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# 3D gait analysis of lower extremity muscle group power in healthy subjects and subacute stroke patients, and task-specific gait interventions in early stroke rehabilitation

PhD dissertation

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## 1. Introduction

There has been an increase in the number of clinical and scientific 3D gait analysis systems worldwide using biomechanical models for describing the movements (kinematics) and forces applied to produce these movements (kinetics) in patients and healthy subjects. This makes further understanding of pathological and healthy gait possible (1). Stroke is the third leading cause of death in Denmark (approx. 10 % of all deaths per year) with 11000 persons hospitalised due to stroke per year (2;3). Two thirds of hospitalised stroke patients are reported to suffer from a leg hemiparesis at stroke onset (4;5), and approximately 66 % of hospitalised patients are still dependent walkers at discharge (4;5). Therefore, achieving independent walking and “normal” gait appearance are of a high priority for stroke patients during goal setting in rehabilitation (6). Suitable gait analysis reference data may be a valuable tool to compare impaired variables to healthy variables. In addition, it might assist therapists in choosing appropriate gait training strategies when treating pathological gait patterns; e.g. training hip flexor muscles to increase gait speed (if this is the problem) and reaching “normal” values. In determining reference data, the most relevant gait analysis variables must be determined and considered in relation to the multiple possibilities. In particular, work has been performed, in which the peak power of ankle plantarflexors (A2-S), hip extensors (H1-S), hip flexors (H3-S), hip abductors (H3-F), and knee extensors (K3-S) have been suggested to be strongly correlated to walking speed in chronic stroke patients (7-13). This is also well established in healthy subjects (14;15). Only one study has estimated reference data in power values but did not include reference intervals, which reflect the variability in the muscle power of healthy subjects (16). These variables are associated with the demographic parameters of gender, age, and height in healthy subjects (17-20). Therefore, reference data and intervals were investigated. It was hypothesised that gender, age and height could partly determine A2-S, H1-S, H3-S, H3-F and K3-S in healthy subjects (study 1).

In doing so, appropriate estimation methods of a mean peak power are required. Usually, standard settings in gait analysis systems provide a mean power curve (MPC) based on individual power curves, and defines the mean peak power (MPP) from the MPC. Despite this, MPC will underestimate MPP compared to a MPP based on power peaks from each single power curve (SPC), because differences in the time to peak power (TTP) and difference in configuration are observed in individual power curves (1;21). Consequently, this diminishes the peak power identified on the MPC (phase cancellation). To my knowledge, no studies have investigated this methodological problem, so it was hypothesised that MPP based on MPC would underestimate MPP compared

to SPC for A2-S, H1-S, H3-S, H3-F, and K3-S in subacute stroke patients and healthy subjects. Secondly, it was hypothesised that the variability in TTP for the power variables A2-S, H1-S, H3-S, H3-F and K3-S will correlate negatively with walking speed in subacute stroke patients (study 2).

In addition, to target gait rehabilitation in subacute stroke patients it is important that future work seek to explain changes in gait performance as well as gait appearance (i.e. increased gait speed) in stroke patients. Only two studies have investigated changes in power and work variables following an intervention for chronic stroke patients (12;13). Improvements in power were reported in impaired and unimpaired plantarflexors (A2-S) and in unimpaired hip flexor (H3-S) when gait speed increased (0.69 to 0.83 m/s) (12). Knowledge of this may assist clinicians to identify pathological gait patterns and target gait rehabilitation. Even so, power analyses of subacute stroke patients with impaired gait performance has not been investigated in the same manner, and are required to target gait training in this subgroup during early rehabilitation. It was hypothesised, that (1) A2-S, H1-S, H3-S, H3-F and K3-S in the impaired and unimpaired lower extremity would improve in subacute stroke patients where gait speed increases, (2) improvements in power variables will correlate to changes in walking speed, and (3) changes in power strategies of the patient during gait rehabilitation will be similar to the power strategies of healthy matched subjects when walking speed increases (study 3).

Recently, robotic gait training has been introduced as a new gait training approach in stroke rehabilitation favouring exercises of symmetrical “normal” gait patterns with many repetitions and prolonged intensive training sessions. Unlike body weight supported treadmill training (BWSTT) and conventional overground physical therapy, it reduces the comprehensive work load of physical therapists during the management of gait training. Although, gait training combining robotic training and conventional physical therapy improves gait independence in non-ambulatory stroke patients (22-24), robotic training alone seems no better than conventional physical therapy or BWSTT when gait speed are measured (22). In addition, Hidler et al. (2008) and Hornby et al. (2008) reported that conventional physical therapy and BWSTT, respectively, improved gait speed and endurance when compared to Lokomat<sup>®</sup> training (25;26). Although the Lokomat<sup>®</sup> releases the physical therapist from a comprehensive work load during gait training, are there any additional benefits of Lokomat<sup>®</sup> training in stroke patients? Based on the nature of the set-up and function of the Lokomat<sup>®</sup>, and considering the asymmetrical gait pattern in most stroke patients (27-29), gait quality (i.e. measured as gait symmetry) is suggested to be the goal of Lokomat<sup>®</sup> training. It was

hypothesised that Lokomat<sup>®</sup> training would improve gait symmetry compared to gait training conducted by a physical therapist (study 4).

The overall aim of this PhD thesis was to utilize gait analyses to estimate reference data for power generation and absorption in major lower extremity muscle groups during gait, and to identify the power determinants of gait speed improvements in subacute patients with low walking performance during early gait rehabilitation. In addition, the effect of Lokomat<sup>®</sup> training is investigated based on gait qualities. Hopefully, the findings will contribute to more targeted gait training in a subgroup of stroke patients during early rehabilitation. The thesis is based on four papers.

