23.8	5.8	45.5	29.6	32.1	35.1	24.6	156.6
	PD15	3	-7.2	-10.2	-44.0	-14.4	-3.8	-4.3	-10.3	13.6
	PD20	3	-28.5	-21.1	-19.2	-5.8	-23.8	-23.0	18.2	9.1
	PD21	3	-49.5	-46.8	-37.5	-25.5	-34.2	-18.0	-0.7	20.0
	PD22	3	14.0	8.7	14.8	29.4	14.8	31.0	8.5	-5.6
	PD11	2	13.1	40.6	68.5	57.6	27.1	57.0	-10.1	-41.8
	PD23	2	53.3	24.9	55.2	49.5	15.7	21.3	74.1	18.0
	PD28	2	-11.9	-10.2	4.2	1.5	-18.3	16.7	91.4	-6.9
no change	PD14	3	5.3	8.5	64.9	46.3	3.2	28.1	-41.0	-36.8
	PD27	3	21.4	15.7	-13.6	-16.2	3.9	11.1	41.8	64.6
	PD07	2	-1.5	10.6	47.0	57.2	26.7	58.8	-27.8	66.4
	PD12	2	-21.0	-22.0	-42.4	-45.7	-24.1	-59.1	-36.7	55.6
	PD17	2	30.1	21.3	11.8	4.8	6.7	-4.2	36.0	10.4
	PD18	2	33.3	32.2	15.9	-3.8	22.5	6.8	80.6	50.0
	PD26	2	-5.2	-3.8	-3.3	-3.3	-4.3	-21.0	13.6	45.6

Summary of the reach test findings:

In the frontal plane average hip, pelvis, and torso ROM increased.
In the sagittal plane average torso ROM increased but average hip and pelvis ROM slightly decreased.

Significant results

- Torso ROM in the frontal plane increased due to the intervention

Subjective evaluation had no connection to improvements/ deteriorations in reach test ROM values.

2.4 Discussion

One spinal mobilization treatment session led to several changes in people with PD. In gait trials speed, stride width and length, stance time, DLS time, and step length (left leg) significantly improved. Major ROM changes in gait occured in the hip and pelvis segments. Hip ROM in the sagittal (right hip significantly increased) and transverse planes increased. Pelvis ROM in the transverse plane significantly decreased. MDRT analysis showed increased average hip, pelvis, and torso (significant) ROM values in the frontal plane after the intervention. In the sagittal plane torso ROM increased after the spinal mobilization. All these findings support the hypothesis that PD patients benefit from a spinal mobilization treatment.

Hass et al.[25] did a large cohort study with 310 people with PD and evaluated quantitatve gait data for these individuals. Among other things, this study provided mean velocity, stride length, step width, swing time, stance time and DLS time values for participants with PD at three different Hoehn and Yahr stage levels (≤1.5, 2-2.5, 3-4). The comparison of the data from the present study with the data of the moderately affected group (Hoehn and Yahr stage 2-2.5) of the study from Hass et al. showed very similar values in most parameters. However, mean stance time and DLS time values were lower in the present study compared to the study of Hass et al.. Only 17 people with PD were enrolled in the present study and most of them were very active. This unusually high activity level for people with moderate PD could be a reason for the lower single limb stance time and DLS time values compared to the findings of Hass et al..

Several studies reported a reduction in gait speed and stride length in individuals with PD when compared to aged matched control participants [26–29]. The results of the present study support these previous findings. PD participants were walking with a decreased velocity, a decreased stride length and step length, and an increased cycle time and stance time when compared to control participants.

A recently published study showed that a reduced step length also persists when PD participants and control participants walk at a similar velocity. The author suggested that the abnormal control of step length is independent of walking speed in people with PD and could be caused by a lack of automaticity. People with PD, therefore, require more attention to maintain their step size [30]. In the present study walking speed and stride length, as well as stride width, stance time, DLS

time and step length significantly improved due to the spinal mobilization treatment. These findings suggest that abnormal gait in people with PD is not only caused by a lack of automaticity and might also be the result of an increased muscle tone.

Quality of life is, among other things, related to spinal ROM and balance [31]. Restricted spinal mobility is associated with difficulties in performing heavy housework and climbing stairs [32]. Schenkman et al. [21] found a decreased spine ROM in patients with Hoehn and Yahr stages between 1.5 and 3 when compared to individuals with the same age, body mass index, and gender. Ickenstein et al. [33] detected significantly worse balance impairments in people with PD when compared to healthy control participants. The MDRT analysis of the current study clearly showed improvements in torso ROM (significant increase of torso ROM in the frontal plane) after the spinal mobilization. This fact with the finding of a significantly decreased stride width in gait trials, which indicates more balance and self-confidence, may help people with PD to handle activities of daily living better and therefore improve their quality of life.

Spinal cord stimulation is an effective surgical therapy for chronic intractable pain. Agari and Date [34] showed that spinal cord stimulation significantly decreases pain and improves abnormal posture and gait in people with PD. They also mentioned that the effect of spinal cord stimulation may be an indirect effect of pain reduction. The prevalence of back pain in people with PD ranges from 59.6%[35] to 74%[36]. In people with PD, musculoskeletal pain, which can cause back pain, may result from a combination of factors, including rigidity, arthralgic pain, skeletal deformity, and mechanical factors [37]. Pain was not evaluated in the present study and therefore it is not possible to conclude that the improvements in gait parameters were a result of a decreased rigidity. It could also be a result of a decrease in pain or both, a decrease in rigidity and a decrease in pain. This is one of the main limitations of the current study. Further studies in this area of research should include pain evaluation of PD participants to be able to clearly identify the reason for the improvement of gait parameters.

Another limitation of the present study is the small number of participants, especially for the control group (13 subjects). Increasing the number of control participants may lead to significant differences between PD participants and control participants too.

PD participants received one spinal mobilization therapy and only the immediate effect of the treatment was evaluated. It is still unclear how long the positive effect on gait parameters will last and what effects an intervention for several weeks would have. A larger study is planned for the future with the aim to answer these remaining open questions.

2.5 Conclusion

Significantly improved gait parameters were found immediately after PD participants received one spinal mobilization treatment.

3 Part II: Stroke study

3.1 Introduction

Worldwide approximately 9 million new stroke events occur every year [38]. The ability to walk becomes impaired in more than 80% of people who suffered a stroke. Most people regain some ability to walk but the majority struggle with an inefficient, asymmetrical or unsafe gait and a limited mobility. Body weight supported (BWS) treadmill training, initially developed for people with spinal cord injuries, is recognized as a promising therapy to improve gait rehabilitation of post-stroke patients [5]. BWS allows people to carry a reduced percentage of their weight while physical therapists work to reintroduce a coordinated gait pattern. The findings of some randomized studies suggest that BWS treadmill training may be more effective than conventional gait training for improving gait parameters such as stride length and paretic limb stance time [39], [40]. Mulroy et al. [41] found an association between increased walking speed and increased maximal hip extension angle during late stance after BWS treadmill training in post-stroke patients. However, no conventional gait training group was involved in this study and therefore the comparison of their findings between the BWS treadmill intervention and a traditional gait intervention was not possible. No studies are available, which compared joint ROM values between BWS treadmill training and traditional gait training in post-stroke patients. The purpose of this study was therefore to utilize BWS training during initial rehabilitation of post-stroke people and to analyze gait mechanics in addition to spatiotemporal gait parameters within 48 hours of discharge from the in-patient rehabilitation facility. The goal thereby was to compare BWS gait training with conventional over-ground gait training. The first specific aim of the study was to determine if stroke patients who are trained with BWS show better gait parameters than those trained without BWS. The second specific aim was to analyze how closely the gait parameters of both groups resemble normal gait of an age-matched population without stroke.

3.2 Material and methods

This study was conducted in cooperation with the Naples Community Hospital Brookdale Center for Healthy Aging and Rehabilitation (Naples, FL, USA). 10 stroke subjects (mean BMI 26.3±3.6) were randomized into two groups: (1) BWS treadmill training for retraining walking (BWS group), and (2) conventional, over-ground gait training (CT group). 13 age-matched healthy subjects (mean BMI 25.6±3.9) were enrolled in this study (Control group). Within 48 hours of discharge from the rehabilitation hospital, the post-stroke participants came to the Florida Gulf Coast University (Fort Myers, FL, USA) for gait analysis to determine which of the two groups achieved gait parameters most similar to the gait of an age-matched normal population. All participants were instructed to walk at a self-selected paced for the walking trials. The data from these groups represent a subcomponent of a larger investigation. The study was approved by the Florida Gulf Coast University Institutional Review Board.

3.2.1 Motion capturing

For data collection, 44 reflective markers were placed on bony landmarks of the feet, legs, thighs, pelvis, and shoulders of each subject. A 10-camera OQUS 300 1.3MP infrared motion capture system (Qualisys, Gothenburg, Sweden) was used to capture kinematic data. The torso segment was defined by the shoulder markers (right and left) and the iliac crest markers (right and left). Four clustered markers on the lower back served as tracking markers for the torso. The pelvis was defined by the iliac crest markers and the greater trochanter markers. Anterior and posterior superior iliac spine markers were used for tracking the pelvis. The thighs were defined by the greater trochanter markers and the medial and lateral knee markers. Four markers in a cluster on each thigh served as tracking markers for the upper legs. The legs were defined by the medial and lateral knee markers and the medial and lateral ankle makers. Four clustered markers on each leg were used for tracking the legs. Feet were defined by both ankle markers (medial and lateral) and foot markers at the 1^{st} and 5^{th} metatarsal head. The foot markers together with the heel markers were also used for tracking the feet. These marker positions were

chosen according to the guidelines from Visual3D (C-motion, Germantown, MD, USA), which was also the used software for data analysis.

3.2.2 Gait training

All post-stroke participants were treated in the NCH Brookdale Center for Health Aging and Rehabilitation (Naples, FL, USA). Rehabilitation started 2 to 7 days after the stroke occurred. The BWS group received BWS treadmill training. Participants walked on a motorized treadmill, secured by a harness combined with a suspension system releasing body weight. BWS treadmill training is based on the central pattern generator (CPG) theory, which proposes that gait is largely controlled by neurons located at the spinal level. These CPG's can be activated through afferent input associated with mass repetitions of typical gait motion and may lead to neural reorganization [42]. The conventional training group (CT group) received traditional overground gait training.

3.2.3 Data collection

ROM values from the knee, hip and foot progression (foot angle in space in the transverse plane), as well as from the pelvis in space, and from the torso in reference to the pelvis were calculated for the gait trials. ROM data for all three planes (sagittal, frontal, and trasverse) were considered in torso, pelvis, hip, and knee angle. Foot progression angle ROM was evaluated for the transverse plane. Recorded spatiotemporal gait parameters include step length and width, step time, stance time, swing time, gait velocity, stride length, cycle time and double limb support (DLS) time. Length of stay (LOS), units of gait training, and improvement in walking were recorded as well. Walking and improvement of walking were evaluated with the „Functional Independence Measure (FIM)“ [43].

3.2.4 Statistical methods

Averages and standard deviations were computed for all values. Statistical comparisons were performed with SPSS Statistics software (version 11.5). A students t-test served to determine if there were differences between the CT group and the BWS training group in gait parameters of the paretic limb side. A one way analysis of variance (ANOVA) was used for the comparison of all three groups (CT group, BWS group, and normal (control) group). P-values of <0.05 were considered as statistically significant.

3.3 Results

All post-stroke participants were using the same walker for the gait trials. One participant from the BWS group was excluded because this person was not able to use the walker in a proper way due to a lame arm. Table 14 to Table 16 show mean values of all analyzed gait parameters. Figure 23 to Figure 28 illustrate these parameters for all three groups (BWS group, CT group, and control group).

3.3.1 CT group versus BWS training group

Mean FIM walking improvement was higher in the CT group than in the BWS group. Participants in the CT group received fewer units of gait training and had a greater improvement per unit of gait compared to the participants in the BWS group. Average length of stay was lower in the CT group. These values did not show any significant differences between both groups.

Table 13: Mean values and standard deviations for the improvement in walking (FIM), units of received gait training, improvement per units of gait training, and length of stay. BWS=body weight supported gait training group, CT=conventional gait training group.

Parameters	BWS	CT
Walking improvement (FIM)	2.5±1.0	3±1.2
Units of gait training	43±24.5	31±7.1
Improvement/unit of gait	0.07±0.05	0.09±0.04
Length of stay	18.8±5.5	16.8±2.7

The CT group showed higher mean hip ROM values in the frontal plane, and foot progression ROM values of both, the paretic and the non-paretic limb, than the BWS training group. Mean knee ROM of the paretic leg were higher in the CT group, except of knee ROM in the transverse plane. In the non-paretic leg, knee ROM values were higher in the BWS training group. In the BWS training group,

mean torso, and pelvis ROM values were lower than in the conventional training group. For all of these torso and pelvis ROM values, except for torso ROM in the transverse plane, the BWS training group were closer to the values of normal controls as compared to the CT group. In the BWS group, pelvis ROM values in the sagittal ($p=0.05$) and frontal plane ($p=0.029$) were significantly lower than in the CT group. Spatiotemporal data of the paretic limb showed superior properties (longer step length, shorter step time, and shorter swing time) for the BWS training group when compared to the CT group. In addition, faster gait velocity, longer stride length, and a shorter cycle time and DLS time were found in the BWS group. Mean stride width values were slightly lower in the CT group than in the BWS training group. Differences of hip ROM values between the paretic and non-paretic leg were lower in the BWS training group compared to the CT group with the exception of hip ROM in the frontal plane, which showed smaller differences between the affected and unaffected leg in the CT group. Differences of knee ROM values between the paretic and non-paretic leg were lower in the CT group with the exception of knee ROM in the transverse plane. Differences in foot progression ROM between the left and right side was lower in the BWS group. Differences of step length, single limb stance time, and swing time between the paretic and non-paretic limb were lower in the BWS group. The difference of single limb stance time was significantly lower ($p=0.039$) in the BWS training group when compared to the CT group.

Table 14: Mean ROM values [°] for all three groups. BWS=body weight supported training group, CT=conventional training group, Control=control group, Sag=Sagittal plane, Fro=Frontal plane, Tra=Transverse plane, and Foot progr=Foot progression.

Torso	Sag	Fro	Tra
BWS	3.91±2.28	2.59±1.68	2.87±0.71
CT	5.68±1.86	4.87±1.99	4.65±1.49
Control	3.59±1.43	3.60±1.84	3.84±1.86

Pelvis	Sag	Fro	Tra
BWS	5.00±2.10	4.26±1.27	8.47±1.43
CT	8.67±3.68	7.14±1.53	10.88±4.94
Control	2.86±1.13	4.92±1.54	7.71±2.70

Hip	Paretic side			Non-paretic side		
	Sag	Fro	Tra	Sag	Fro	Tra
BWS	27.61±1.33	8.06±1.43	9.38±4.42	32.09±7.42	7.94±1.69	11.00±5.11
CT	27.75±7.24	9.46±1.82	9.32±2.39	34.52±4.81	9.36±3.21	11.55±2.12
Control				39.45±4.91	11.28±1.34	11.50±2.76

Knee	Paretic side			Non-paretic side		
	Sag	Fro	Tra	Sag	Fro	Tra
BWS	38.89±9.42	7.00±0.66	15.12±4.23	48.82±6.42	7.68±3.62	12.97±1.62
CT	41.58±7.44	8.42±1.91	14.93±3.83	47.06±7.61	7.44±1.28	11.99±2.76
Control				60.81±4.51	9.16±2.20	16.97±3.79

Foot progr	Paretic side	Non-paretic side
BWS	7.29±1.16	10.53±2.63
CT	8.90±2.20	12.91±5.31
Control		13.56±4.86

Table 15: Spatiotemporal gait data for all three groups. BWS=body weight supported training group, CT=conventional training group, and Control=control group.

Gait parameter		BWS	CT	Control
Stride width [m]		0.16±0.04	0.14±0.01	0.10±0.02
Stride length [m]		0.73±0.17	0.65±0.20	1.22±0.11
Cycle time [s]		1.92±0.29	2.24±0.58	1.05±0.06
Step length [m]	Paretic side	0.38±0.07	0.31±0.14	
	Non-paretic side	0.35±0.12	0.33±0.09	0.61±0.06
Step time [s]	Paretic side	1.04±0.22	1.30±0.45	
	Non-paretic side	0.89±0.14	0.93±0.25	0.53±0.03
Stance time [s]	Paretic side	1.37±0.25	1.53±0.34	
	Non-paretic side	1.44±0.27	1.83±0.56	0.65±0.05
Swing time [s]	Paretic side	0.55±0.03	0.70±0.25	
	Non-paretic side	0.48±0.09	0.43±0.13	0.41±0.03
Gait velocity [m/s]		0.40±0.15	0.30±0.10	1.16±0.11
DLS time [s]		0.90±0.26	1.12±0.34	0.24±0.04

Table 16: Differences in gait parameters between the left and right limb. BWS=body weight supported training group, CT=conventional training group, Control=control group, Sag=Sagittal plane, Fro=Frontal plane, and Tra=Transverse plane.