Study 1: "Gender, age and height are of minor importance in determining power based on gait analysis in healthy subjects" (*Submitted to Gait & Posture*)

Study 2: "Inconsistent timing of peak power in paretic hip extensors and abductors are negatively correlated to walking speed in subacute stroke patients"

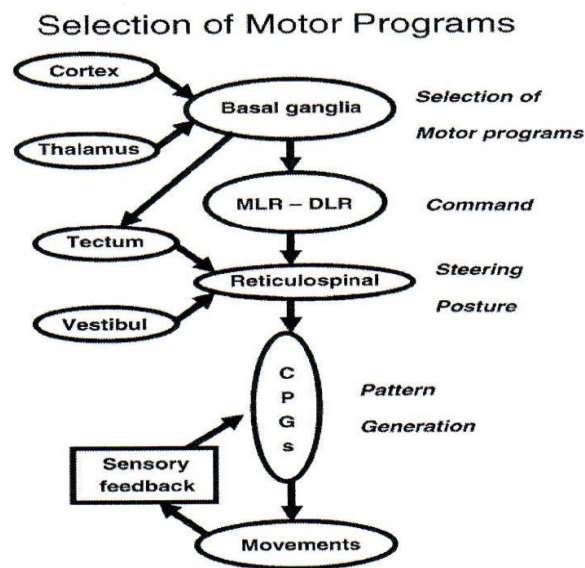
Study 3: "Increased power generation in impaired lower extremities strongly correlate to changes in walking speed in subacute stroke patients" (*Submitted to Clinical Biomechanics*)

Study 4: "The order of combined gait training, including Lokomat<sup>®</sup> and physical therapy, do not influence gait quality in subacute ambulatory stroke patients – A pilot study" (*Submitted to Physiotherapy Theory and Practise*)

## 2. Background

### 2.1. Neural locomotion control systems

The neural control systems of locomotion are complex and involve several neuro-anatomical structures (figure 1), and the precise cortical, subcortical, and spinal control mechanisms are not fully understood in humans. Most of our knowledge in human locomotion control systems are gained from animal studies, i.e. mice, rats, cats, lampreys (30-34), but even so, it is assumed that common basic features of the control systems regarding locomotion exist in humans and animals (e.g. vertebrates, lampreys) (30;33).

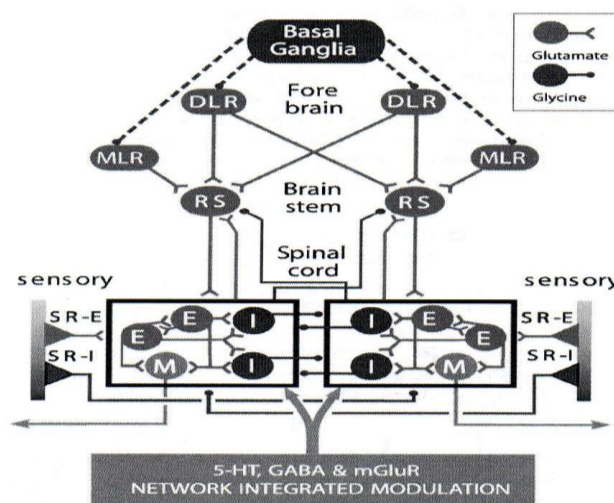


**Fig. 1 – Subsystems involved in the control of goal-directed locomotion. Selection of a motor program is performed in the basal ganglia, which receives inputs from the cortex (pallium) and the thalamus. The basal ganglia output stage (pallidum) inhibits command centres in the diencephalic locomotor region (DLR) and the mesencephalic locomotor region (MLR) during resting conditions. Through a well-controlled inhibition of pallidal regions, the spinal CPG for locomotion can be activated via the reticulospinal (RS) neurons. In the brainstem, information is further integrated based on visual, sensory and vestibular inputs to control both steering and posture. In all vertebrates, the spinal cord CPG neurons are modulated by local sensory feedback.**

Figure 1. Adapted from Grillner, S et al. Neural bases of goal-directed locomotion in vertebrates-An overview. Brain Res Rev. 2008; (57):2-12.

It has been suggested in animal studies, that a central pattern generator (CPG) network in the spinal cord generates the basic locomotor rhythm and synergies which are modulated by sensory input and descended input from the brainstem (31-34). It is proposed, that the CPG controls the basic rhythm of gait by complex interactions between excitatory and inhibitory interneurons in the spinal cord. Excitatory interneurons activate ipsilateral motoneurons in the spinal cord and the in-

hibitory interneurons cross the midline to the contralateral side to inhibit interneurons and motoneurons. This mechanism is modulated by the sensory input from the sensory receptors in the muscle (Ia and II afferents) and tendon receptors (Ib afferents), which is excitatory on ipsilateral side and inhibitory on contralateral side (figure 2). This sensory input is also suggested to control and modulate the CPG system (34;35), and is proposed to be important in gait rehabilitation; e.g. in patients with spinal cord injuries (35). Furthermore, CPGs are most likely subdivided into CPG units in each limb, by which one muscle group (hip flexor, hip extensor, knee flexor etc.) is governed and coordinated by one unit. These CPG interactions may control intralimb and interlimb coordination of the basic locomotion system (31;33).



**Fig. 6 – Locomotor network of the lamprey. Schematic representation of the forebrain, brainstem and spinal components of the neural circuitry that generates rhythmic locomotor activity. All neuron symbols denote populations rather than single cells. The reticulospinal (RS), glutamatergic neurons excite all classes of spinal interneurons and motoneurons. The excitatory interneurons (E) excite all types of spinal neurons, i.e. the inhibitory glycinergic interneurons (I) that cross the midline to inhibit all neuron types on the contralateral side and the motoneurons (M). The stretch receptor neurons are of two types; one excitatory (SR-E), which excites ipsilateral neurons and one inhibitory (SR-I), which crosses the midline to inhibit contralateral neurons. RS neurons receive excitatory synaptic input from the diencephalic and the mesencephalic locomotor regions (DLR and MLR), which in turn receive input from the basal ganglia as well as visual and olfactory input. In addition, metabotropic receptors are also activated during locomotion and are an integral part of the network (5-HT, GABA and mGluR).**

Figure 2. Adapted from Grillner, S et al. Neural bases of goal-directed locomotion in vertebrates-An overview. Brain Res Rev. 2008; (57):2-12.

It is assumed in the human that the CPG is initiated by locomotor regions in the brainstem, mesencephalon, (MLR) and a diencephalic locomotor region (DLR). The signals from MLR and DLR are mediated through reticulospinal neurons (RS) (within the reticulospinal tract) to the spinal cord. In addition, the RS are influenced by postural and visual receptors (located in the tectum and



vestibul) to allow postural control and the sense of orientation in space. These regions are directly and indirectly under the control of the basal ganglia, which play a central part in the selection of i.e. locomotor programs. The MLR and DLR do not “command” the CPG to generate its characteristic gait rhythm without an excitatory input from the basal ganglia (“gate keeper function”). To initiate selection of motor programs a disinhibition of the basal ganglia is required and generated by the motor cortex and thalamus, in which voluntary motor actions are planned. Subcortical motor control, including coordination of the motor plan by cerebellum, is sustained so long as there is no dramatic change in the environment (30-33) (figure 1). If obstacles appear, a new locomotor plan must be generated by the supraspinal motor centres (assumable Brodmann areas 4,5 and 6) and integrated into the subcortical and spinal locomotor systems (30;31). Even though, motor programs are stored, and movements are conducted by subcortical regions of the brain (basal ganglia, brainstem and spinal cord) all movements can be controlled “at will” by the motor cortex (32). The motor cortex is capable of influencing movements of the extremities directly (corticospinal tracts) as well as indirectly (e.g. reticulospinal tracts) at all levels of the central nervous system (30).

Acute stroke often reduces mobility as a result of neurological problems such as motor weakness (hemiparesis), sensory, ataxic, apraxic, and visospatial deficits (36). In the present thesis, stroke patients who experienced a middle cerebral artery infarction (mainstem, deep perforating arteries, or lacunar), which resulted in locomotor deficits were included. It is assumed, that motor corticospinal tracts and sensory pathways in the internal capsule are affected as well as the representation of areas in the motor cortex of the leg (Brodmann area 4) (36). Consequently, it is assumed that the neural locomotor control system is affected with respect to locomotor initiation, selection and execution. Also, Luft et. al. (2008) reported an increased activity in subcortical networks (cerebellum and midbrain) following treadmill training in chronic stroke patients. It was proposed that the subcortical locomotor networks (brainstem, cerebellum, and spinal cord) are modulated during gait training (37). Forrester et. al. (2006) reported no increase in transcranial magnetic stimulation-induced excitability to quadriceps by stimulating its motor cortical area following treadmill training (38). By contrast, Yen et. al. (2008) reported that BWSTT induced corticomotor excitability with regards to the tibialis anterior and abductor hallucis in the unimpaired and impaired limb, respectively (39). This indicates that locomotor recovery might be mediated by increased activity in the cortical and subcortical networks of chronic stroke patients during treadmill training.

## 2.2. *Gait characteristics in healthy subjects*

Initially, a description will be presented displaying the gait characteristics in healthy subjects, and following this, in hemiparetic stroke patients (see 3.4. *Gait characteristics in hemiparetic stroke patients*) and the relationship of spatiotemporal and power variables. These are the primary gait parameters in this thesis, and therefore a description of the kinematic parameters will not appear. However, as the primary parameters are dependent on kinematic parameters they will be discussed when required.

Gait consists of gait cycles (GC), and conventionally, gait characteristics are defined in relation to the GC. The GC is defined as heel strike to heel strike in ipsilateral extremity, and is normalised to 100 percent (%). The GC is divided into a stance (from heel strike to toe off in ipsilateral limb) and swing (from toe off to heel strike in ipsilateral limb) and they can be divided further into three periods related to tasks during gait; (1) weight acceptance, (2) single limb support and (3) limb advancement. The total stance (TS) phase occupies 60 % of the GC and swing phase occupies 40 % during preferred walking speeds (40;41). Additionally, gait can be separated into seven phases. Stance consists of initially the double stance in which initial contact and loading response are present (10 % of GC; from heel strike in ipsilateral limb to toe off in contralateral limb), single limb stance includes mid stance and terminal stance (40 % of GC; from toe off in contralateral limb to heel strike in contralateral limb), and the second double stance phase represents the pre swing phase (10 % of GC; from heel strike in contralateral limb to toe off in ipsilateral limb). The last 40 % of GC is divided into initial swing (13 % of GC) mid-swing (14 % of GC) and terminal swing (13% of GC) (figure 3) (42). Gait characteristics are present in both extremities, and consequently, the gait pattern is symmetrical in healthy subjects.