Gait parameter		BWS	CT	Control
Step length [m]		0.06±0.06	0.09±0.08	0.03±0.02
Single stance time [s]		0.07±0.05	0.31±0.28	0.01±0.01
Swing time [s]		0.09±0.05	0.28±0.26	0.01±0.01
Hip [°]	Sag	4.85±6.08	6.74±6.66	3.02±1.95
	Fro	2.40±0.71	1.38±1.16	1.84±1.80
	Tra	1.60±0.99	3.40±2.46	2.48±1.98
Knee [°]	Sag	10.48±12.39	7.72±8.37	2.35±2.12
	Fro	2.40±1.52	1.32±1.44	2.33±1.62
	Tra	3.45±2.53	3.78±3.88	3.32±2.90
Foot proggression [°]		3.50±2.27	4.62±5.97	2.45±2.48

3.3.2 Control group (healthy subjects)

The control group shower higher hip, knee, and foot progression ROM values than the post-stroke groups. Mean torso ROM of the control group was lower in the sagittal plane than in both post-stroke groups. In the frontal and transverse plane, torso ROM of the control group were lower compared to the CT group but higher compared to the BWS training group. Pelvis ROM values in the sagittal and transverse planes were lower in the control group than in the post-stroke groups. In the frontal plane, mean pelvis ROM of the control group was lower compared to the CT group but higher compared to the BWS group. Spatiotemporal data showed increased gait velocity, stride length, and step length, and decreased stride width, cycle time, DLS time, step time, and single limb stance time for the control group when compared to the post-stroke groups. In the control group, differences of the hip ROM between the left and right hip were lower in sagittal plane when compared to the post-stroke groups. Differences in hip ROM in the frontal plane of the control group were higher than in the CT group but lower than in the BWS group. Differences in hip ROM in the transverse plane of the control group were higher than in the BWS group but lower than in the CT group. Differences in knee ROM in the sagittal plane between the left and right limb were lower in the control group than in both post-stroke groups. Differences in foot progression ROM in the control group was lower than in both post-stroke group. Spatiotemporal gait data

differences, including step length, single limb stance time, and swing time differences between the left and right leg, were lower in the control group when compared to the post-stroke groups. Table 17 shows all significant differences between the control group and any of the post-stroke groups.

Table 17: Significant difference from the ANOVA analysis and the Bonforroni post hoc test. The third column shows differences between groups. 1=BWS therapy group, 2=conventional therapy group, and 3=control group (healthy subjects).

Parameter	p-values (ANOVA)	Bonforroni post hoc test p-values
Non-paretic hip sag	0.048	no significant differences
Non-paretic hip fro	0.016	1-3: p=0.021
Non-paretic knee sag	<0.0001	1-3: p=0.004, 2-3: p=0.001
Non-paretic knee trans	0.017	2-3: p=0.031
Pelvis sag	<0.0001	1-2: p=0.05, 2-3: p<0.0001
Pelvis fro	0.016	1-2: p=0.029, 2-3: p=0.032
Speed	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
Stride wide	0.001	1-3: p=0.002, 2-3: p=0.012
Stride length	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
Cycle time	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
Non-paretic step length	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
Non-paretic step time	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
Non-paretic stance time	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
DLS time	<0.0001	1-3: p<0.0001, 2-3: p<0.0001
Difference stance time	0.001	1-2: p=0.039, 2-3: p=0.001
Difference swing time	0.002	2-3: p=0.002

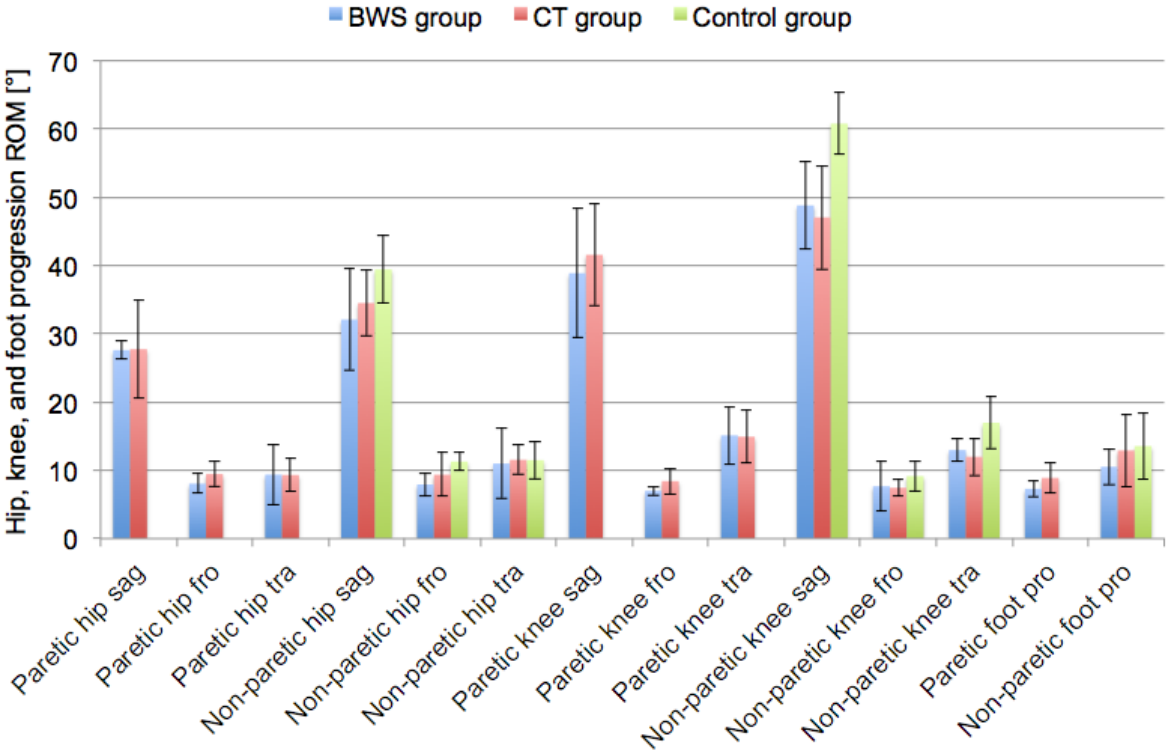


Figure 23: Mean hip, knee, and foot progression ROM values for all three groups. BWS group=body weight supported training group, CT group=conventional training group, sag=sagittal plane, fro=frontal plane, and tra=transverse plane.

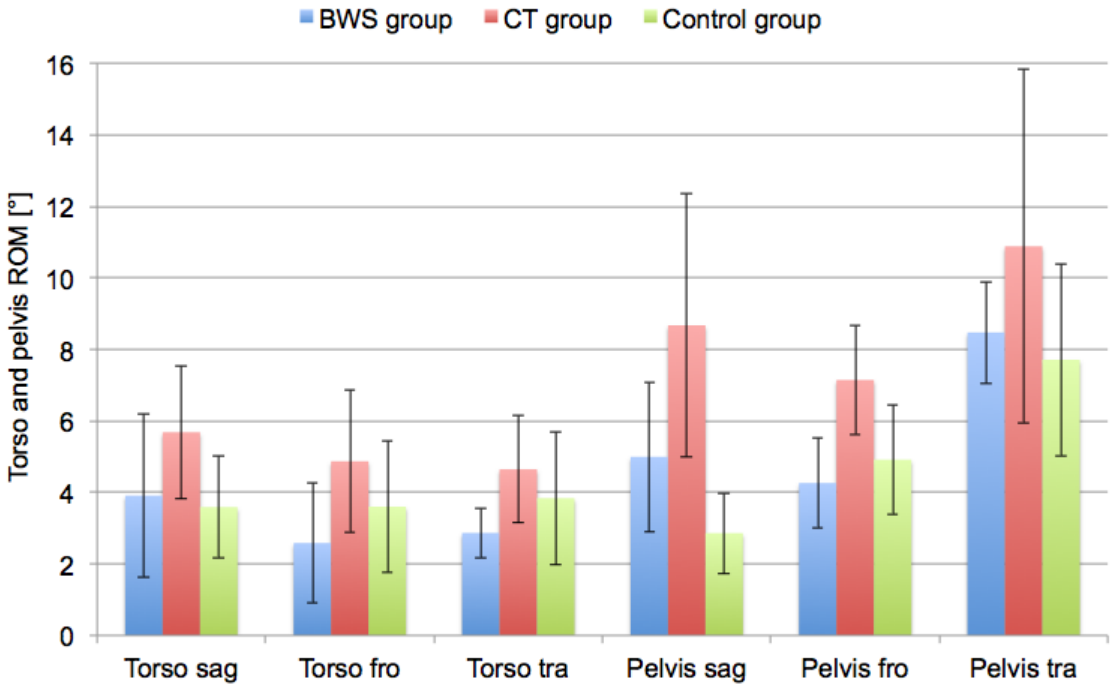


Figure 24: Mean torso and pelvis ROM values for all three groups. BWS group=body weight supported training group, CT group=conventional training group, sag=sagittal plane, fro=frontal plane, and tra=transverse plane.

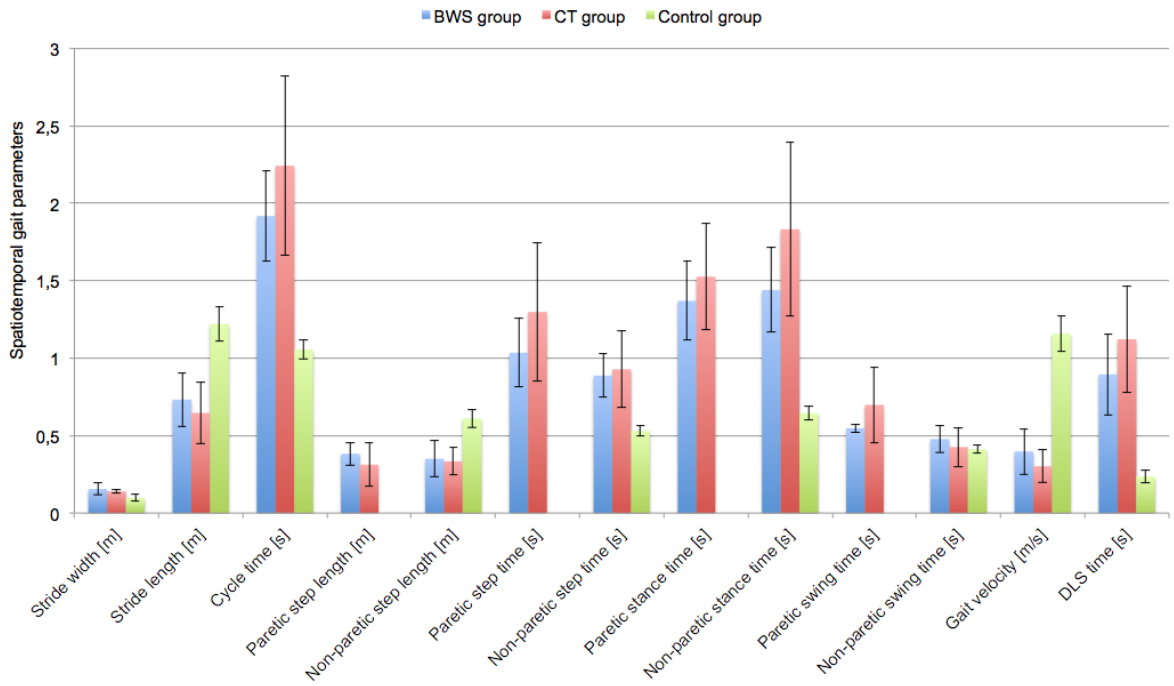


Figure 25: Spatiotemporal gait parameters for all three groups. BWS group=body weight supported training group, CT group=conventional training group.

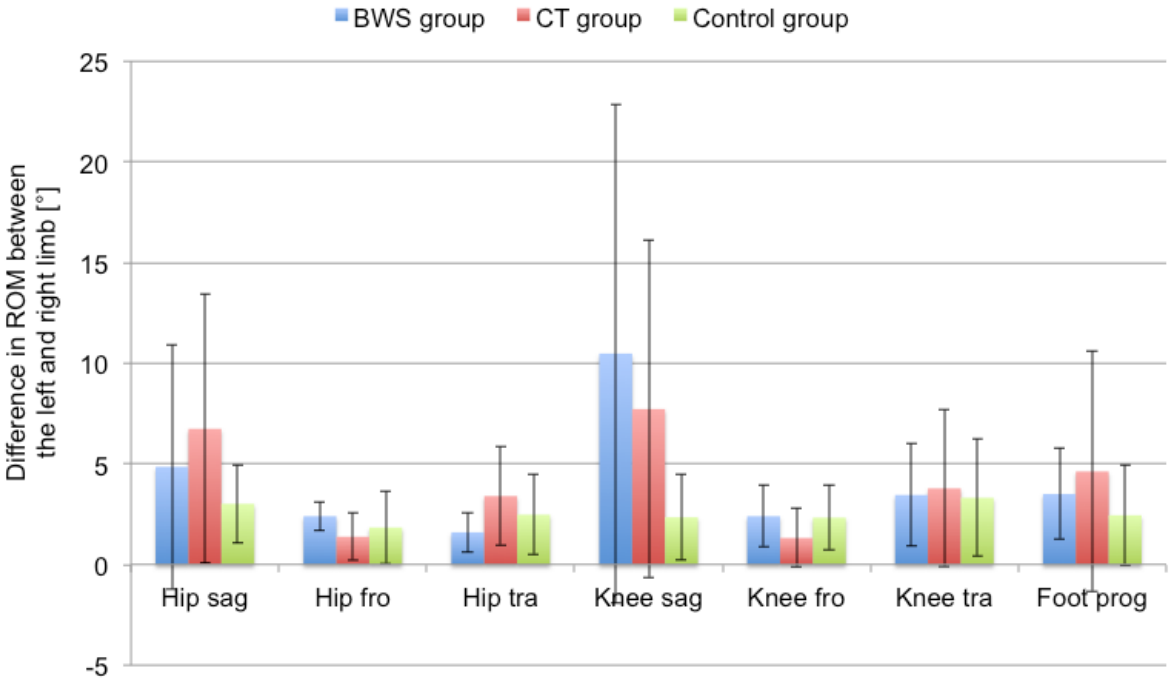


Figure 26: Difference in hip, knee, and foot progression (=Foot prog) ROM between the left and right limb. BWS group=body weight supported training group, CT group=conventional training group, sag=sagittal plane, fro=frontal plane, tra=transverse plane.

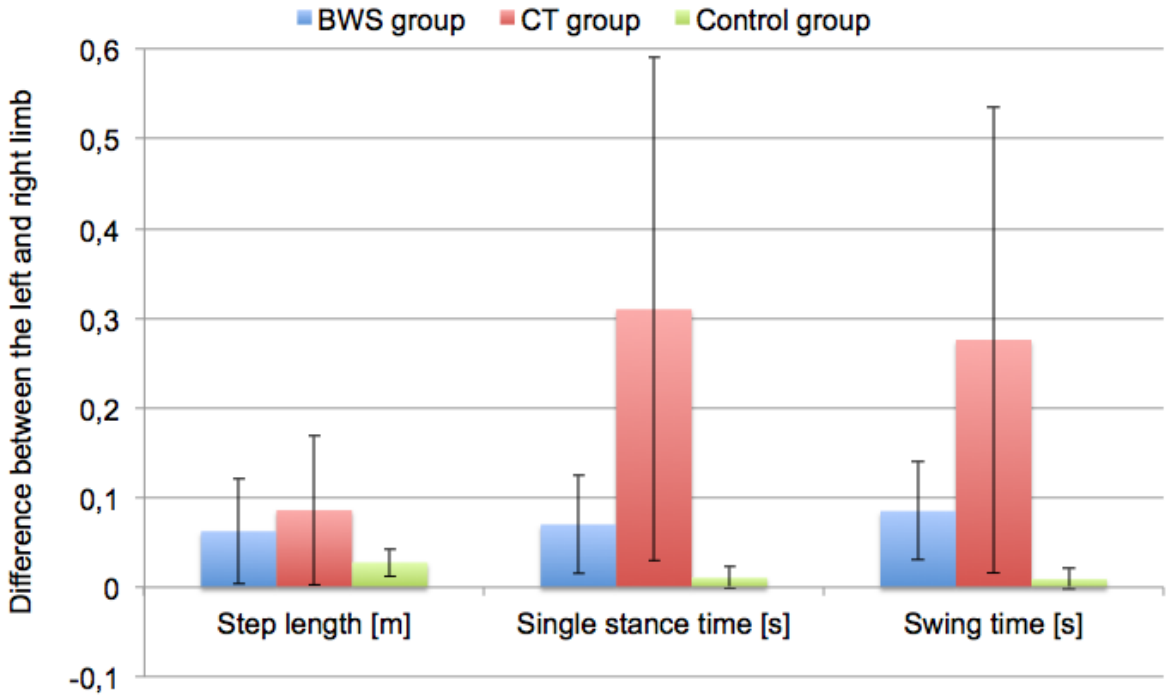


Figure 27: Difference in step length, single stance time, and swing time between the left and right limb. BWS group=body weight supported training group, CT group=conventional training group.

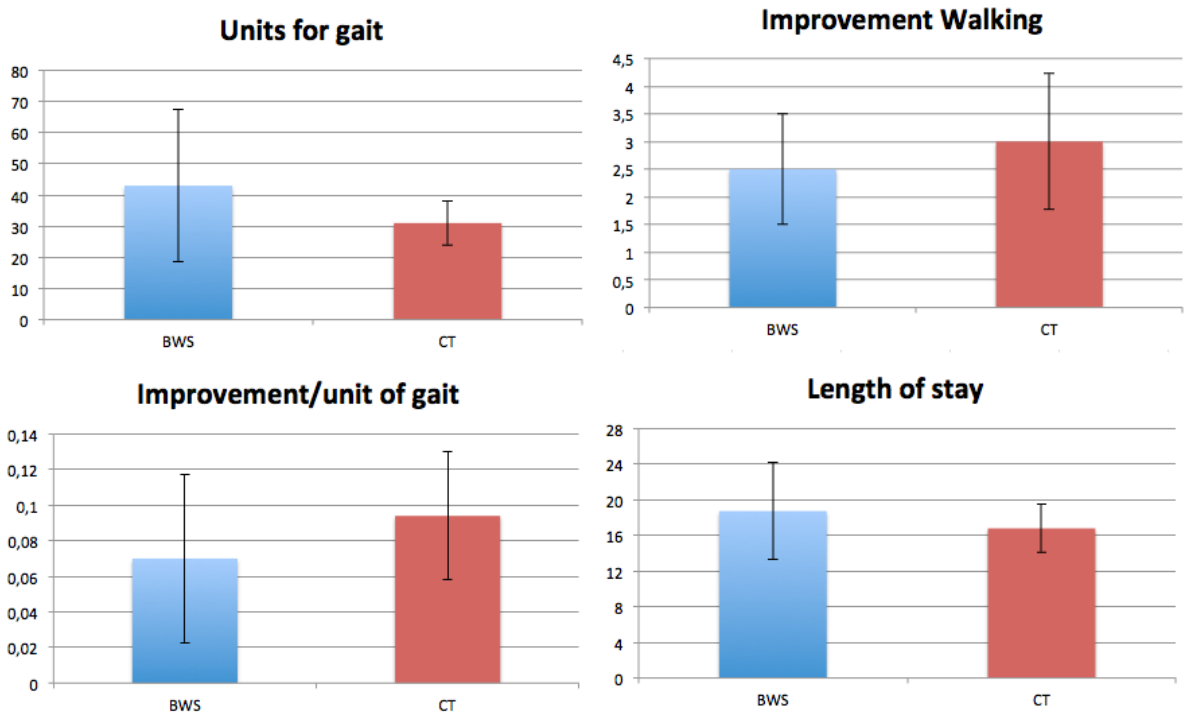


Figure 28: Mean values and standard deviations for units of received gait training, improvement in walking (FIM), improvement per unit of gait training, and length of stay. BWS=body weight supported gait training group, CT=conventional gait training group.