**Table 2.1**

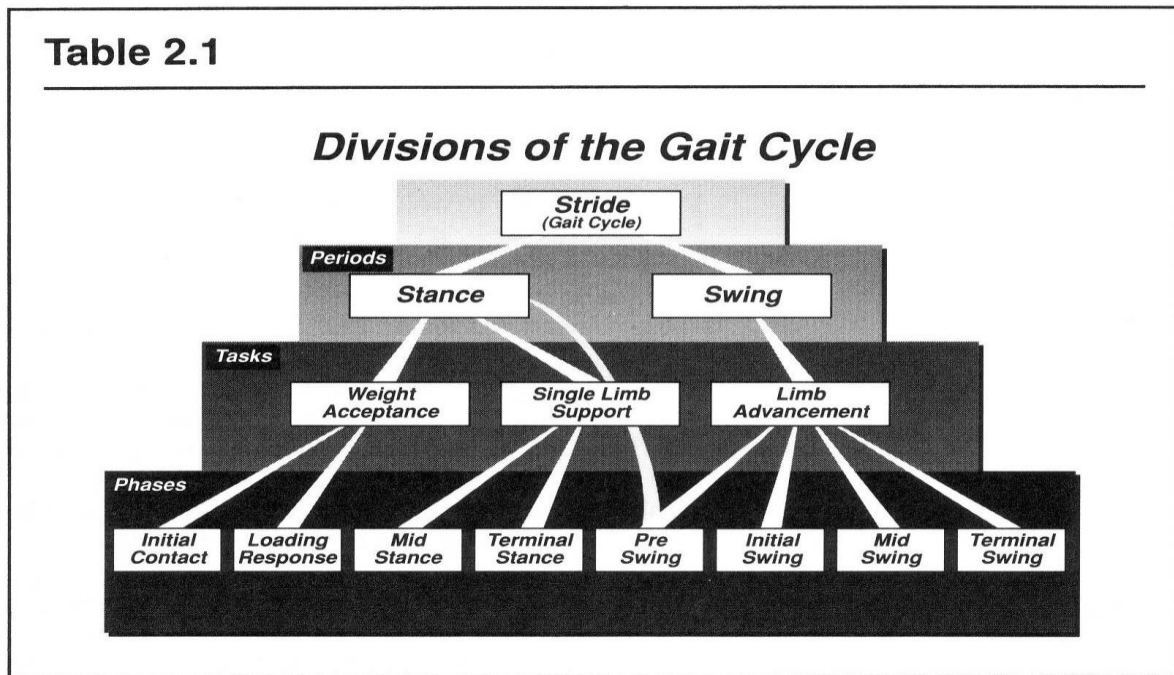


Figure 3. Adapted from Perry, J (1992) *Gait Analysis: Normal and Pathological Function*. SLACK incorporated

Additionally, gait is described with regards to spatiotemporal parameters (e.g. gait speed, step length, stride length, cadence, ratios expressing gait symmetry), kinematics (joint angles), and kinetics (moments, power and work) where the kinetic parameters explain the kinematic and spatiotemporal parameters.

Gait speed is defined in relation to step length (and stride length) and cadence in healthy subjects (40;41;43), and the preferred gait speed is approximately 1.3 m/s (7;20;44-46). Although, often no gender-specific differences are reported at the preferred gait speed there is a difference in the step length and cadence. Males prefer to walk with a longer step length than females and females walk at a higher cadence than males (20;45-47). This is because of males are taller than females (7;20;47), and when adjusted for height females have a larger stride length than males (20). Although, no gender-specific differences are observed in strategies with regards to step length and cadence, when gait speed is increased (41;45;46), contradicting findings have been observed when healthy subjects reduce their gait speed. Oberg et al. (1993) reported that strategies were equal at the preferred walking speed (45), whereas Waters et al. (1988) observed no differences between genders with regards to step length and cadence (46). Even so, gait cycle periods are related to gait as the stance phase decreases and swing phase increases with increases in gait speed (41). Reference data has been investigated in detail with regards to spatiotemporal and kinematic parameters in healthy subjects and the relationship to gait speed (40;41;44-50). Additionally, age is

negatively correlated to gait speed (19;40;44-50) and in kinematics (19;40;48-50), height is positively correlated to gait speed (40;48), and gender-specific differences are observed when measuring spatiotemporal and kinematic parameters (20;40;44-50)

The numbers of papers, which have investigated the instantaneous power production during gait by muscle groups in the lower extremity, are limited in healthy subjects when compared to spatiotemporal and kinematic parameters. The kinetic peak power produced at hip, knee and ankle in the sagittal (15;18-21) and frontal planes (21) have been investigated in healthy subjects. Peak power generation of the ankle plantarflexors and absorption of knee extensors correlate to walking speed (51). Furthermore, several papers have reported that peak power the hip, knee, and ankle increases with walking speed (7;14;15;18;51), and the time to peak power appears to be consistent when depicted on a mean power curve at different speeds (14;51). It has been suggested that such a sensitive relationship between gait speed and power is the result of a central controlled motor system dependent on afferent input in gait (51). Moreover, the peak concentric power of the plantarflexors (A2-S), hip extensors (H1-S), hip flexors (H3-S), hip abductors (H3-F), and the eccentric work by knee extensors (K3-S) seem to have the largest contribution to the preferred gait speed in healthy subjects (21) (figure 4). In the elderly, the A2-S is reduced (18;19) and is compensated by and increase in H1-S to maintain walking speed (17;18). In addition, gender-specific differences appear in power variables by which K3-S is lower in males than females (20). In addition, Kerrigan et al. (1998) has suggested the same trend in A2-S and H1-S (20). As specified earlier, females walk with a higher cadence compared to males and consequently the angular velocity is larger in females during gait. This may cause the larger peak powers observed (20).

To my knowledge, only one previous study has estimated reference data for power parameters in healthy subjects (16). This interesting work was performed by Lelas et al. (2003) who calculated prediction models for power variables in the sagittal plan (A2-S, H1-S, H3-S and K3-S) adjusting for four walking speeds (16). They observed that the four different walking speeds ranging from approximately 0.5 m/s to 2 m/s explained 85 % of the variation in the model for A2-S, 76 % of the variation in the model for H1-S, 89 % of the variation in the model for H3-S and 92 % of the variation in the model for K3-S. Even so, some limitations are present in this paper. Firstly, the age span from 19 to 40 years makes the prediction models difficult to apply to the elderly, as power is related to age in healthy subjects (17;19). In addition, the majority of gait pathologies are present in the elderly; e.g. initial stroke is typically in patients over 60 years old (4;52). Secondly, no reference

intervals were estimated. A reference interval provides clinicians with an approximated “normal” reference power value which will appear 95 % of the time in a healthy subject.