3.4 Discussion

In the BWS group, ROM differences between the left and right limb were higher for knee ROM in the sagittal plane, and lower for hip ROM in the sagittal and transverse planes when compared to the CT group. Pelvis and torso ROM values (with exception of torso ROM in the transverse plane) were closer to normal values in the BWS group. In the BWS group, torso ROM in the frontal and sagittal plane were lower than in the control group. These lower ROM values in the BWS group can be caused by a restriction of upper body mobility due to the harness during BWS gait training. Spatiotemporal gait data of the paretic limb showed superior values for the BWS group (longer step length, shorter step time, and shorter swing time) when compared to the CT group. Gait velocity, stride length, cycle time, and DLS time also showed superior values for the BWS group. Larger stride width, which indicates less balance control, was noticed in the BWS group when compared to the CT group. This apperently lower balance control may be caused by a lower balance benefit in the BWS group due to the harness support. Step length, single stance time, and swing time differences between the left and right limb were lower in the BWS group than in the CT group. These findings go along with the results from McCain et al. [44], who also found better gait symmetry in a BWS treadmill training group compared to a CT group. Most of the ROM values and spatiotemporal gait data showed superior values for the BWS group. However, the CT group received less units of gait training, stayed fewer days in the hospital, and showed a higher improvement in walking than the BWS group.

Liu et al. [45] showed that walking with a rolling walker alters the gait pattern compared to walking without any assistive device. Altered gait parameters included decreased speed, swing time, step and stride length, and increased DLS and stance time. In a comparison between walking with three different walkers and unassisted walking in older adults, Protas et al. [46] did not find any significant differences between walking with an assistive device called WalkAbout and walking without a device in regard to gait speed, stride lengths, 5-minutes walk distance, and oxygen consumption. Walking with the other two analyzed walkers showed differences in the analyzed parameters. Melis et al. [47] determined the influence of walkers, crutches and cances on assisted-gait and found slower gait velocity and a

forward flexed posture in participants who walked with a walker. All post-stroke participants in this study used a walker for the gait trials. This fact may be the reason for the big differences in gait parameters between the post-stroke groups and the control group. Torso and pelvis ROM values were closer to values of normal people in the BWS group, whereby it seems that BWS training is superior to the CT in regard to regain a normal posture during walking.

Overground gait training is the common physical therapy to improve gait in post-stroke patients. However, in a review of nine studies involving 499 participants, States et al. [48] did not find sufficient evidence to determine if overground physical therapy gait training benefits gait function in patients with chronic stroke. On the other hand, Franceschini et al. [49] compared functional outcomes between BWS treadmill training and overground gait training and did not find any difference. A review of fifteen trials (622 participants) by the Cochrane Collaboration indicated that there were no significant differences between tradmill training, with or without BWS, and other interventions on walking after stroke [50]. In contrast to these studies, some studies are available which suggest that BWS treadmill training may be more effective than conventional gait training for improving gait parameters [39], [40]. These contradictory results show that further research is necessary to clearly show if BWS treadmill training is superior to other intervention in regard to restore gait functions in post-stroke patients.

The main limitation of this study is the small number of participants in the BWS and CT groups, which makes the interpretation of the findings very difficult. All significant results (lower pelvis ROM values in the sagittal and frontal planes, and lower difference in single stance time between both limbs in the BWS group) indicate that BWS training is superior to CT. On the other hand, the CT group received less units of gait training, and showed a higher improvement in walking than the BWS group. Therefore, at this time, it is not possible to make a clear statement whether BWS training or CT is more efficient for gait recovery in post-stroke patients. More participants will be recruited within the next months.

3.5 Conslusion

Clear conclusions regarding whether BWS training or CT has more advantages in regaining normal gait pattern in post-stroke patients were at this stage of the study not possible because of the small number of participants.

Bibliography

[1] M. Whittle, *Gait analysis : an introduction*. Edinburgh; New York: Butterworth-Heinemann, 2002.

[2] P. C. Montgomery und B. H. Connolly, *Clinical applications for motor control*. Thorofare, NJ: SLACK, 2002.

[3] Duysens und Van de Crommert HW, „Neural control of locomotion; The central pattern generator from cats to humans“, *Gait Posture*, Bd. 7, Nr. 2, S. 131–141, März 1998.

[4] D. A. Rosenbaum, *Human motor control*. Amsterdam: Elsevier Inc, 2010.

[5] R. Verma, K. N. Arya, P. Sharma, und R. K. Garg, „Understanding gait control in post-stroke: implications for management“, *J Bodyw Mov Ther*, Bd. 16, Nr. 1, S. 14–21, Jan. 2012.

[6] J. Perry, J. M. Burnfield, und L. M. Cabico, *Gait analysis : normal and pathological function*. Thorofare, NJ: SLACK, 2010.

[7] C-Motion, „Marker Set Guidelines“. 14-Sep-2012.

[8] M. B. Youdim und P. Riederer, „Understanding Parkinson’s disease“, *Sci. Am.*, Bd. 276, Nr. 1, S. 52–59, Jan. 1997.

[9] J. A. Obeso, M. C. Rodriguez-Oroz, C. G. Goetz, C. Marin, J. H. Kordower, M. Rodriguez, E. C. Hirsch, M. Farrer, A. H. V. Schapira, und G. Halliday, „Missing pieces in the Parkinson’s disease puzzle“, *Nat. Med.*, Bd. 16, Nr. 6, S. 653–661, Juni 2010.

[10] L. Swinn, *Parkinson’s disease : theory and practice for nurses*. London; Philadelphia: Whurr, 2005.

[11] K. Wirdefeldt, H.-O. Adami, P. Cole, D. Trichopoulos, und J. Mandel, „Epidemiology and etiology of Parkinson’s disease: a review of the evidence“, *European Journal of Epidemiology*, Bd. 26, Nr. S1, S. 1–58, Mai 2011.

[12] C. Ramaker, J. Marinus, A. M. Stiggelbout, und B. J. Van Hilten, „Systematic evaluation of rating scales for impairment and disability in Parkinson’s disease“, *Mov. Disord.*, Bd. 17, Nr. 5, S. 867–876, Sep. 2002.

[13] C. G. Goetz, W. Poewe, O. Rascol, C. Sampaio, G. T. Stebbins, C. Counsell, N. Giladi, R. G. Holloway, C. G. Moore, G. K. Wenning, M. D. Yahr, und L. Seidl, „Movement Disorder Society Task Force report on the Hoehn and Yahr staging scale: status and recommendations“, *Mov. Disord.*, Bd. 19, Nr. 9, S. 1020–1028, Sep. 2004.

[14] „The Unified Parkinson’s Disease Rating Scale (UPDRS): status and recommendations“, *Mov. Disord.*, Bd. 18, Nr. 7, S. 738–750, Juli 2003.

[15] J. Jankovic und E. Tolosa, *Parkinson’s disease and movement disorders*.

Baltimore: Williams & Wilkins, 1998.
[16] A. Gordin, S. Kaakkola, und H. Teräväinen, *Parkinson's disease*. Philadelphia, PA: Lippincott Williams & Wilkins, 2003.
[17] R. Pahwa, K. E. Lyons, und W. C. Koller, *Therapy of Parkinson's disease*. New York: M. Dekker, 2004.
[18] G. J. Hankey, *Stroke : your questions answered*. Edinburgh; New York: Churchill Livingstone, 2002.
[19] G. Gillen und A. Burkhardt, *Stroke rehabilitation : a function-based approach*. St. Louis, Mo.: Mosby, 2004.
[20] D. Tan, M. Danoudis, J. McGinley, und M. E. Morris, „Relationships between motor aspects of gait impairments and activity limitations in people with Parkinson's disease: a systematic review", *Parkinsonism Relat. Disord.*, Bd. 18, Nr. 2, S. 117–124, Feb. 2012.
[21] M. L. Schenkman, K. Clark, T. Xie, M. Kuchibhatla, M. Shinberg, und L. Ray, „Spinal movement and performance of a standing reach task in participants with and without Parkinson disease", *Phys Ther*, Bd. 81, Nr. 8, S. 1400–1411, Aug. 2001.
[22] A. Delitto, S. Z. George, L. R. Van Dillen, J. M. Whitman, G. Sowa, P. Shekelle, T. R. Denninger, und J. J. Godges, „Low back pain", *J Orthop Sports Phys Ther*, Bd. 42, Nr. 4, S. A1–57, Apr. 2012.
[23] R. A. Newton, „Validity of the multi-directional reach test: a practical measure for limits of stability in older adults", *J. Gerontol. A Biol. Sci. Med. Sci.*, Bd. 56, Nr. 4, S. M248–252, Apr. 2001.
[24] K. A. Olson, *Manual physical therapy of the spine*. Edinburgh: Saunders, 2008.
[25] C. J. Hass, P. Malczak, J. Nocera, E. L. Stegemöller, A. Shukala, I. Malaty, C. E. Jacobson 4th, M. S. Okun, und N. McFarland, „Quantitative normative gait data in a large cohort of ambulatory persons with Parkinson's disease", *PLoS ONE*, Bd. 7, Nr. 8, S. e42337, 2012.
[26] S. O'Shea, M. E. Morris, und R. Iansek, „Dual task interference during gait in people with Parkinson disease: effects of motor versus cognitive secondary tasks", *Phys Ther*, Bd. 82, Nr. 9, S. 888–897, Sep. 2002.
[27] J. M. Bond und M. Morris, „Goal-directed secondary motor tasks: their effects on gait in subjects with Parkinson disease", *Arch Phys Med Rehabil*, Bd. 81, Nr. 1, S. 110–116, Jan. 2000.
[28] M. E. Morris, R. Iansek, T. A. Matyas, und J. J. Summers, „Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms", *Brain*, Bd. 119 (Pt 2), S. 551–568, Apr. 1996.
[29] O. Sofuwa, A. Nieuwboer, K. Desloovere, A.-M. Willems, F. Chavret, und I.

Jonkers, „Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group", *Arch Phys Med Rehabil*, Bd. 86, Nr. 5, S. 1007–1013, Mai 2005.
[30] M. K. Y. Mak, „Reduced step length, not step length variability is central to gait hypokinesia in people with Parkinson's disease", *Clin Neurol Neurosurg*, Aug. 2012.
[31] S. Imagama, Y. Matsuyama, Y. Hasegawa, Y. Sakai, Z. Ito, N. Ishiguro, und N. Hamajima, „Back muscle strength and spinal mobility are predictors of quality of life in middle-aged and elderly males", *Eur Spine J*, Bd. 20, Nr. 6, S. 954–961, Juni 2011.
[32] S. D. Ryan und L. P. Fried, „The impact of kyphosis on daily functioning", *J Am Geriatr Soc*, Bd. 45, Nr. 12, S. 1479–1486, Dez. 1997.
[33] G. W. Ickenstein, H. Ambach, A. Klöditz, H. Koch, S. Insenmann, H. Reichmann, und T. Ziemssen, „Static posturography in aging and Parkinson's disease", *Front Aging Neurosci*, Nr. 4:20, Aug. 2012.
[34] T. Agari und I. Date, „Spinal cord stimulation for the treatment of abnormal posture and gait disorder in patients with Parkinson's disease", *Neurol. Med. Chir. (Tokyo)*, Bd. 52, Nr. 7, S. 470–474, 2012.
[35] F. Etchepare, S. Rozenberg, T. Mirault, A.-M. Bonnet, C. Lecorre, Y. Agid, P. Bourgeois, und B. Fautrel, „Back problems in Parkinson's disease: an underestimated problem", *Joint Bone Spine*, Bd. 73, Nr. 3, S. 298–302, Mai 2006.
[36] D. Broetz, M. Eichner, T. Gasser, M. Weller, und J. P. Steinbach, „Radicular and nonradicular back pain in Parkinson's disease: a controlled study", *Mov. Disord.*, Bd. 22, Nr. 6, S. 853–856, Apr. 2007.
[37] A. D. Ha und J. Jankovic, „Pain in Parkinson's disease", *Mov. Disord.*, Bd. 27, Nr. 4, S. 485–491, Apr. 2012.
[38] World Health Organization, „The Global Burden of Disease: 2004 update (2008)", 2008.
[39] E. Høyer, R. Jahnsen, J. K. Stanghelle, und L. I. Strand, „Body weight supported treadmill training versus traditional training in patients dependent on walking assistance after stroke: a randomized controlled trial", *Disabil Rehabil*, Bd. 34, Nr. 3, S. 210–219, 2012.
[40] Y. Laufer, R. Dickstein, Y. Chefez, und E. Marcovitz, „The effect of treadmill training on the ambulation of stroke survivors in the early stages of rehabilitation: a randomized study", *J Rehabil Res Dev*, Bd. 38, Nr. 1, S. 69–78, Feb. 2001.
[41] S. J. Mulroy, T. Klassen, J. K. Gronley, V. J. Eberly, D. A. Brown, und K. J. Sullivan, „Gait parameters associated with responsiveness to treadmill training with body-weight support after stroke: an exploratory study", *Phys Ther*, Bd. 90, Nr. 2, S. 209–223, Feb. 2010.

[42] V. G. DePaul, L. R. Wishart, J. Richardson, T. D. Lee, und L. Thabane, „Varied overground walking-task practice versus body-weight-supported treadmill training in ambulatory adults within one year of stroke: a randomized controlled trial protocol“, *BMC Neurol*, Bd. 11, S. 129, 2011.
[43] Granger CV und Hamilton BB, „The Functional Independence Measure“, in *Measuring Health: A Guide to Rating Scales and Questionnaires*, Bd. Second ed, McDowell I und Newell C, Hrsg. New York: Oxford University Press, 1987, S. 115–121.
[44] K. J. McCain, F. E. Pollo, B. S. Baum, S. C. Coleman, S. Baker, und P. S. Smith, „Locomotor treadmill training with partial body-weight support before overground gait in adults with acute stroke: a pilot study“, *Arch Phys Med Rehabil*, Bd. 89, Nr. 4, S. 684–691, Apr. 2008.
[45] H. H. Liu, M. McGee, W. Wang, und M. Persson, „Comparison of gait characteristics between older rolling walker users and older potential walker users“, *Arch Gerontol Geriatr*, Bd. 48, Nr. 3, S. 276–280, Juni 2009.
[46] E. J. Protas, M. L. Raines, und S. Tissier, „Comparison of spatiotemporal and energy cost of the use of 3 different walkers and unassisted walking in older adults“, *Arch Phys Med Rehabil*, Bd. 88, Nr. 6, S. 768–773, Juni 2007.
[47] E. H. Melis, R. Torres-Moreno, H. Barbeau, und E. D. Lemaire, „Analysis of assisted-gait characteristics in persons with incomplete spinal cord injury“, *Spinal Cord*, Bd. 37, Nr. 6, S. 430–439, Juni 1999.
[48] R. A. States, E. Pappas, und Y. Salem, „Overground physical therapy gait training for chronic stroke patients with mobility deficits“, *Cochrane Database Syst Rev*, Nr. 3, S. CD006075, 2009.
[49] M. Franceschini, S. Carda, M. Agosti, R. Antenucci, D. Malgrati, und C. Cisari, „Walking after stroke: what does treadmill training with body weight support add to overground gait training in patients early after stroke?: a single-blind, randomized, controlled trial“, *Stroke*, Bd. 40, Nr. 9, S. 3079–3085, Sep. 2009.
[50] A. M. Moseley, A. Stark, I. D. Cameron, und A. Pollock, „Treadmill training and body weight support for walking after stroke“, *Cochrane Database Syst Rev*, Nr. 4, S. CD002840, 2005.
[51] C-Motion, Inc., „Creating a Virtual Laboratory: Example 1“, *http://www.c-motion.com/v3dwiki/index.php?title=Creating_a_Virtual_Laboratory:_Example_1*. 06-Aug-2012.
[52] Fahn S, Elton R, Members of the UPDRS Development Committe, „UNIFIED PARKINSON’S DISEASE RATING SCALE (UPDRS)“. www.mdvu.org/library/ratingscales/pd/updrs.pdf, 09-Okt-2012.

List of Figures

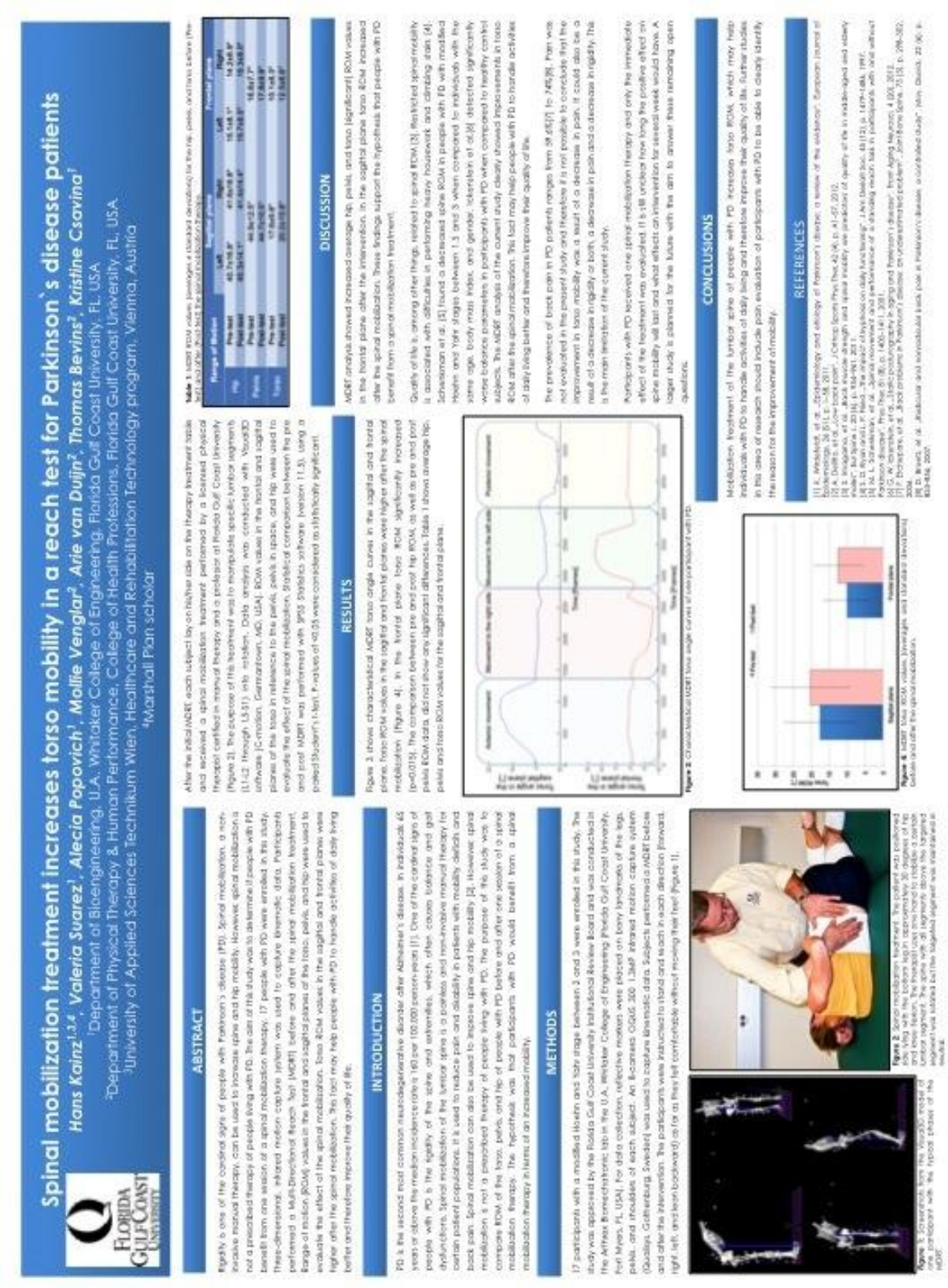
List of Tables

List of Abbreviations

BK	Back
BMES	Biomedical Engineering Society
BMI	Body Mass Index
BWS	Body weight support
CNS	Central nervous system
COMT	Catechol-O-methyltransferase
CPG	Central Pattern Generator
CT	Conventional training
DBS	Deep brain simulation
DLS	Double limb support
FGCU	Florida Gulf Coast University
FIM	Functional Independence Measure
GABA	γ–aminobutyric acid
GPi	Globus pallidus internal segment
HY	Hoehn and Yahr
LGP	Lateral globus pallidus
LOS	Length of stay
LTH	Left thigh
MDRT	Multi-Directional Reach Test
MGP	Medial globus pallidus
PD	Parkinson`s disease
PIGD	Postural imbalance and gait disorder
ROM	Range of motion
RTH	Right thigh
SLR	Step length ratio
SNC	substantia nigra pars compacta
SNR	substantia nigra pars reticulata
STN	subthalamic nucleus
TIA	Transient ischemic attack
TSR	Temporal symmetry ratio
UPDRS	Unified Parkinson`s Disease Rating Scale

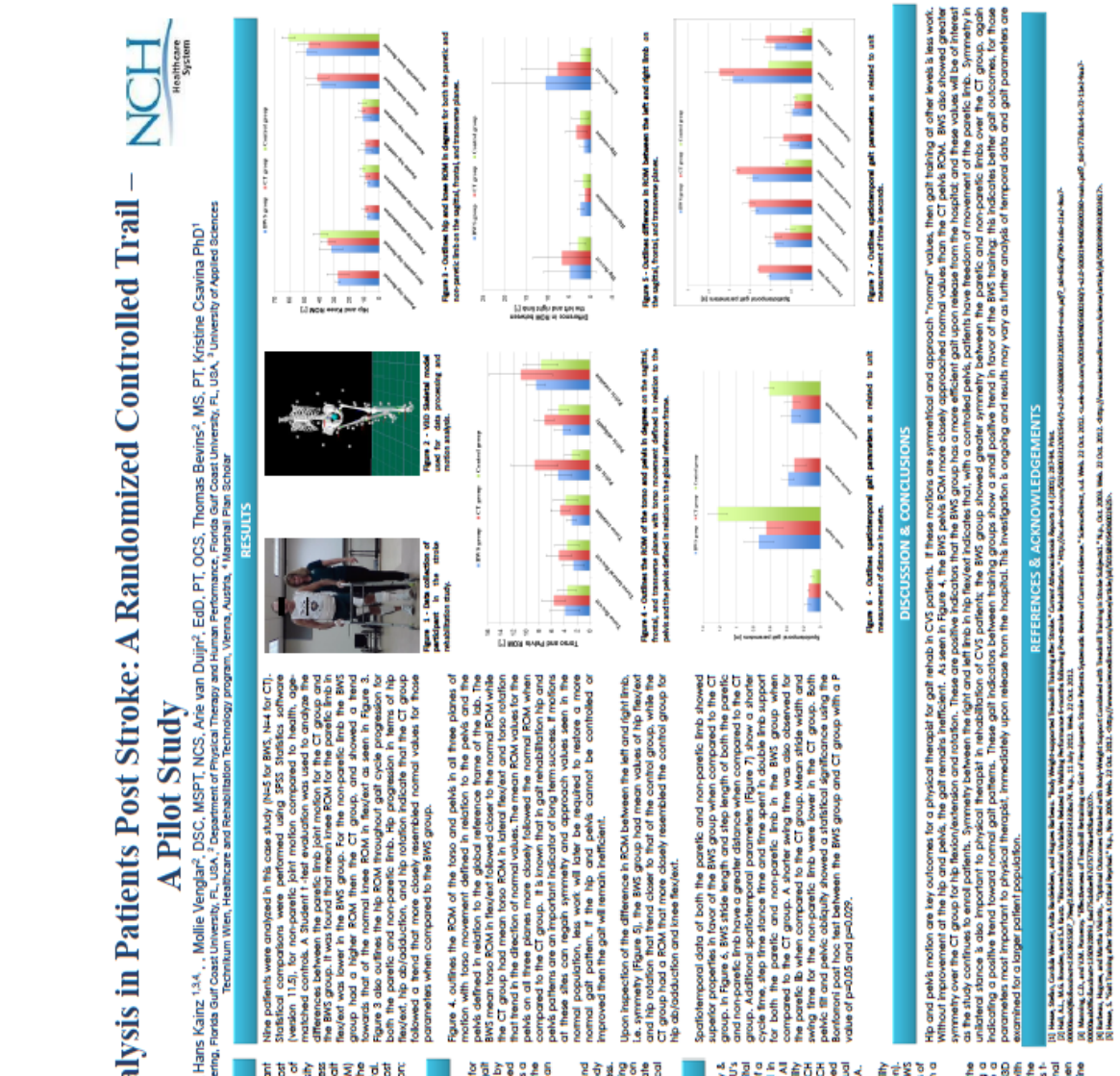
A: BMES poster I

The results of the MDRT analysis of the PD study were presented at the BMES conference with following poster:



B: BMES poster II

Some results of the stroke study were presented at the BMES conference with following poster:



C: Creation of a virtual marker in a static trial

If a necessary marker is missing in the static trial, it is often not possible to build the model for motion analysis. Assuming that the subject has symmetric anatomical landmarks, it is in some cases possible to calculate the offset of a missing marker with the information from the same marker on the other limb. If this is possible, a landmark for the missing marker can be created, whereby the original model can be applied. This process takes following steps:

1. Creation of a virtual lab
2. Projection of three markers from one segment to a virtual lab plane
3. Creation of a virtual segment
4. Same for the other limb
    a. Projection of three markers from one segment (same segment as in the other limb) to a virtual lab plane
    b. Creation of a virtual segment
5. Projection of a marker (equal marker to the missing marker but on the other limb) into all three planes of the virtual segment and calculation of the distances from the virtual segment to the marker
6. Creation of a landmark for the missing marker (with the virtual segment and the offset values from the calculation)

All this steps are explained in detail with following example:
The right lateral knee marker is missing in a static trial. There is no model for the right thigh and right shank because it would require the right lateral knee marker.

1. Creation of a virtual lab (if there is no virtual lab existing)

For a virtual lab you need at least three landsmarks. Following instructions for the creation of a virtual lab are from the c-motion wiki documentation. [51]

Create following four Landmarks:

Landmark Name= Lab_Origin
Starting Point= Enter nothing
Existing Segment= LAB
Offset Using the Following ML/AP/AXIAL Offset X=0 , Y=0 , Z=0
Calibration Only Landmark= Leave Unchecked

Landmark Name= Lab_X
Starting Point= Enter nothing
Existing Segment= LAB
Offset Using the Following ML/AP/AXIAL Offset X=0.1 , Y=0 , Z=0
Calibration Only Landmark= Leave Unchecked

Landmark Name= Lab_Y
Starting Point= Enter nothing
Existing Segment= LAB
Offset Using the Following ML/AP/AXIAL Offset X=0 , Y=0.1 , Z=0
Calibration Only Landmark= Leave Unchecked

Landmark Name= Lab_Z
Starting Point= Enter nothing
Existing Segment= LAB
Offset Using the Following ML/AP/AXIAL Offset X=0 , Y=0 , Z=0.1
Calibration Only Landmark= Leave Unchecked

A subset of three of these landmarks can be used to represent the segment coordinate system.

Switch to the segment tab in model builder mode.
→ In the segment name combo box type „Virtual Lab“
→ Check the Kinematic Only Check Box
→ Select the Create button

Define Proximal Joint and Radius
Lateral= None / Joint= Lab_Z / Medial= None / Radius= 0.001
Define Distal Joint and Radius
Lateral= None / Joint= Lab_Origin / Medial= None / Radius= 0.001

Extra Target To Define Orientation (if needed)

Location Lateral= Lab_X

Select Tracking Markers

Use Calibration Targets for Tracking Checked

→ Select Build Model

Figure 29 shows a screenshot of a subject with a missing lateral knee marker. In the bottom left corner of this figure the segment coordinate system from the virtual lab is shown.

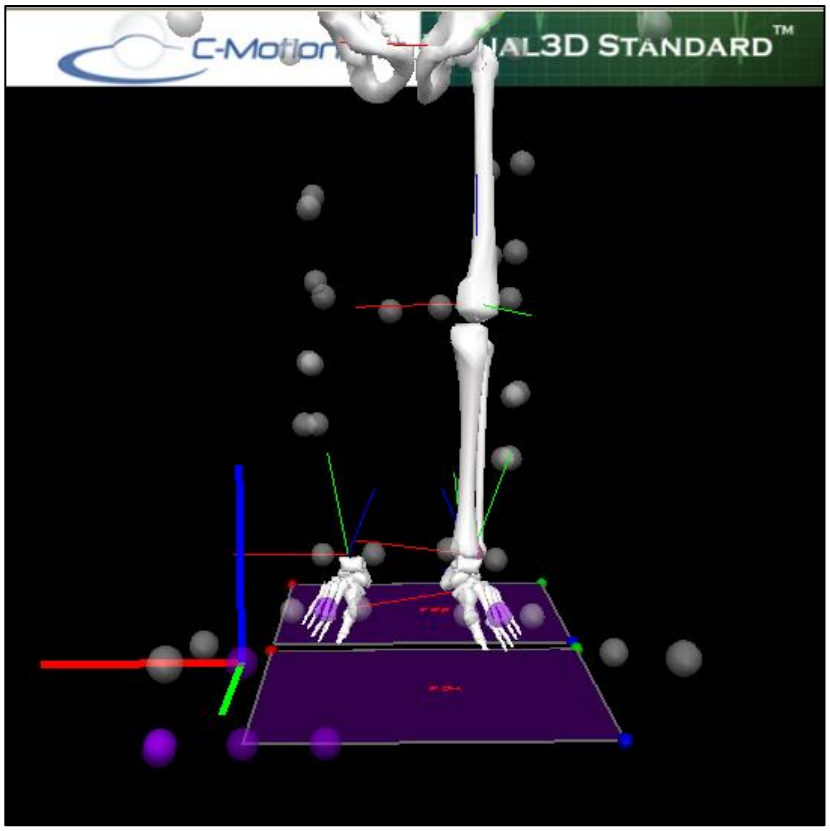


Figure 29: This screenshot shows an example with a missing right lateral knee marker in the static trial. There are no segments for the right thigh and right shank because it would require a right lateral knee marker to build these model segments. In the bottom left corner the segment coodinate system of the virtual lab is shown.

2. Projection of three markers to a virtual lab plane

It is necessary to project three markers from one segment to a virtual lab plane. In the example (missing lateral knee marker) three markers from the thigh or shank can be used because the lateral knee marker is a part of both segments. In this

instruction three markers of the shank (LMK, LMA, LLA) were projected to the frontal plane of the virtual lab.

Landmark Name	LMK_frontal plane
Starting Point	Lab Origin
Targets and/or Landmarks	*Checked*
Ending Point	Lab X
Lateral Object	Lab Z
Project From	LMK
Calibration Only Landmark	*Checked*

Landmark Name	LMA_frontal plane
Starting Point	Lab Origin
Targets and/or Landmarks	*Checked*
Ending Point	Lab X
Lateral Object	Lab Z
Project Form	LMA
Calibration Only Landmark	*Checked*

Landmark Name	LLA_frontal plane
Starting Point	Lab Origin
Targets and/or Landmarks	*Checked*
Ending Point	Lab X
Lateral Object	Lab Z
Project Form	LLA
Calibration Only Landmark	*Checked*

3. Create a virtual segment

Three markers of the shank were projected to the frontal plane of the virtual lab. Therefore, the next step is to create a kinematic only segment for the left virtual shank.

Define Proximal Joint and Radius

Lateral None Joint LMK_frontal plane Radius 0.01

Extra Target Joint and Radius

Lateral None Joint LMA_frontal plane Radius 0.01

Extra Target To Define Orientation (if needed)

Location Lateral LLA_frontal plane

Select Tracking Markers

LLA LMA LMK

Figure 30 shows all projected markers and the coordinate system of the left virtual shank.

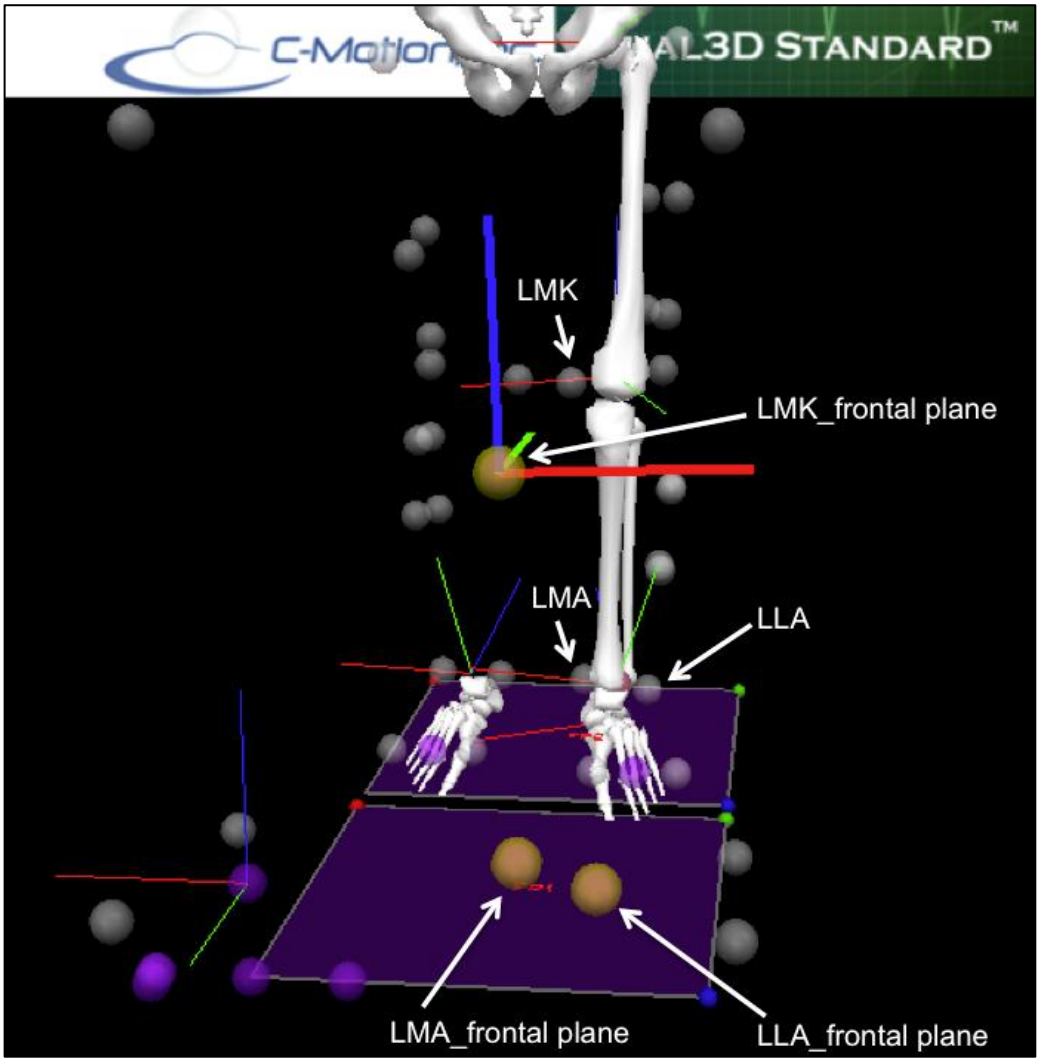


Figure 30: This modified screenshot shows all projected markers (LMA_frontal plane, LLA_frontal plane, and LMK_frontal plane) and there original markers (LMA, LLA, and LMK). The bold coordinate system with the LMK_frontal plane marker in its origin represents the coordinate system of the left virtual shank.