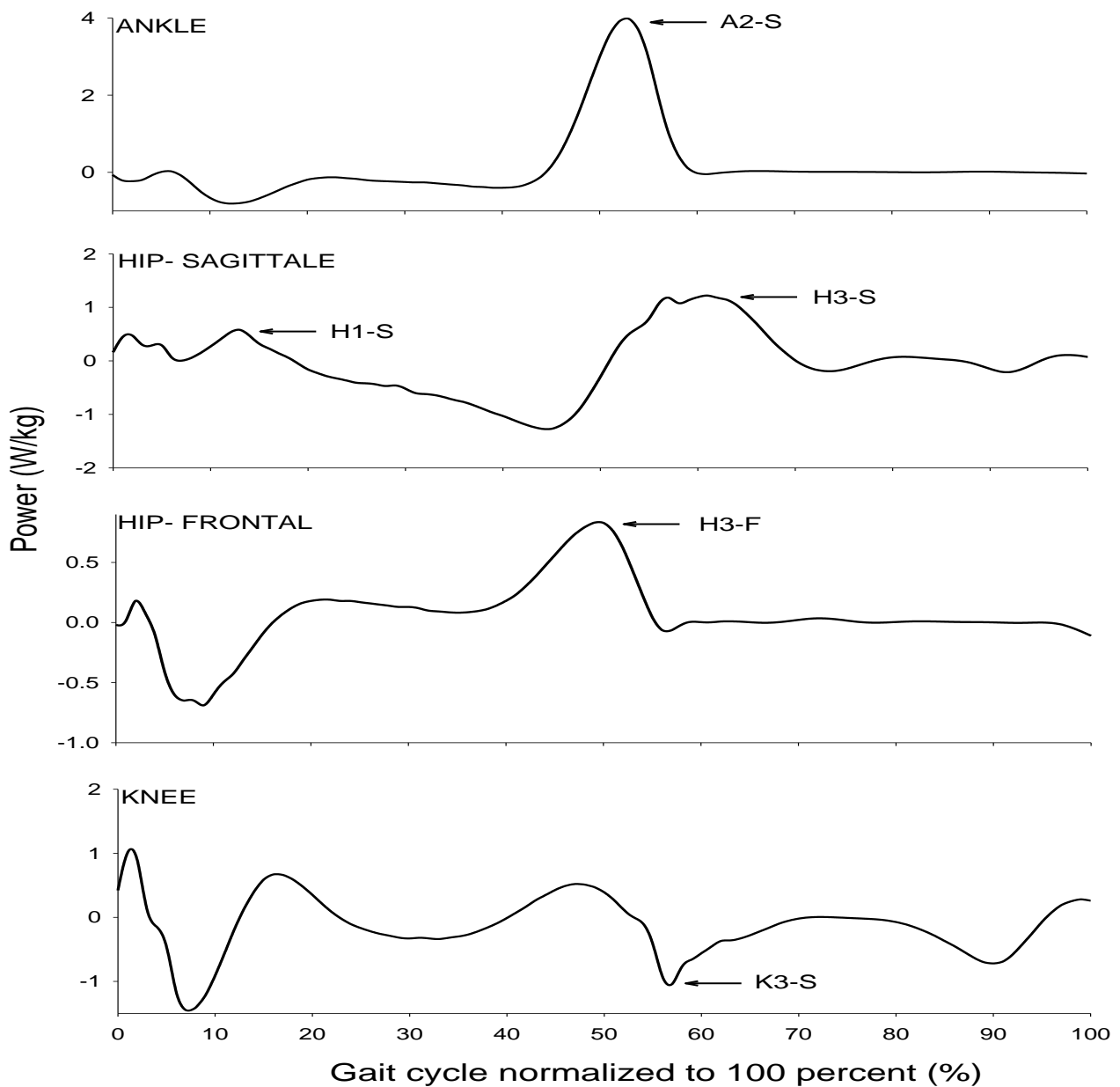


Figure 4. Power peaks in plantarflexion (A2-S), hip extension (H1-S), hip flexion (H3-S), hip abduction (H3-F) and knee extension (K3-S) based on a single power curve in a typical healthy subject (male, 57 years, 173 cm tall) walking at preferred walking speed (1.43 m/s) (figure was adapted from study 3)

In my opinion, reference data is clinical relevant only when an estimated reference interval is present in which the variability of the power of healthy subjects power is represented. This information is excluded if a reference value or a prediction model is only published.

### *2.3. Recovery of function and gait after stroke*

The overall recovery after stroke is dependent on the severity of the neurological deficits and functional dependency (52), and the time course from stroke onset to the final recovery state is related to natural spontaneously recovery and rehabilitation. The degree of neurological and functional recovery, including spontaneously recovery, seems to be largest within the first month of stroke onset, and is suggested to peak approximately three months later (90 days post stroke) (53;54). Variability in the time to optimal stroke recovery is dependent on the initial neurological and functional deficit, in which patients with mild stroke deficits recover earlier than patients with severe stroke deficits (53;54). Additionally, 95 % of stroke survivors do not improve their walking performance after three months (5), and at this point approximately 60 to 65 % of all patients continue to experience walking deficits (5;55).

Hemiparesis is a major neurological deficit in stroke patients, and two thirds of hospitalised stroke patients are reported to suffer from a leg hemiparesis at stroke onset (4;5). This jeopardizes the independent walking performance and lifestyle of stroke patients. Variability in the amount of hemiparesis (none to very severe) are present, and only 55 % of patients with an initially severe leg hemiparesis are reported to reach an independent gait with or without assistance at discharge. In comparison, 89 % of patients with an initially mild leg hemiparesis are reported to reach an independent gait with or without assistance at discharge (5). Consequently, walking performance targets are important in stroke rehabilitation (53), and is also a priority for patients (6).