4. Same for the other limb

a. Projection of three markers from one segment to a virtual lab plane

The same markers from the same segment of the other limb are projected to the frontal plane of the virtual lab.

Landmark Name	RMK_frontal plane
Starting Point	Lab Origin
Targets and/or Landmarks	*Checked*

Ending Point	Lab X
Lateral Object	Lab Z
Project From	RMK
Calibration Only Landmark	*Checked*

Landmark Name	RMA_frontal plane
Starting Point	Lab Origin
Targets and/or Landmarks	*Checked*
Ending Point	Lab X
Lateral Object	Lab Z
Project Form	RMA
Calibration Only Landmark	*Checked*

Landmark Name	RLA_frontal plane
Starting Point	Lab Origin
Targets and/or Landmarks	*Checked*
Ending Point	Lab X
Lateral Object	Lab Z
Project Form	RLA
Calibration Only Landmark	*Checked*

b. <u>Creation of a virtual segment</u>

The projected markers can be used to create a kinematic only segment for the right virtual shank.

Define Proximal Joint and Radius

Lateral	None	Joint	RMK_frontal plane	Radius	0.01

Extra Target Joint and Radius

Lateral	None	Joint	RMA_frontal plane	Radius	0.01

Extra Target To Define Orientation (if needed)

Location Lateral RLA_frontal plane

Select Tracking Markers

RLA RMA RMK

Check if the coordinate system of the right virtual shank has the same alignment as in the left virtual shank.

5. Projection of a marker into all three planes of the virtual segment and calculation of the distances from the virtual segment to the marker

To be able to locate the missing marker, it is necessary to project the marker, which is equal to the missing marker but on the other limb, into all three planes of the virtual segment. Following landmarks and calculations serve to evulate the offset of the missing marker to the virtual shank segment:

Landmarks and calculations for the anterior distance

Landmark Name LLK_YZ

Starting Point LMK_frontal plane

Targets and/or Landmarks *Checked*

Ending Point LMA_frontal plane

Lateral Object LLA_frontal plane

Project Form LLK

Calibration Only Landmark *Checked*

Create a Subject Data/Metric Item

Name= LLK_AP

Value or Expression= DISTANCE(LLK,LLK_YZ)

Landmarks and calculations for the axial distance

Landmark Name LMK_lat

Starting Point	LMK
Existing Segment	*Checked*
Existing Segment	Left Virtual Shank
Offsets	ML=0.1 , AP=0 , AXIAL=0
Calibration Only Landmark	*Checked*

Landmark Name	LLK_XZ
Starting Point	LMK
Targets and/or Landmarks	*Checked*
Ending Point	LMK_frontal plane
Lateral Object	LMK_lat
Project From	LLK
Calibration Only Landmark	*Checked*

Create a Subject Data/Metric Item

Name= LLK_AXIAL
Value or Expression= DISTANCE(LLK,LLK_XZ)

<u>Landmarks and calculations for the medial-lateral distance</u>

Landmark Name	LMK_axial
Starting Point	LMK
Existing Segment	*Checked*
Existing Segment	Left Virtual Shank
Offsets	ML=0 , AP=0 , AXIAL=0.1
Calibration Only Landmark	*Checked*

Landmark Name	LLK_XY
Starting Point	LMK
Targets and/or Landmarks	*Checked*

Ending Point LMK_frontal plane

Lateral Object LMK_axial

Project From LLK

Calibration Only Landmark *Checked*

Create a Subject Data/Metric Item

Name= LLK_ML

Value or Expression= DISTANCE(LLK,LLK_XY)

6. Create landmark for the missing marker

With the values calsulated in step 5 it is possible to locate the RLK marker.

Landmark Name RLK

Starting Point None

Existing Segment *Checked*

Existing Segment Right Virtual Shank

Offsets ML=-LLK_ML , AP=LLK_AP , AXIAL=-LLK_AXIAL

Calibration Only Landmark *Checked*

Figure 31 shows the RLK landmark and the right thigh and shank model segment, which can be created now because all necessary markers are present.

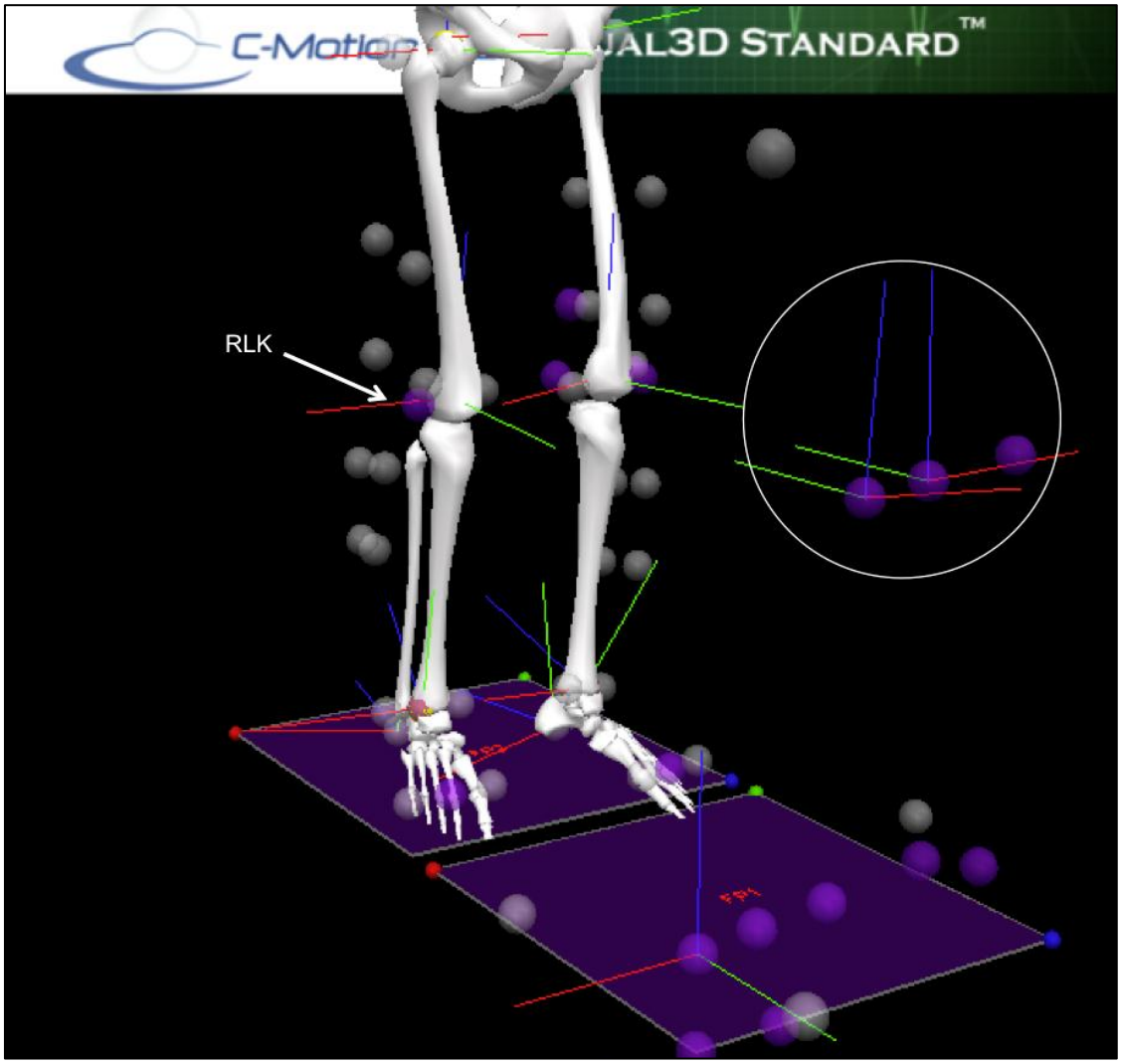


Figure 31: Modified screenshot with the RLK landmark, which leads to segments for the right thigh and right shank. Inside the white circle the coordinate systems for the left and right virtual shank are shown.

Conclusion:

It is possible to create a landmark for a missing marker in a static trial under the assumption that both limbs have the same anatomical geometry. In subjects, who got a joint replacement or had any similar surgery, it is not recommend to create a landmark for a missing marker.

D: Creation of a virtual marker in a dynamic trial

Creating a virtual marker in a dynamic trial is much easier than the creation of a virtual marker in a static trial because we can take the offset of the missing marker from the static trial and use this information for the definition of the virtual marker in the dynamic trial.
All this steps are explained in detail with following example:
The right lateral knee marker is missing in a dynamic trial. There is no model for the right thigh and right shank because it would require the right lateral knee marker.

To solve this problem we only have to create a new landmark with the name „RLK“, which requires following steps:

- Add new Landmark
- Landmark Name: RLK
- Starting Point: RSK 1
- Ending Point: RSK 2
- Lateral object: RSK 4
- Offset of Existing Caliration: RLK
- → Apply → Offset values will appear at the ML, AP and AXIAL boxes
- Check „Offset Using the Following ML/AP/AXIAL Offsets“
- Apply
- Save Model Template

E: Pelvis ROM pipeline

The calculation of the pelvis angle in space and the pelvis range of motion values in the sagittal, frontal and transversal plane were done with following pipeline:

```
Compute_Model_Based_Data
/RESULT_NAME=PELVIS_ANGLE_hk
/FUNCTION=JOINT_ANGLE
/SEGMENT=RPV
/REFERENCE_SEGMENT=Virtual Lab
/RESOLUTION_COORDINATE_SYSTEM=
! /USE_CARDAN_SEQUENCE=FALSE
! /NORMALIZATION=FALSE
! /NORMALIZATION_METHOD=
! /NORMALIZATION_METRIC=
! /NEGATEX=FALSE
! /NEGATEY=FALSE
! /NEGATEZ=FALSE
/AXIS1=Z
! /AXIS2=Y
/AXIS3=X
;

!LPelvis_ROM_(Tilt)_Calculation

Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MAX_Tilt
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LPelvis_ANGLE_MAX_Tilt
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MAX_Tilt
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
```

```
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MIN_Tilt
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LPelvis_ANGLE_MIN_Tilt
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MIN_Tilt
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LPelvis_ANGLE_MAX_Tilt+LPelvis_ANGLE_MIN_Tilt
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LPelvis_ROM_Tilt
! /RESULT_FOLDER=PROCESSED
;

!LPelvis_ROM_(Obliquity)_Calculation

Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MAX_Drop
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LPelvis_ANGLE_MAX_Drop
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MAX_Drop
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
```

```
! /RETAIN_NO_DATA_VALUES=FALSE
;

Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MIN_Drop
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LPelvis_ANGLE_MIN_Drop
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MIN_Drop
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LPelvis_ANGLE_MAX_Drop+LPelvis_ANGLE_MIN_Drop
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LPelvis_ROM_Drop
! /RESULT_FOLDER=PROCESSED
;

!LPelvis_ROM_(Rotation)_Calculation

Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MAX_Rot
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LPelvis_ANGLE_MAX_Rot
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
```

```
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MAX_Rot
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MIN_Rot
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LPelvis_ANGLE_MIN_Rot
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=PELVIS_ANGLE_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LPelvis_ANGLE_MIN_Rot
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LPelvis_ANGLE_MAX_Rot+LPelvis_ANGLE_MIN_Rot
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LPelvis_ROM_Rot
! /RESULT_FOLDER=PROCESSED
;
```

F: Thorax ROM pipeline

The calculation of the thorax angle in reference to the pelvis and the thorax range of motion values in the sagittal, frontal and transversal plane were done with following pipeline:

```
Compute_Model_Based_Data
/RESULT_NAME=Thorax_Pelvis_angle_hk
/FUNCTION=JOINT_ANGLE
/SEGMENT=RTA
/REFERENCE_SEGMENT=RPV
/RESOLUTION_COORDINATE_SYSTEM=
! /USE_CARDAN_SEQUENCE=FALSE
! /NORMALIZATION=FALSE
! /NORMALIZATION_METHOD=
! /NORMALIZATION_METRIC=
! /NEGATEX=FALSE
! /NEGATEY=FALSE
! /NEGATEZ=FALSE
! /AXIS1=X
! /AXIS2=Y
! /AXIS3=Z
;

!LThorax_ROM_(F-E)_Calculation

Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MAX_F-E
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LThorax_ANGLE_MAX_F-E
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MAX_F-E
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
```

```
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MIN_F-E
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LThorax_ANGLE_MIN_F-E
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MIN_F-E
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LThorax_ANGLE_MAX_F-E+LThorax_ANGLE_MIN_F-E
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LThorax_ROM_F-E
! /RESULT_FOLDER=PROCESSED
;

!LThorax_ROM_(AB-AD)_Calculation

Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MAX_AB-AD
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LThorax_ANGLE_MAX_AB-AD
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MAX_AB-AD
/GENERATE_MEAN_AND_STDDEV=FALSE
```

```
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MIN_AB-AD
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LThorax_ANGLE_MIN_AB-AD
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MIN_AB-AD
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LThorax_ANGLE_MAX_AB-AD+LThorax_ANGLE_MIN_AB-AD
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LThorax_ROM_AB-AD
! /RESULT_FOLDER=PROCESSED
;

!LThorax_ROM_(Rotation)_Calculation

Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MAX_Rot
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LThorax_ANGLE_MAX_Rot
! /RESULT_METRIC_FOLDER=PROCESSED
```

```
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MAX_Rot
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MIN_Rot
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;

Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LThorax_ANGLE_MIN_Rot
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=Thorax_Pelvis_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LThorax_ANGLE_MIN_Rot
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;

Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LThorax_ANGLE_MAX_Rot+LThorax_ANGLE_MIN_Rot
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LThorax_ROM_Rot
! /RESULT_FOLDER=PROCESSED
;
```

G: Foot progression ROM pipeline

The calculation of the foot progression angle in space and the foot progression angle range of motion values in the transversal plane were done with following pipeline:

```
Compute_Model_Based_Data
/RESULT_NAME=left_foot_progression_angle
/FUNCTION=JOINT_ANGLE
/SEGMENT=LMF
/REFERENCE_SEGMENT=Virtual Lab
/RESOLUTION_COORDINATE_SYSTEM=
! /USE_CARDAN_SEQUENCE=FALSE
! /NORMALIZATION=FALSE
! /NORMALIZATION_METHOD=
! /NORMALIZATION_METRIC=
! /NEGATEX=FALSE
! /NEGATEY=FALSE
! /NEGATEZ=FALSE
! /AXIS1=X
! /AXIS2=Y
! /AXIS3=Z
;
Compute_Model_Based_Data
/RESULT_NAME=right_foot_progression_angle
/FUNCTION=JOINT_ANGLE
/SEGMENT=RMF
/REFERENCE_SEGMENT=Virtual Lab
/RESOLUTION_COORDINATE_SYSTEM=
! /USE_CARDAN_SEQUENCE=FALSE
! /NORMALIZATION=FALSE
! /NORMALIZATION_METHOD=
! /NORMALIZATION_METRIC=
! /NEGATEX=FALSE
! /NEGATEY=FALSE
! /NEGATEZ=FALSE
! /AXIS1=X
! /AXIS2=Y
! /AXIS3=Z
;

!L_foot_progression_angle_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=left_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=L_left_foot_progression_angle_MAX
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
```

```
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=L_left_foot_progression_angle_MAX
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=left_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=L_left_foot_progression_angle_MAX
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=left_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=L_left_foot_progression_angle_MIN
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=L_left_foot_progression_angle_MIN
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=left_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=L_left_foot_progression_angle_MIN
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=L_left_foot_progression_angle_MAX+L_left_foot_progression_angle_M
IN
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=left_foot_progression_ROM
! /RESULT_FOLDER=PROCESSED
;

!R_foot_progression_angle_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=right_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=right_foot_progression_angle_MAX
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
```

```
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=right_foot_progression_angle_MAX
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=right_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=right_foot_progression_angle_MAX
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=right_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=right_foot_progression_angle_MIN
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=right_foot_progression_angle_MIN
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=right_foot_progression_angle
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=right_foot_progression_angle_MIN
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=right_foot_progression_angle_MAX+right_foot_progression_angle_MIN
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=right_foot_progression_angle_ROM
! /RESULT_FOLDER=PROCESSED
;
```