### *2.4. Gait characteristics in stroke patients*

In general, stroke patients walk with a lower step length, stride length and cadence compared to healthy subjects (8;28;43;56-58), and consequently, they walk more slowly than healthy subjects (7;8;27;28;56-59). The direction of the step length difference between the impaired extremity (IE) and unimpaired extremity (UE) appears to be individually dependent (9;56;60;61), but it seems to be larger in the IE in most patients (61). First (20 %) and second (31 %) double stance are extended and the single limb support phase (SLS) (20 %) and swing period (30 %) are significantly decreased in IE compared to healthy subjects (57). Consequently, total stance (TS) is increased in the IE and UE (62), SLS is reduced in the IE (56), and SLS in the UE tends to be similar or slightly decreased compared to healthy subjects (27;28;56;57). Additionally, stroke patients spend more time in SLS in the UE and less time in the IE compared to speed-matched healthy subjects (60;63). TS and double stance are longer in the UE compared to the IE (56;64), and SLS seems

reduced in the IE compared to the UE (64). Consequently, the period of swing is larger in the IE compared to the UE, and symmetry ratios (SR), based on SLS (IE/UE) and swing phase (UE/IE), differs from healthy subjects ( $SR < 1$ ) (27-29). Similarly, symmetry ratios based on step length (SLR =  $[1 - (\text{step length in the impaired extremity} / \text{step length in the unimpaired extremity})]$ ), with perfect symmetry defined as 0, differ from healthy subjects (61).

This demonstrates an asymmetric gait pattern in stroke patients, and indicates that stroke patients differ from the “normal” division of gait (42) with regards to the degree of time spent in the gait phases. Differences remain even after adjusting for speed in healthy subjects. In addition, the variability in spatiotemporal parameters are larger in stroke patients compared to healthy subjects indicating a larger inter-individual variation in patients (27-29).

Also, large inter-individual differences exist in the gait patterns of hemiparetic patients, and these gait patterns are different from healthy subjects. Consequently, compensatory strategies appear in stroke patients to maintain gait function (9;56;60;63;65-69). This is partly due to motor control deficits (5;53;55) which is negatively correlated to gait speed (27;28;56;65;70-72). Overall gait patterns have been described in stroke patients with different self selected gait speeds, motor control deficits, and stages of stroke (subacute or chronic). The patterns described below emphasize the movement characteristics of the IE.

In chronic stroke patients common gait features are initial contact with increased plantar flexion or drop foot causing a flat foot on ground contact. This causes knee hyperextension and relates to the small degree of range of motion in ankle, knee and hip during stance (9;56;57;60;63;66). Additionally, hip circumduction is present and is associated with increased hip abduction, hip hiking and pelvis retraction (pelvis externally rotated) for toe clearance in swing phase (9;56;57;60;63). Typically, this promotes a gait strategy in which a mass extension motion pattern (ankle, knee and hip extension) during the stance phase and a mass flexion motion pattern (ankle, knee and hip flexion) to ensure the advancement of the leg in the swing phase is used for body progression (56;68). These differences observed in the gait patterns of healthy subjects and patients do not disappear when adjusting for gait speed (60;63). However, the gait patterns of patients are more similar to healthy subjects if gait is initiated with heel strike (initial contact) (9;56).

To my knowledge, only one study described the gait pattern of subacute stroke patients. Three characteristic patterns are dominant in patients with a reduced walking speed (0.16 m/s; n=12) in which the focus is on knee and ankle kinematics during stance. The first pattern features an increased plantarflexion and an almost immediate knee extension toward zero at initial con-

tact and a reduced dorsiflexion in SLS (Extension Thrust Pattern). The second displays an increased stiff-knee flexion and dorsiflexion during total stance (Stiff-Knee Pattern), and the last pattern demonstrates an increased knee flexion and dorsiflexion which varies during initial contact and SLS (Buckling-Knee Pattern). During swing all patients displayed a reduced hip, knee and ankle range of motion and a majority of patients showed a pelvis elevation on the paretic side, and pelvis retraction was identified through the entire gait cycle (hip circumduction). Patients with faster walking speeds (0.55 m/s; n=6) experienced normal ankle, knee and hip angles measured in sagittal plane during stance, and gait was initiated with a normal heel strike (65).

Consequently, the overall gait patterns displays similarities and differences (Buckling-Knee Pattern and Stiff-Knee Pattern) in the gait patterns of subacute and chronic patients. In particular, subacute patients with low walking performance (0.16 m/s) differ from stroke patients with higher walking performance (0.55 m/s).

As an explanation of these gait characteristics (based on kinematic and spatiotemporal parameters) kinetics are a valuable tool. In particular, power produced by the major muscle groups at hip, knee, and ankle in sagittal plane seem to determine walking speed in chronic stroke patient (9;11). In the IE power produced by hip extensors in early stance (H1-S) (9), hip abductors in SLS (H3-F) (9), ankle plantarflexors (A2-S) and hip flexors (H3-S) in the pre-swing phase (9-11), and knee extensors in stance (K1-S) (9) and pre-swing phases (K3-S) (9;11) have shown to be significantly correlated to gait speed. The same trends are observed in the unimpaired extremity (9-11). Differences in peak power between and within legs indicate an inter- and intra-limb compensatory strategy to attain the preferred gait speed, in which the UE compensates for the IE (7;10;12;13). Different strategies can be present within IE (9;10).