H: Ankle ROM pipeline

The calculation of the foot ankle angle (foot in reference to the shank) and the ankle range of motion values in the sagittal plane were done with following pipeline:

```
Compute_Model_Based_Data
/RESULT_NAME=L_virtual_foot_ankle_angle_hk
/FUNCTION=JOINT_ANGLE
/SEGMENT=LMF
/REFERENCE_SEGMENT=LSK
/RESOLUTION_COORDINATE_SYSTEM=
! /USE_CARDAN_SEQUENCE=FALSE
! /NORMALIZATION=FALSE
! /NORMALIZATION_METHOD=
! /NORMALIZATION_METRIC=
! /NEGATEX=FALSE
! /NEGATEY=FALSE
! /NEGATEZ=FALSE
! /AXIS1=X
! /AXIS2=Y
! /AXIS3=Z
;
Compute_Model_Based_Data
/RESULT_NAME=R_virtual_foot_ankle_angle_hk
/FUNCTION=JOINT_ANGLE
/SEGMENT=RMF
/REFERENCE_SEGMENT=RSK
/RESOLUTION_COORDINATE_SYSTEM=
! /USE_CARDAN_SEQUENCE=FALSE
! /NORMALIZATION=FALSE
! /NORMALIZATION_METHOD=
! /NORMALIZATION_METRIC=
! /NEGATEX=FALSE
! /NEGATEY=FALSE
! /NEGATEZ=FALSE
! /AXIS1=X
! /AXIS2=Y
! /AXIS3=Z
;
!LAnkle_ROM_sagittal plane_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
```

```
/SIGNAL_NAMES=L_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LANKLE_ANGLE_MAX_sag_hk
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LANKLE_ANGLE_MAX_sag_hk
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=L_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LANKLE_ANGLE_MAX_sag_hk
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=L_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LANKLE_ANGLE_MIN_sag_hk
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LANKLE_ANGLE_MIN_sag_hk
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=L_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LANKLE_ANGLE_MIN_sag_hk
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
```

```
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LANKLE_ANGLE_MAX_sag_hk+LANKLE_ANGLE_MIN_sag_hk
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LANKLE_ROM_sag_hk
! /RESULT_FOLDER=PROCESSED
;
!RAnkle_ROM_sagittal plane_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=R_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RANKLE_ANGLE_MAX_sag_hk
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RANKLE_ANGLE_MAX_sag_hk
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=R_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RANKLE_ANGLE_MAX_sag_hk
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=R_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RANKLE_ANGLE_MIN_sag_hk
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=RHS
```

```
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RANKLE_ANGLE_MIN_sag_hk
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=R_virtual_foot_ankle_angle_hk
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RANKLE_ANGLE_MIN_sag_hk
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=RANKLE_ANGLE_MAX_sag_hk+RANKLE_ANGLE_MIN_sag_hk
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=RANKLE_ROM_sag_hk
! /RESULT_FOLDER=PROCESSED
;
```

I: Knee ROM pipeline

The calculation of the knee angle (shank in reference to the thigh) and the knee range of motion values in the sagittal, frontal and transversal plane were done with following pipeline:

```
!LKnee_ROM_sag_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MAX_sag
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LKNEE_ANGLE_MAX_sag
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MAX_sag
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MIN_sag
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LKNEE_ANGLE_MIN_sag
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MIN_sag
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
```

```
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LKNEE_ANGLE_MAX_sag+LKNEE_ANGLE_MIN_sag
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LKNEE_ROM_sag
! /RESULT_FOLDER=PROCESSED
;
!RKnee_ROM_sag_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MAX_sag
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RKNEE_ANGLE_MAX_sag
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MAX_sag
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MIN_sag
/SELECT_X=TRUE
! /SELECT_Y=FALSE
! /SELECT_Z=FALSE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RKNEE_ANGLE_MIN_sag
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MIN_sag
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=RKNEE_ANGLE_MAX_sag+RKNEE_ANGLE_MIN_sag
/SIGNAL_FOLDER=PROCESSED
```

```
/RESULT_NAME=RKNEE_ROM_sag
! /RESULT_FOLDER=PROCESSED
;
!LKnee_ROM_fro_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MAX_fro
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LKNEE_ANGLE_MAX_fro
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MAX_fro
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MIN_fro
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LKNEE_ANGLE_MIN_fro
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MIN_fro
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LKNEE_ANGLE_MAX_fro+LKNEE_ANGLE_MIN_fro
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LKNEE_ROM_fro
! /RESULT_FOLDER=PROCESSED
;
```

```
!RKnee_ROM_fro_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MAX_fro
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RKNEE_ANGLE_MAX_fro
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MAX_fro
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MIN_fro
! /SELECT_X=FALSE
/SELECT_Y=TRUE
! /SELECT_Z=FALSE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RKNEE_ANGLE_MIN_fro
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MIN_fro
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=RKNEE_ANGLE_MAX_fro+RKNEE_ANGLE_MIN_fro
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=RKNEE_ROM_fro
! /RESULT_FOLDER=PROCESSED
;
!LKnee_ROM_tra_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
```

```
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MAX_tra
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LKNEE_ANGLE_MAX_tra
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MAX_tra
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MIN_tra
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=LHS
/END_AT_EVENT=LHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=LKNEE_ANGLE_MIN_tra
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=LKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=LKNEE_ANGLE_MIN_tra
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=LKNEE_ANGLE_MAX_tra+LKNEE_ANGLE_MIN_tra
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=LKNEE_ROM_tra
! /RESULT_FOLDER=PROCESSED
;
!RKnee_ROM_tra_Calculation
Event_Global_Maximum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MAX_tra
```

```
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RKNEE_ANGLE_MAX_tra
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MAX_tra
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Event_Global_Minimum
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MIN_tra
! /SELECT_X=FALSE
! /SELECT_Y=FALSE
/SELECT_Z=TRUE
/START_AT_EVENT=RHS
/END_AT_EVENT=RHS
;
Metric_Signal_Value_At_Event
/RESULT_METRIC_NAME=RKNEE_ANGLE_MIN_tra
! /RESULT_METRIC_FOLDER=PROCESSED
/SIGNAL_TYPES=LINK_MODEL_BASED
/SIGNAL_NAMES=RKNEE_ANGLE
! /SIGNAL_FOLDER=ORIGINAL
/EVENT_NAME=RKNEE_ANGLE_MIN_tra
/GENERATE_MEAN_AND_STDDEV=FALSE
! /APPEND_TO_EXISTING_VALUES=FALSE
! /GENERATE_VECTOR_LENGTH_METRIC=FALSE
! /RETAIN_NO_DATA_VALUES=FALSE
;
Subtract_Signals
/SIGNAL_TYPES=METRIC+METRIC
/SIGNAL_NAMES=RKNEE_ANGLE_MAX_tra+RKNEE_ANGLE_MIN_tra
/SIGNAL_FOLDER=PROCESSED
/RESULT_NAME=RKNEE_ROM_tra
! /RESULT_FOLDER=PROCESSED
;
```

J: Unified Parkinson's Disease Rating Scale (UPDRS)

Detailed description of the UPDRS: [52]

I. MENTATION, BEHAVIOR AND MOOD

1. Intellectual Impairment
0 = None.
1 = Mild. Consistent forgetfulness with partial recollection of events and no other difficulties.
2 = Moderate memory loss, with disorientation and moderate difficulty handling complex problems. Mild but definite impairment of function at home with need of occasional prompting.
3 = Severe memory loss with disorientation for time and often to place. Severe impairment in handling problems.
4 = Severe memory loss with orientation preserved to person only. Unable to make judgements or solve problems. Requires much help with personal care. Cannot be left alone at all.

2. Thought Disorder (Due to dementia or drug intoxication)
0 = None.
1 = Vivid dreaming.
2 = "Benign" hallucinations with insight retained.
3 = Occasional to frequent hallucinations or delusions; without insight; could interfere with daily activities.
4 = Persistent hallucinations, delusions, or florrid psychosis. Not able to care for self.

3. Depression
0 = None.
1 = Periods of sadness or guilt greater than normal, never sustained for days or weeks.
2 = Sustained depression (1 week or more).
3 = Sustained depression with vegetative symptoms (insomnia, anorexia, weight loss, loss of interest).
4 = Sustained depression with vegetative symptoms and suicidal thoughts or intent.

4. Motivation/Initiative
0 = Normal.
1 = Less assertive than usual; more passive.
2 = Loss of initiative or disinterest in elective (nonroutine) activities.
3 = Loss of initiative or disinterest in day to day (routine) activities.
4 = Withdrawn, complete loss of motivation.

II. ACTIVITIES OF DAILY LIVING

5. Speech
0 = Normal.
1 = Mildly affected. No difficulty being understood.

2 = Moderately affected. Sometimes asked to repeat statements.
3 = Severely affected. Frequently asked to repeat statements.
4 = Unintelligible most of the time.

6. Salivation
0 = Normal.
1 = Slight but definite excess of saliva in mouth; may have nighttime drooling.
2 = Moderately excessive saliva; may have minimal drooling.
3 = Marked excess of saliva with some drooling.
4 = Marked drooling, requires constant tissue or handkerchief.

7. Swallowing
0 = Normal.
1 = Rare choking.
2 = Occasional choking.
3 = Requires soft food.
4 = Requires NG tube or gastrotomy feeding.

8. Handwriting
0 = Normal.
1 = Slightly slow or small.
2 = Moderately slow or small; all words are legible.
3 = Severely affected; not all words are legible.
4 = The majority of words are not legible.

9. Cutting food and handling utensils
0 = Normal.
1 = Somewhat slow and clumsy, but no help needed.
2 = Can cut most foods, although clumsy and slow; some help needed.
3 = Food must be cut by someone, but can still feed slowly.
4 = Needs to be fed.

10. Dressing
0 = Normal.
1 = Somewhat slow, but no help needed.
2 = Occasional assistance with buttoning, getting arms in sleeves.
3 = Considerable help required, but can do some things alone.
4 = Helpless.

11. Hygiene
0 = Normal.
1 = Somewhat slow, but no help needed.
2 = Needs help to shower or bathe; or very slow in hygienic care.
3 = Requires assistance for washing, brushing teeth, combing hair, going to bathroom.
4 = Foley catheter or other mechanical aids.

12. Turning in bed and adjusting bed clothes
0 = Normal.
1 = Somewhat slow and clumsy, but no help needed.

2 = Can turn alone or adjust sheets, but with great difficulty.
3 = Can initiate, but not turn or adjust sheets alone.
4 = Helpless.

13. Falling (unrelated to freezing)
0 = None.
1 = Rare falling.
2 = Occasionally falls, less than once per day.
3 = Falls an average of once daily.
4 = Falls more than once daily.

14. Freezing when walking
0 = None.
1 = Rare freezing when walking; may have starthesitation.
2 = Occasional freezing when walking.
3 = Frequent freezing. Occasionally falls from freezing.
4 = Frequent falls from freezing.

15. Walking
0 = Normal.
1 = Mild difficulty. May not swing arms or may tend to drag leg.
2 = Moderate difficulty, but requires little or no assistance.
3 = Severe disturbance of walking, requiring assistance.
4 = Cannot walk at all, even with assistance.

16. Tremor
Symptomatic complaint of tremor in any part of body.
0 = Absent.
1 = Slight and infrequently present.
2 = Moderate; bothersome to patient.
3 = Severe; interferes with many activities.
4 = Marked; interferes with most activities.

17. Sensory complaints related to parkinsonism
0 = None.
1 = Occasionally has numbness, tingling, or mild aching.
2 = Frequently has numbness, tingling, or aching; not distressing.
3 = Frequent painful sensations.
4 = Excruciating pain.

III. MOTOR EXAMINATION

18. Speech
0 = Normal.
1 = Slight loss of expression, diction and/or volume.
2 = Monotone, slurred but understandable; moderately impaired.
3 = Marked impairment, difficult to understand.
4 = Unintelligible.

19. Facial Expression
0 = Normal.
1 = Minimal hypomimia, could be normal "Poker Face".
2 = Slight but definitely abnormal diminution of facial expression.
3 = Moderate hypomimia; lips parted some of the time.
4 = Masked or fixed facies with severe or complete loss of facial expression; lips parted 1/4 inch or more.

20. Tremor at rest
(head, upper and lower extremities)
0 = Absent.
1 = Slight and infrequently present.
2 = Mild in amplitude and persistent. Or moderate in amplitude, but only intermittently present.
3 = Moderate in amplitude and present most of the time.
4 = Marked in amplitude and present most of the time.

21. Action or Postural Tremor of hands
0 = Absent.
1 = Slight; present with action.
2 = Moderate in amplitude, present with action.
3 = Moderate in amplitude with posture holding as well as action.
4 = Marked in amplitude; interferes with feeding.

22. Rigidity
Judged on passive movement of major joints with patient relaxed in sitting position. Cogwheeling to be ignored.
0 = Absent.
1 = Slight or detectable only when activated by mirror or other movements.
2 = Mild to moderate.
3 = Marked, but full range of motion easily achieved.
4 = Severe, range of motion achieved with difficulty.

23. Finger Taps
Patient taps thumb with index finger in rapid succession.
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

24. Hand Movements
Patient opens and closes hands in rapid succesion.
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in

ongoing movement.
4 = Can barely perform the task.

25. Rapid Alternating Movements of Hands
Pronation-supination movements of hands, vertically and horizontally, with as large an amplitude as possible, both hands simultaneously.
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

26. Leg Agility
Patient taps heel on the ground in rapid succession picking up entire leg. Amplitude should be at least 3 inches.
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

27. Arising from Chair
Patient attempts to rise from a straightbacked chair, with arms folded across chest.
0 = Normal.
1 = Slow; or may need more than one attempt.
2 = Pushes self up from arms of seat.
3 = Tends to fall back and may have to try more than one time, but can get up without help.
4 = Unable to arise without help.

28. Posture
0 = Normal erect.
1 = Not quite erect, slightly stooped posture; could be normal for older person.
2 = Moderately stooped posture, definitely abnormal; can be slightly leaning to one side.
3 = Severely stooped posture with kyphosis; can be moderately leaning to one side.
4 = Marked flexion with extreme abnormality of posture.

29. Gait
0 = Normal.
1 = Walks slowly, may shuffle with short steps, but no festination (hastening steps) or propulsion.
2 = Walks with difficulty, but requires little or no assistance; may have some festination, short steps, or propulsion.
3 = Severe disturbance of gait, requiring assistance.

4 = Cannot walk at all, even with assistance.

30. Postural Stability
Response to sudden, strong posterior displacement produced by pull on shoulders while patient erect with eyes open and feet slightly apart. Patient is prepared.
0 = Normal.
1 = Retropulsion, but recovers unaided.
2 = Absence of postural response; would fall if not caught by examiner.
3 = Very unstable, tends to lose balance spontaneously.
4 = Unable to stand without assistance.

31. Body Bradykinesia and Hypokinesia
Combining slowness, hesitancy, decreased armswing, small amplitude, and poverty of movement in general.
0 = None.
1 = Minimal slowness, giving movement a deliberate character; could be normal for some persons. Possibly reduced amplitude.
2 = Mild degree of slowness and poverty of movement which is definitely abnormal. Alternatively, some reduced amplitude.
3 = Moderate slowness, poverty or small amplitude of movement.
4 = Marked slowness, poverty or small amplitude of movement.

IV. COMPLICATIONS OF THERAPY (In the past week)

A. DYSKINESIAS

32. Duration: What proportion of the waking day are dyskinesias present?
Historical information.
0 = None
1 = 1-25% of day.
2 = 26-50% of day.
3 = 51-75% of day.
4 = 76-100% of day.

33. Disability: How disabling are the dyskinesias?
Historical information; may be modified by office examination.
0 = Not disabling.
1 = Mildly disabling.
2 = Moderately disabling.
3 = Severely disabling.
4 = Completely disabled.

34. Painful Dyskinesias: How painful are the dyskinesias?
0 = No painful dyskinesias.
1 = Slight.
2 = Moderate.
3 = Severe.
4 = Marked.

35. Presence of Early Morning Dystonia (Historical information)
0 = No
1 = Yes

B. CLINICAL FLUCTUATIONS

36. Are "off" periods predictable?
0 = No
1 = Yes

37. Are "off" periods unpredictable?
0 = No
1 = Yes

38. Do "off" periods come on suddenly, within a few seconds?
0 = No
1 = Yes

39. What proportion of the waking day is the patient "off" on average?
0 = None
1 = 1-25% of day.
2 = 26-50% of day.
3 = 51-75% of day.
4 = 76-100% of day.

C. OTHER COMPLICATIONS

40. Does the patient have anorexia, nausea, or vomiting?
0 = No
1 = Yes

41. Any sleep disturbances, such as insomnia or hypersomnolence?
0 = No
1 = Yes

42. Does the patient have symptomatic orthostasis?
Record the patient's blood pressure, height and weight on the scoring form.
0 = No
1 = Yes

K: Gait ROM raw data of the PD study

Table 18: Pre and post hip ROM data [°] from all PD subjects. Green values represent improvements (closer to ROM data from normal subjects) and red values represent deteriorations.

Patient	Sagittal plane				Frontal plane				Transversal plane			
	Left		Right		Left		Right		Left		Right	
	pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
PD28	32.9	32.7	32.3	32.5	9.4	9.2	9.9	9.6	9.0	9.5	9.2	9.4
PD27	26.2	27.5	26.5	28.9	7.3	8.9	7.7	9.0	13.1	14.2	15.3	14.5
PD26	34.5	34.6	38.1	39.6	10.4	10.1	9.0	8.3	12.7	14.0	10.9	11.0
PD25	23.8	26.1	24.0	25.6	4.4	4.6	5.6	4.3	10.6	10.7	11.7	7.9
PD23	43.2	44.9	36.6	39.8	13.9	14.3	12.6	12.6	15.6	17.8	17.5	16.7
PD22	33.4	34.9	30.1	30.9	6.7	7.1	8.6	7.5	9.3	11.1	13.0	12.9
PD21	35.0	34.3	35.1	35.2	5.6	5.8	11.1	12.0	13.5	12.5	9.2	9.7
PD20	32.5	34.4	27.1	28.9	5.0	6.2	5.5	5.3	10.3	10.6	7.8	7.7
PD18	46.2	45.4	45.1	45.8	6.4	7.0	10.7	11.7	14.4	15.4	16.5	20.3
PD17	24.8	26.0	24.8	27.1	9.4	9.2	7.9	9.5	14.6	15.9	11.2	14.0
PD15	42.6	41.7	35.2	35.6	9.2	8.3	11.8	11.5	11.9	11.1	9.3	10.5
PD14	38.0	39.0	40.2	39.3	13.0	13.2	11.7	11.9	13.5	14.5	12.7	12.0
PD12	42.7	42.4	47.2	48.8	8.6	9.5	9.0	10.3	10.7	8.1	7.2	8.4
PD11	40.9	39.0	37.1	37.8	10.0	10.7	10.0	12.2	11.9	11.1	9.2	10.8
PD07	29.8	32.0	30.9	33.4	10.9	7.8	10.2	9.6	15.3	20.9	12.4	16.0
PD06	29.7	32.9	30.8	34.2	5.3	5.2	7.5	8.5	8.4	9.5	6.6	9.4
PD04	31.7	31.9	27.1	29.9	6.5	7.9	5.4	4.9	13.0	11.1	12.1	12.4

Table 19: Pre and post pelvis ROM data [°] from all PD subjects. Green values represent improvements (closer to ROM data from normal subjects) and red values represent deteriorations.

Patient	Pre-Tilt	Post-Tilt	Pre-Drop	Post-Drop	Pre-Rot	Post-Rot
PD28	2.5	5.9	7.9	9.1	8.6	6.9
PD27	2.4	2.4	3.7	4.2	7.2	7.6
PD26	2.6	3.4	4.3	4.8	7.3	7.7
PD25	4.0	4.1	2.8	2.4	11.2	11.8
PD23	4.8	4.4	5.8	6.9	11.0	9.8
PD22	3.7	3.2	2.8	2.9	9.4	9.0
PD21	3.2	3.4	4.6	4.9	6.4	7.0
PD20	2.0	2.2	2.1	3.0	8.5	8.6
PD18	2.9	4.0	3.0	3.6	6.3	5.1
PD17	2.3	2.1	2.4	2.5	4.8	4.0
PD15	5.8	5.0	5.7	4.9	12.0	11.8
PD14	1.9	2.0	8.2	5.9	13.6	11.2
PD12	2.7	3.0	5.2	5.3	8.7	8.0
PD11	3.6	3.4	7.0	8.4	9.6	9.7
PD07	3.7	3.8	6.0	2.7	6.1	3.7

PD06	2.3	2.3	1.8	2.5	5.6	5.3
PD04	4.3	3.3	4.4	5.0	9.3	8.0

Table 20: Pre and post torso ROM data [°] from all PD subjects. Green values represent improvements (closer to ROM data from normal subjects), blue values represent no change (same difference to ROM data from normal subjects) and red values represent deteriorations.

Patient	Pre-sag	Post-sag	Pre-fro	Post-fro	Pre-tra	Post-tra
PD28	3.6	6.3	7.2	8.9	6.8	5.4
PD27	3.1	2.7	3.4	4.5	4.8	4.9
PD26	2.4	2.6	3.3	4.5	3.4	4.1
PD25	2.3	2.8	1.3	2.3	1.9	3.0
PD23	2.2	1.6	5.8	6.4	2.8	4.3
PD22	2.4	1.8	2.8	2.2	3.7	3.2
PD21	2.3	3.2	5.2	4.9	2.4	3.0
PD20	3.0	2.6	0.9	1.6	2.4	3.4
PD18	2.5	3.5	5.6	4.9	3.7	5.2
PD17	1.7	2.2	2.0	1.6	3.2	3.0
PD15	6.1	5.8	5.3	4.7	6.7	7.3
PD14	3.8	3.2	7.7	5.3	7.2	3.8
PD12	2.3	2.3	1.4	3.8	3.1	4.7
PD11	4.7	3.7	2.9	4.4	4.2	2.7
PD07	4.2	2.8	6.6	3.3	7.5	3.9
PD06	2.3	2.6	2.1	2.2	1.4	2.7
PD04	4.7	3.1	3.7	4.7	4.2	5.1

Table 21: Hip, pelvis and torso ROM data [°] from all normal (healthy) subjects.

Subject	Hip						Pelvis			Torso		
	Sagittal plane		Frontal plane		Transversal plane		Sag. plane	Fro. plane	Tra. plane	Sag. plane	Fro. plane	Tra. plane
	left	right	left	right	left	right						
Norm04	40.2	41.4	8.8	11.9	5.8	10.1	3.3	4.6	9.8	2.7	3.5	4.0
Norm05	47.0	45.7	13.1	13.9	12.3	19.8	2.2	3.6	6.6	2.5	3.6	4.8
Norm06	37.2	32.7	12.7	8.3	9.5	6.7	2.3	2.6	8.4	2.2	1.2	1.4
Norm07	28.2	27.2	11.5	12.4	12.1	10.8	2.5	2.7	6.7	2.5	0.8	1.9
Norm08	44.2	36.0	12.3	12.0	9.3	7.6	1.9	6.5	3.8	4.4	1.8	2.7
Norm09	41.7	38.5	14.1	11.9	15.3	13.8	3.1	4.4	5.3	2.7	3.1	2.2
Norm10	45.4	41.5	10.4	9.5	15.5	13.2	6.3	4.8	6.3	6.9	3.9	5.0
Norm11	37.6	39.1	13.4	7.6	9.0	9.4	2.8	3.7	7.2	4.9	6.4	8.8
Norm12	36.1	39.9	11.0	10.7	12.6	8.8	2.1	7.5	10.5	3.9	5.0	4.4
Norm13	43.5	40.4	9.8	7.6	13.1	12.9	3.1	5.9	13.2	3.3	2.4	3.8
Norm15	46.6	46.2	12.5	12.8	15.0	14.0	1.9	7.3	11.2	2.6	4.6	3.4
Norm16	36.0	32.4	10.6	11.5	10.9	11.6	2.9	5.1	5.2	2.9	7.0	4.3
Norm17	38.7	40.4	13.2	10.2	12.2	7.8	2.6	5.1	5.9	3.3	3.4	3.2

L: Raw spatiotemporal gait parameters of the PD study

Averages	Speed [m/s]	Stride wide [m]	Stride length [m]	Cycle time [s]	Double limb support time [s]
PD04_pre	0.84	0.10	0.93	1.11	0.25
PD04_post	0.91	0.09	0.95	1.04	0.21
PD 06_pre	0.89	0.12	0.98	1.10	0.27
PD 06_post	0.99	0.11	1.05	1.06	0.24
PD 07_pre	0.97	0.14	0.97	1.00	0.28
PD 07_post	1.07	0.15	1.05	0.98	0.24
PD 11_pre	1.06	0.10	1.31	1.24	0.30
PD 11_post	1.08	0.09	1.32	1.22	0.26
PD 12_pre	1.50	0.12	1.28	0.85	0.14
PD 12_post	1.57	0.11	1.32	0.84	0.11
PD 14_pre	1.25	0.08	1.38	1.11	0.24
PD 14_post	1.22	0.07	1.36	1.12	0.25
PD 15_pre	1.37	0.11	1.43	1.04	0.20
PD 15_post	1.34	0.10	1.40	1.04	0.22
PD 17_pre	0.91	0.12	1.01	1.10	0.25
PD 17_post	0.93	0.12	1.04	1.12	0.23
PD 18_pre	1.16	0.11	1.28	1.11	0.23
PD 18_post	1.16	0.12	1.30	1.12	0.24
PD 20_pre	0.82	0.09	0.96	1.16	0.32
PD 20_post	0.95	0.09	1.00	1.06	0.28
PD 21_pre	1.03	0.11	1.26	1.23	0.27
PD 21_post	1.03	0.09	1.27	1.23	0.28
PD 22_pre	0.98	0.10	1.01	1.03	0.32
PD 22_post	0.95	0.09	1.00	1.06	0.28
PD 23_pre	1.12	0.12	1.26	1.12	0.25
PD 23_post	1.14	0.11	1.27	1.12	0.24
PD 25_pre	0.99	0.14	1.02	1.03	0.28
PD 25_post	1.12	0.14	1.09	0.98	0.21
PD 26_pre	1.19	0.12	1.19	1.00	0.21
PD 26_post	1.17	0.11	1.18	1.01	0.21
PD 27_pre	1.01	0.10	1.04	1.02	0.22
PD 27_post	1.08	0.09	1.11	1.03	0.20
PD 28_pre	1.18	0.11	1.17	0.99	0.25
PD 28_post	1.15	0.11	1.13	0.99	0.27
Norm04	1.30	0.10	1.27	0.98	0.21
Norm05	1.17	0.12	1.26	1.08	0.24
Norm06	1.00	0.14	1.05	1.05	0.30
Norm07	1.00	0.10	0.99	0.99	0.22
Norm08	1.13	0.09	1.14	1.01	0.23
Norm09	1.08	0.09	1.17	1.08	0.24

Norm10	1.15	0.11	1.29	1.12	0.25
Norm11	1.29	0.13	1.38	1.07	0.21
Norm12	1.16	0.09	1.25	1.07	0.24
Norm13	1.13	0.07	1.29	1.13	0.22
Norm15	1.35	0.06	1.37	1.02	0.18
Norm16	1.03	0.10	1.19	1.15	0.33
Norm17	1.27	0.08	1.22	0.96	0.21

Averages	Left Step Length [m]	Right Step Length [m]	Left Step Time [s]	Right Step Time [s]	Left Stance Time [s]	Right Stance Time [s]	Left Swing Time [s]	Right Swing Time [s]
PD04_pre	0.47	0.45	0.55	0.55	0.69	0.66	0.43	0.44
PD04_post	0.49	0.47	0.52	0.52	0.63	0.62	0.41	0.42
PD 06_pre	0.50	0.48	0.57	0.53	0.69	0.68	0.41	0.42
PD 06_post	0.53	0.52	0.54	0.52	0.65	0.65	0.41	0.41
PD 07_pre	0.50	0.46	0.50	0.50	0.65	0.62	0.37	0.36
PD 07_post	0.56	0.49	0.49	0.48	0.61	0.61	0.37	0.37
PD 11_pre	0.65	0.66	0.61	0.63	0.77	0.76	0.47	0.48
PD 11_post	0.66	0.66	0.62	0.60	0.73	0.74	0.49	0.48
PD 12_pre	0.62	0.66	0.43	0.43	0.50	0.49	0.35	0.36
PD 12_post	0.66	0.66	0.42	0.42	0.49	0.46	0.36	0.38
PD 14_pre	0.70	0.68	0.57	0.53	0.67	0.67	0.44	0.43
PD 14_post	0.68	0.68	0.57	0.55	0.68	0.68	0.44	0.44
PD 15_pre	0.71	0.72	0.52	0.52	0.63	0.61	0.41	0.43
PD 15_post	0.71	0.68	0.53	0.51	0.64	0.62	0.40	0.42
PD 17_pre	0.52	0.49	0.54	0.56	0.67	0.67	0.42	0.44
PD 17_post	0.54	0.50	0.55	0.57	0.68	0.67	0.44	0.44
PD 18_pre	0.66	0.62	0.57	0.53	0.66	0.69	0.45	0.42
PD 18_post	0.66	0.63	0.57	0.55	0.66	0.68	0.45	0.44
PD 20_pre	0.48	0.48	0.60	0.56	0.74	0.75	0.43	0.41
PD 20_post	0.48	0.52	0.53	0.53	0.68	0.66	0.38	0.39
PD 21_pre	0.64	0.62	0.61	0.61	0.75	0.75	0.48	0.48
PD 21_post	0.64	0.63	0.62	0.61	0.75	0.75	0.48	0.49
PD 22_pre	0.47	0.54	0.53	0.50	0.68	0.67	0.35	0.36
PD 22_post	0.48	0.51	0.53	0.52	0.67	0.66	0.39	0.39
PD 23_pre	0.60	0.65	0.55	0.58	0.70	0.67	0.43	0.45
PD 23_post	0.62	0.65	0.55	0.57	0.69	0.66	0.43	0.46
PD 25_pre	0.52	0.50	0.52	0.51	0.68	0.62	0.34	0.41
PD 25_post	0.56	0.53	0.48	0.49	0.59	0.59	0.38	0.39
PD 26_pre	0.57	0.62	0.50	0.50	0.61	0.60	0.39	0.39
PD 26_post	0.57	0.61	0.51	0.50	0.61	0.60	0.40	0.40
PD 27_pre	0.51	0.53	0.52	0.51	0.62	0.62	0.40	0.40

PD 27_post	0.56	0.56	0.52	0.51	0.62	0.61	0.41	0.41
PD 28_pre	0.58	0.59	0.48	0.50	0.62	0.62	0.37	0.37
PD 28_post	0.57	0.56	0.49	0.50	0.63	0.63	0.36	0.36
Norm04	0.63	0.65	0.49	0.48	0.59	0.59	0.38	0.38
Norm05	0.62	0.64	0.55	0.53	0.65	0.67	0.42	0.41
Norm06	0.52	0.54	0.51	0.54	0.69	0.65	0.37	0.39
Norm07	0.47	0.51	0.49	0.49	0.60	0.60	0.38	0.38
Norm08	0.56	0.58	0.50	0.50	0.62	0.62	0.39	0.39
Norm09	0.57	0.60	0.55	0.53	0.65	0.67	0.42	0.41
Norm10	0.67	0.62	0.56	0.57	0.69	0.68	0.43	0.44
Norm11	0.67	0.71	0.53	0.54	0.63	0.64	0.43	0.43
Norm12	0.64	0.61	0.55	0.52	0.65	0.66	0.43	0.40
Norm13	0.63	0.66	0.57	0.57	0.68	0.68	0.46	0.46
Norm15	0.71	0.67	0.50	0.52	0.61	0.59	0.41	0.43
Norm16	0.59	0.60	0.57	0.59	0.75	0.73	0.40	0.43
Norm17	0.62	0.60	0.49	0.47	0.58	0.59	0.38	0.37

M: Reach test ROM raw data of the PD study

Table 22: Pre and post reach test hip ROM data [°] from all PD subjects. Green values represent improvements (closer to ROM data from normal subjects) and red values represent deteriorations.

Patient	Sagittal plane				Frontal plane			
	Left		Right		Left		Right	
	pre	post	pre	post	pre	post	pre	post
PD28	57.2	50.4	58.9	52.9	30.7	32.0	26.9	27.3
PD27	30.9	37.5	33.2	38.4	8.1	7.0	11.7	9.8
PD26	30.7	29.1	34.1	32.8	12.3	11.9	12.1	11.7
PD25	9.3	13.8	9.3	16.4	8.4	3.9	7.2	4.2
PD23	34.5	52.9	38.2	47.7	21.0	32.6	21.4	32.0
PD22	49.3	56.2	50.5	54.9	13.5	15.5	14.3	18.5
PD21	57.4	29.0	57.9	30.8	13.6	8.5	9.8	7.3
PD20	39.6	28.3	37.4	29.5	18.2	14.7	13.8	13.0
PD18	23.1	30.8	20.5	27.1	11.3	13.1	13.1	12.6
PD17	41.5	54.0	46.9	56.9	21.1	23.6	20.7	21.7
PD15	40.5	37.6	40.2	36.1	29.1	16.3	21.6	18.5
PD14	46.8	49.3	48.5	52.6	7.4	12.2	8.0	11.7
PD12	78.1	61.7	77.1	60.1	20.5	11.8	20.8	11.3
PD11	34.3	38.8	28.3	39.8	12.4	20.9	9.9	15.6
PD07	61.7	60.8	62.1	68.7	21.5	31.6	21.5	33.8
PD06	36.4	30.6	39.6	33.1	5.1	7.4	5.1	7.8
PD04	20.2	25.0	24.1	25.5	2.2	3.2	2.7	3.5

Table 23: Pre and post reach test pelvis ROM data [°] from all PD subjects. Green values represent improvements (closer to ROM data from normal subjects) and red values represent deteriorations.

Patient	Pre-Tilt	Post-Tilt	Pre-Drop	Post-Drop
PD28	61.7	50.4	19.8	23.1
PD27	41.0	42.6	8.1	9.0
PD26	41.4	39.6	14.3	11.3
PD25	19.5	23.0	10.9	8.3
PD23	52.8	61.1	32.0	38.8
PD22	49.3	56.6	14.5	19.0
PD21	55.3	36.4	12.2	10.0
PD20	46.2	35.2	16.1	12.4
PD18	27.5	33.7	17.7	18.9
PD17	55.4	59.1	28.4	27.2
PD15	50.6	48.7	27.8	26.6
PD14	43.5	44.9	14.6	18.7
PD12	69.8	53.0	18.6	7.6
PD11	37.3	47.4	20.0	31.4

PD07	43.0	54.5	17.0	27.0
PD06	38.2	32.7	4.3	5.3
PD04	31.2	41.2	5.7	7.7

Table 24: Pre and post reach test torso ROM data [°] from all PD subjects. Green values represent improvements (closer to ROM data from normal subjects) and red values represent deteriorations.