Power strategies to increase walking speed among high functioning hemiparetic patients in the chronic stage following stroke (HFH – 0.78 m/s to 1.25 m/s) have show no difference to speed-matched healthy subjects. Power generation (A2-S, H3-S) and absorption (K3-S) increased in both extremities and these muscle groups seem to be important in the increase in gait speed. However, low functioning hemiparetic patients (LFH) were not the same as speed-matched healthy subjects to improve gait speed (0.45 m/s to 0.62 m/s), and only an inter-limb compensation was evident with and increasing hip flexor power at pre swing (H3-S) in UE with increased gait speed (7).

Two studies have investigated the changes in power and work variables in relation to improvements in gait speed in chronic stroke patients. Knowledge of power and work variables



changes in this context and might be important in the targeted gait rehabilitation of stroke patients. Teixeira-Salmela et al. (2001) found no significant changes in kinetics; however, power profiles indicated improvements (amplitude higher) in both the IE and UE. Particularly, there was a trade off between hip and ankle power, in which hip power compensated for ankle power with increases in gait speed. This trend was identified in both extremities. In addition, the UE had larger power values than the IE, and although the IE improved, there was a trend towards the UE compensating for the IE to increase gait speed (13). Parvataneni et al. (2007) investigated work variables based on power profiles during similar interventions (cardiovascular training including muscle strength exercises) (12). Positive hip extension work during weight acceptance (H1-S) in both extremities was responsible for increased gait speed among patients with chronic stroke. Ankle plantarflexor work of the IE in the pre swing phase (A2-S) also contributed to gait speed changes. Surprisingly, small changes were observed in hip flexor work (H3-S) (12).

In summary, these studies indicate that power generation and absorption in major muscle groups in the lower extremity (sagittal plane hip, knee, and ankle) might be important to increase walking speed in chronic stroke patients during gait rehabilitation. In addition, an increased gait speed might influence the pathological gait pattern, by which their gait pattern may be similar to that of healthy subjects.

## *2.5. Gait interventions in stroke rehabilitation*

In general, there is insufficient evidence to conclude that one gait training approach improves gait performance following stroke more than any other approach (73-76). Even so, metaanalysis indicated limited evidence using a mix method approach to improve a patients functional independence (73;74), and further, repetitive task specific gait interventions appear to improve gait speed and endurance (75-77).

Barbeau et al. introduced the concept of a body weight support treadmill training for humans (BWSTT) in 1987 (78) based on an animal study in which a spinalised cat generated a rhythmical walking pattern during BWSTT (79). With this approach the possibility to increase training intensity and duration in a highly repetitive task-orientated fashion was introduced in the gait rehabilitation of neurological patients with stroke and spinal cord injury. This preliminary animal study was followed by a larger study (n=100) in chronic stroke patients in which BWSTT was compared to treadmill training without body weight support. Stroke patients dependent on assistance during walking experienced an improved walking speed, walking endurance and reduced

walking dependency following BWSTT (80). This indicated a positive effect of BWSTT in non-ambulatory stroke patients. Moseley et al. conducted a systematic Cochrane review, in which no evidence was observed to support BWSTT improvements in gait independence, speed or endurance more than any physical therapy approach (73). Similarly to Pollock et al. (2007) (74) a mix of methods were identified in improving walking performance in independent walkers following (treadmill training (with or without body weight support) and a task-orientated physical therapy approach (81)).

Even so, one major limitation of BWSTT is the working load to the physical therapist during gait training. This has been minimized with the introduction of robotics in neurorehabilitation with the preservation of the benefits of BWSTT. In addition, gait qualities such as time spent in the stance and swing phase for both extremities and step length symmetry during walking can be practised in a safe and highly repetitive task-orientated setting. Two gait robotics, the Gait Trainer<sup>®</sup> (82) and the Lokomat<sup>®</sup> (83), have been compared with conventional gait interventions in several trials. Although, these trials displayed mixed results in improvements in walking speed and walking independence in ambulatory and non-ambulatory stroke patients (chronic and subacute) (25;26;84-88), gait training combining robotic training and conventional physical therapy improved gait independence (from non-ambulatory to ambulatory walkers; Functional Ambulatory Scale (FAC) >3) in stroke patients (22-24). This indicates a promising clinical impact of robotics in the recovery of walking independence in stroke patients. In contrast, two studies reported that conventional physical therapy and BWSTT, respectively, improved gait speed more than Lokomat<sup>®</sup> training in ambulatory patients (25;26). Consequently, studies have investigated the effect of Lokomat<sup>®</sup> training on gait qualities secondary to walking speed, because of the symmetrical gait pattern trained in the Lokomat<sup>®</sup>, and the characteristic asymmetrical gait pattern observed in stroke patients (27-29). Time spent in single limb support in the impaired extremity (SLS) has been investigated in two trials. The first study demonstrated a positive effect of Lokomat<sup>®</sup> training compared to physical therapy for subacute non-ambulatory patients (84), and the second study reported a significant increase in SLS with BWSTT compared to Lokomat<sup>®</sup> training for chronic ambulatory patients (26). Similarly, there was no strong evidence in favour of one specific gait intervention for gait symmetry in chronic ambulatory stroke patients when comparing Lokomat<sup>®</sup> and BWSTT (26;85). Furthermore, in clinical practice most gait rehabilitation is provided by physical therapists using conventional therapy, and intensive rehabilitation is usually provided in the subacute phase following stroke, in which the largest benefits of recovery can be observed (54). In summary, it seems reasonable to investigate

the additional effects of the Lokomat<sup>®</sup> in low performing ambulatory subacute stroke patients (walking speed < 0.5 m/s) measuring gait qualities.

### 3. Methods

#### 3.1. 3D gait analysis system

Gait analysis was performed with the Vicon 612 3D motion analysis system (Vicon Motion Systems Limited, Unit 14 Minns Business Park, West Way, Oxford OX2 0JB, UK) including eight infrared strobe light video cameras (VCam / SVCam sampled at 100 Hz) and two steady digital cameras (sampled frequency at 25 Hz). Thirty nine polypropylene retro-reflective markers (14 mm) were attached to the skin to create a full body animation of the subject (figure 5). Sixteen markers were used for analysing the lower extremity kinematic data which were based on the Plug-In-Gait markers model (89-91).