Patient	Pre-sag	Post-sag	Pre-fro	Post-fro
PD28	26.6	50.9	30.6	28.5
PD27	7.9	11.2	4.8	7.9
PD26	11.8	13.4	6.8	9.9
PD25	7.0	14.3	5.7	9.3
PD23	22.0	38.3	16.1	19.0
PD22	24.8	26.9	7.1	6.7
PD21	14.0	13.9	10.0	12.0
PD20	18.7	22.1	9.9	10.8
PD18	6.2	11.2	5.4	8.1
PD17	8.6	11.7	6.7	7.4
PD15	25.2	22.6	12.5	14.2
PD14	18.3	10.8	9.5	6.0
PD12	39.0	24.7	13.3	20.7
PD11	17.9	16.1	7.9	4.6
PD07	33.1	23.9	13.7	22.8
PD06	5.9	16.6	5.6	10.8
PD04	12.2	15.2	5.3	13.6

N: Change in reach test ROM of the PD study

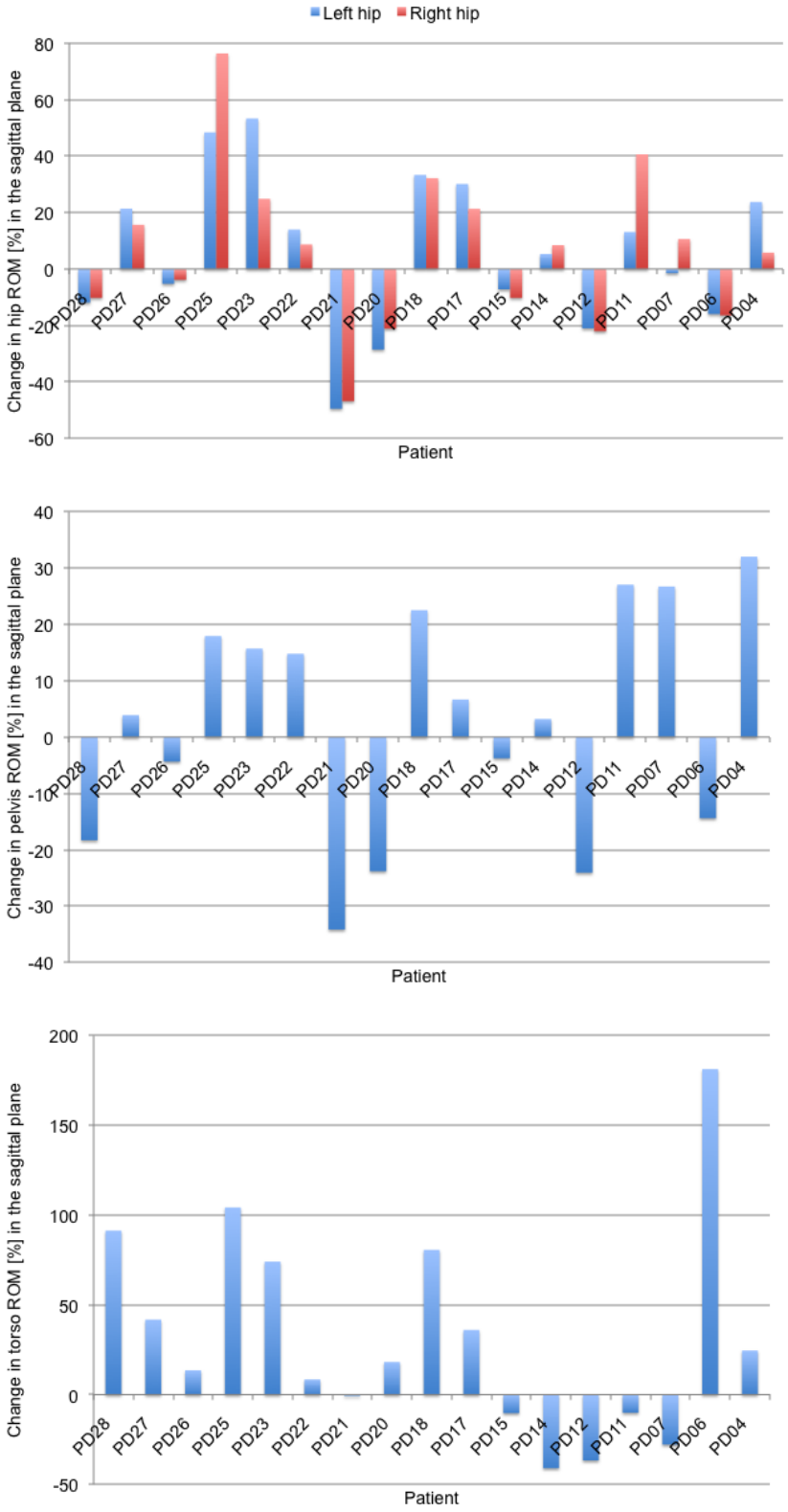


Figure 32: Change in hip, pelvis and torso ROM [%] in the sagittal plane. Positve columns represent improvements in ROM and negative columns represent deteriorations in ROM.

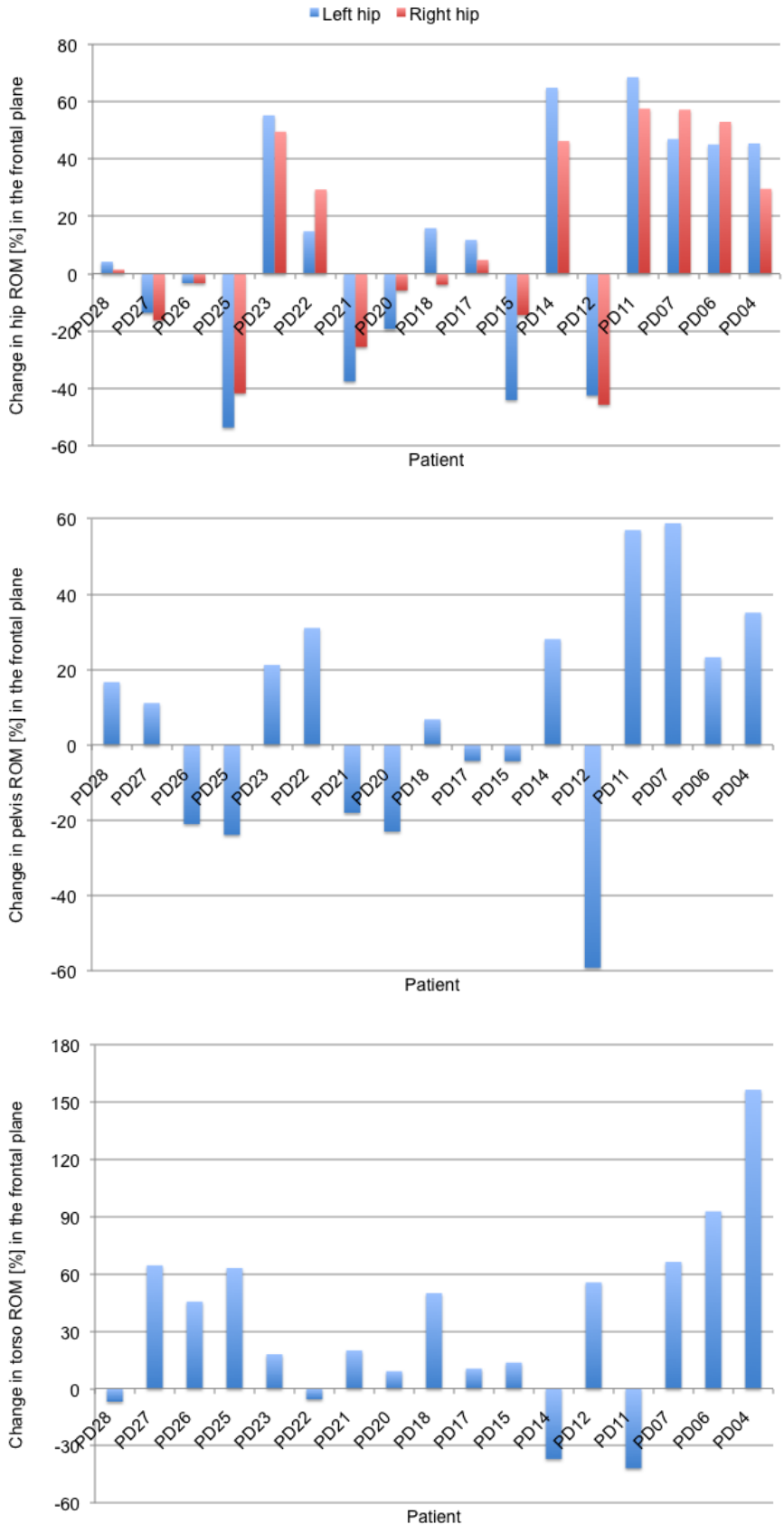


Figure 33: Change in hip, pelvis and torso ROM [%] in the frontal plane. Positve columns represent improvements in ROM and negative columns represent deteriorations in ROM.

O: Gait raw data of the stroke study

Table 25: Hip and knee ROM data [°] for all participants. Group 1=body weight supported training group, group 2=conventional training group, group 3=control group, sag=sagittal plane, fro=frontal plane, and tra=transverse plane.

Subject	Group	affected hip ROM			non-affected hip ROM			affected knee ROM			non-affected knee ROM		
		sag	fro	tra	sag	fro	tra	sag	fro	tra	sag	fro	tra
Str2	1	28,6	6,7	15,1	42,6	8,3	17,9	25,3	7,7	20,3	51,9	9,3	14,6
Str4	1	26,7	7,0	5,9	28,9	10,0	6,4	47,0	6,6	16,8	45,9	4,4	11,2
Str6	1	26,2	8,9	10,6	25,5	6,0	11,7	41,1	7,4	12,2	41,5	12,0	14,1
Str8	1	28,9	9,6	5,9	31,4	7,5	8,0	42,2	6,3	11,2	56,0	5,0	12,0
Str3	2	25,4	6,6	11,2	26,4	4,9	10,3	41,5	6,3	13,1	35,9	6,2	14,2
Str7	2	35,1	8,7	8,8	37,7	8,6	15,0	29,8	7,4	12,4	52,2	7,4	11,1
Str10	2	16,9	10,7	12,2	34,9	9,7	10,2	41,0	11,4	13,7	42,4	8,3	7,9
Str11	2	33,4	10,7	8,1	38,5	13,9	10,0	46,2	8,2	21,7	51,4	9,1	11,9
Str12	2	28,0	10,6	6,3	35,1	9,7	12,2	49,4	8,8	13,8	53,4	6,2	14,8
Nor04	3				40,8	10,4	7,9				64,5	7,4	19,9
Nor05	3				46,4	13,5	16,1				58,6	8,1	18,2
Nor06	3				34,9	10,5	8,1				57,8	11,4	13,3
Nor07	3				28,6	12,3	11,7				49,0	9,7	16,4
Nor08	3				40,1	12,1	8,4				62,2	13,5	20,2
Nor09	3				40,1	13,0	14,5				62,1	12,1	17,3
Nor10	3				43,4	10,0	14,4				64,8	10,3	22,5
Nor11	3				38,4	10,5	9,2				63,8	8,1	12,4
Nor12	3				38,0	10,8	10,7				59,4	7,0	11,8
Nor13	3				41,9	8,7	13,0				63,4	5,7	12,0
Nor15	3				46,3	12,5	14,3				66,6	9,4	22,8
Nor16	3				34,2	11,1	11,2				58,5	8,5	17,7
Nor17	3				39,7	11,3	10,0				59,8	7,9	16,1

Table 26: Foot progression, torso, and pelvis ROM data [°] for all participants. Group 1=body weight supported training group, group 2=conventional training group, group 3=control group, sag=sagittal plane, fro=frontal plane, and tra=transverse plane.

Subject	Group	Foot progression ROM		Torso ROM			Pelvis ROM		
		affected	non-affected	sag	fro	tra	sag	fro	tra
Str2	1	6,1	9,3	7,3	1,6	3,6	7,7	6,1	8,0
Str4	1	8,4	8,0	2,4	5,1	3,3	3,1	4,1	7,0
Str6	1	8,2	14,1	3,1	1,9	2,1	5,6	3,4	8,5
Str8	1	6,5	10,7	2,8	1,7	2,5	3,6	3,4	10,4
Str3	2	8,0	7,3	4,2	2,5	3,6	3,3	5,5	3,1
Str7	2	12,5	11,7	5,7	4,0	5,2	12,3	7,4	13,6
Str10	2	8,4	16,0				11,9	6,6	16,2
Str11	2	6,6	20,4	8,3	6,7	6,5	8,4	9,6	11,6
Str12	2	9,0	9,2	4,5	6,3	3,3	7,5	6,6	9,9
Nor04	3		9,0	2,7	3,5	4,0	3,3	4,6	9,8

Nor05	3		13,2		3,6	4,8	2,2	3,6	6,6
Nor06	3		12,2	2,2	1,2	1,4	2,3	2,6	8,4
Nor07	3		13,5	2,6	0,9	2,0	2,7	2,9	7,1
Nor08	3		7,7	4,4	1,8	2,7	1,9	6,5	3,8
Nor09	3		12,0	2,7	3,1	2,2	3,1	4,4	5,3
Nor10	3		14,2	6,9	3,9	5,0	6,3	4,8	6,3
Nor11	3		10,6	4,9	6,4	8,8	2,8	3,7	7,2
Nor12	3		13,0		5,0	4,4	2,1	7,5	10,5
Nor13	3		12,3	3,3	2,4	3,8	3,1	5,9	13,2
Nor15	3		15,0		4,6	3,3	1,9	7,2	10,9
Nor16	3		27,9	2,9	7,0	4,3	2,9	5,1	5,2
Nor17	3		15,7	3,3	3,4	3,2	2,6	5,1	5,9

Table 27: Spatiotemporal gait data for all participants. Subj=Subject, Gr=Group, Group 1=body weight supported training group, group 2=conventional training group, group 3=control group, Sp=Speed, Str wid=Stride wide, Str len=Stride length, Cyc time=Cycle time, Aff=Affected side, Not aff=Not affected side, and DLS time=double limb support time.

Subj	Gr	Sp [m/s]	Str wid [m]	Str len [m]	Cyc time [s]	Step length [m]		Step time [s]		Stance time [s]		Swing time [s]		DLS time [s]
						Aff	Not aff	Aff	Not aff	Aff	Not aff	Aff	Not aff	
Str2	1	0,51	0,20	0,86	1,67	0,41	0,45	0,86	0,80	1,18	1,22	0,51	0,44	0,70
Str4	1	0,28	0,13	0,62	2,24	0,30	0,32	1,16	1,10	1,64	1,67	0,57	0,60	1,09
Str6	1	0,26	0,12	0,55	2,09	0,35	0,20	1,28	0,83	1,53	1,68	0,56	0,40	1,15
Str8	1	0,54	0,17	0,90	1,67	0,47	0,43	0,84	0,82	1,13	1,19	0,55	0,47	0,64
Str3	2	0,40	0,16	0,54	1,35	0,31	0,23	0,64	0,68	1,01	1,00	0,34	0,35	0,62
Str7	2	0,27	0,13	0,71	2,64	0,38	0,32	1,52	1,14	1,86	2,20	0,81	0,42	1,50
Str10	2	0,14	0,13	0,36	2,51	0,07	0,29	1,78	0,69	1,55	2,27	0,92	0,27	1,31
Str11	2	0,32	0,13	0,86	2,73	0,41	0,46	1,49	1,21	1,80	2,19	0,87	0,60	1,23
Str12	2	0,38	0,15	0,76	1,98	0,39	0,37	1,06	0,92	1,41	1,50	0,55	0,49	0,95
Nor04	3	1,30	0,10	1,27	0,98		0,64		0,49		0,59		0,38	0,21
Nor05	3	1,17	0,12	1,26	1,08		0,63		0,54		0,66		0,42	0,24
Nor06	3	1,00	0,14	1,05	1,05		0,53		0,53		0,67		0,38	0,30
Nor07	3	1,00	0,10	0,99	0,99		0,49		0,49		0,60		0,38	0,22
Nor08	3	1,13	0,09	1,14	1,01		0,57		0,50		0,62		0,39	0,23
Nor09	3	1,08	0,09	1,17	1,08		0,58		0,54		0,66		0,42	0,24
Nor10	3	1,15	0,11	1,29	1,12		0,64		0,57		0,69		0,44	0,25
Nor11	3	1,29	0,13	1,38	1,07		0,69		0,54		0,64		0,43	0,21
Nor12	3	1,16	0,09	1,25	1,07		0,62		0,54		0,66		0,42	0,24
Nor13	3	1,13	0,07	1,29	1,13		0,64		0,57		0,68		0,46	0,22
Nor15	3	1,34	0,06	1,37	1,02		0,69		0,51		0,59		0,43	0,16
Nor16	3	1,03	0,10	1,19	1,15		0,60		0,58		0,74		0,42	0,33
Nor17	3	1,27	0,08	1,22	0,96		0,61		0,48		0,59		0,38	0,21

Table 28: Differences in gait parameters between the left and right limb. Gr=Group, Group 1=body weight supported training group, group 2=conventional training group, group 3=control group, Step len=Step length, Sta time=Stance time, Swi time=Swing time, sag=sagittal plane, fro=frontal plane, tra=transverse plane, Foot progr=Foot progression.

Subject	Gr	Step len [m]	Sta time [s]	Swi time [s]	Hip ROM [°]			Knee ROM [°]			Foot progr [°]
					sag	fro	tra	sag	fro	tra	
Str2	1	0,04	0,04	0,07	13,9	1,6	2,7	26,6	1,5	5,6	3,3
Str4	1	0,02	0,03	0,03	2,2	3,0	0,5	1,1	2,2	5,6	0,5
Str6	1	0,15	0,15	0,16	0,8	3,0	1,1	0,4	4,6	1,9	5,9
Str8	1	0,04	0,06	0,08	2,5	2,0	2,1	13,8	1,3	0,7	4,3
Str3	2	0,08	0,01	0,01	1,03	1,7	0,7	5,6	0,1	1,2	0,7
Str7	2	0,05	0,34	0,39	2,6	0,1	6,2	22,4	0,0	1,2	0,8
Str10	2	0,23	0,72	0,65	17,9	1,0	2,0	1,4	3,1	5,8	7,6
Str11	2	0,05	0,39	0,27	5,1	3,2	2,0	5,2	0,8	9,7	13,8
Str12	2	0,02	0,09	0,06	7,1	0,9	5,9	4,0	2,6	1,0	0,2
Nor04	3	0,02	0,00	0,00	1,2	3,1	4,3	1,9	2,9	7,3	0,5
Nor05	3	0,01	0,02	0,01	1,3	0,8	7,5	1,6	2,5	2,2	3,3
Nor06	3	0,02	0,04	0,02	4,5	4,4	2,8	0,0	0,3	2,7	1,7
Nor07	3	0,03	0,00	0,00	2,8	0,2	2,2	0,8	0,5	0,8	5,1
Nor08	3	0,03	0,00	0,00	8,1	0,3	1,7	3,2	3,0	0,2	0,8
Nor09	3	0,03	0,02	0,01	3,3	2,2	1,5	0,3	2,3	5,7	1,6
Nor10	3	0,06	0,01	0,01	3,8	0,8	2,3	1,4	3,4	7,9	1,8
Nor11	3	0,03	0,01	0,00	1,5	5,8	0,4	2,7	2,5	0,2	3,0
Nor12	3	0,03	0,01	0,03	3,8	0,3	3,7	8,4	3,2	1,4	2,5
Nor13	3	0,03	0,00	0,00	3,1	2,1	0,2	1,3	0,8	3,1	0,5
Nor15	3	0,05	0,00	0,00	0,8	0,1	1,5	2,4	1,1	3,1	0,6
Nor16	3	0,00	0,02	0,03	3,5	0,8	0,6	3,2	1,4	0,7	1,0
Nor17	3	0,02	0,01	0,01	1,5	3,0	3,5	3,3	6,4	7,8	9,4