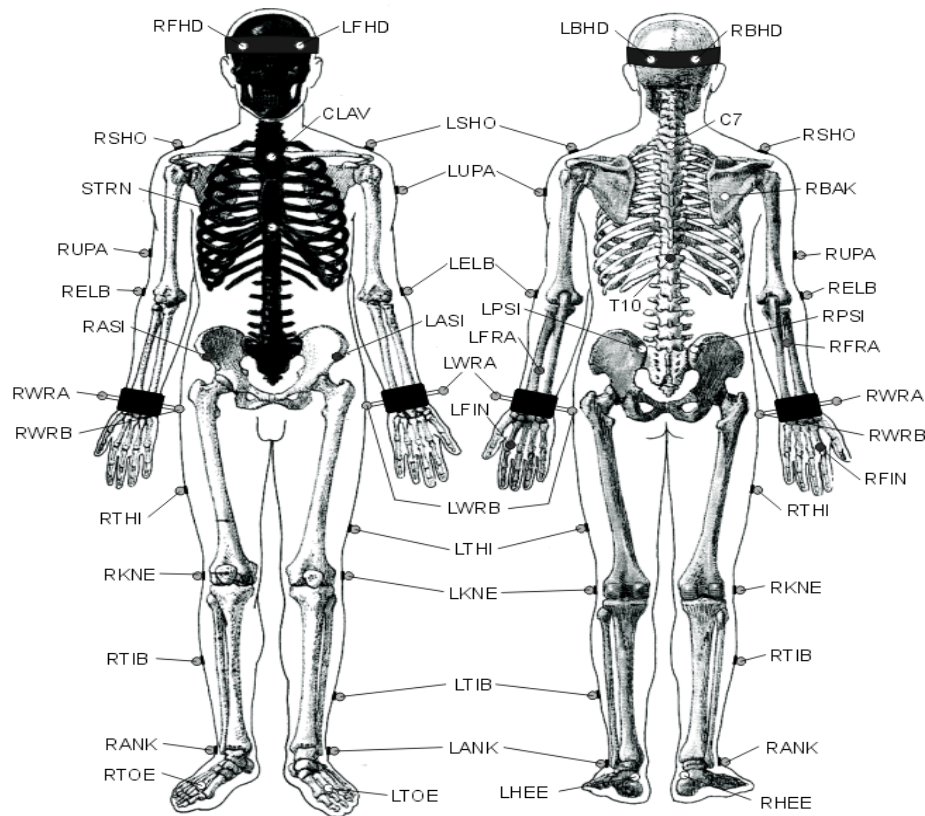


Figure 5. The following describes in detail where the Plug-in-Gait markers should be placed on the subject. Only left sided markers are listed. The positioning was identical for the right side. (Adapted from Vicon®, Polygon manual)

Markers used for data analyses were attached bilaterally on the lateral malleolus of the ankle (R/LANK), over the head of the 2<sup>th</sup> metatarsal (R/LTOE), which is in a horizontal plane with a heel marker (R/LHEE), on the lateral epicondyl of the femur (R/LKNEE), on the anterior iliac spine superior and posterior (R/LASI & R/LPSI), one stick wand marker on the shank in line with the plane of the ankle and knee joint centres (R/LTIB – stick wand not shown) and one stick wand

marker on the thigh, in line with the plane of knee and hip joint centres (R/LTHI – stick wand not shown). Strobe video and force plate (AMTI - model OR6-7 - sampled at 2000 Hz; Advanced Mechanical Technology, INC., 176 Waltham Street, Watertown, MA 02472, USA) data were synchronized and the marker trajectories during data sampling were filtered using a Woltring filter routine for interpolating, smoothing and differentiating the data (92). Static and dynamic camera calibration was performed prior to each new gait analysis. The force threshold was 20 N and was used for detecting gait cycle events on the force plate. Recorded anthropometric data consisted of height, weight, leg length, ankle width, knee width and the distance between left and right anterior and superior iliac spine, and in combination with ground reaction forces in the sagittal and frontal plane and kinematic data, power was calculated using inverse dynamics (1;93).

The subjects walked bare-foot at their preferred walking speed on a 10 meter walkway so at least three full hits were registered on the force plate for each extremity. One length of the walkway equated to one trial, and it was only possible to hit the force plate once in each trial. The subjects were not informed of the embedded force plate to avoid compensatory gait strategies. Gait cycles were normalized to 100 % for each trial which consisted of two consecutive steps initiated and ending with heel strike on same leg. Heel strike on ipsilateral side, toe-off on contralateral side, heel strike on contralateral side, toe-off on ipsilateral side and heel strike on ipsilateral side were recorded for data processing. These events were estimated by software that indicating heel strike and toe-off on the force plate during the trial and were manually determined by the operator for the contralateral foot.

Despite the potential variability of measurements in the gait analysis system (i.e. variability in the participants preferred gait speed, anthropometric measurements, marker placement, calibration of cameras, definition of heel strike and toe-off in the gait cycle for contralateral limb (data processing)) repeatability in healthy subjects is consistent for spatiotemporal parameters, kinematics and kinetics (moment) (90). Kadaba et al. (1989) reported that intra-observer variability ranged from a Coefficient of Multiple Correlation (CMC) of approximately 0.95 to 0.99 (CMC of one equals excellent repeatability) in kinematics and kinetics (sagittal plan: hip, knee and ankle; frontal plan: hip). The coefficient of variance (CV) in gait speed and stride length was 2.9 and 1.7, respectively (90). In addition, McGinley et al. (2008) concluded that hip, knee, and ankle kinematics in the sagittal and frontal plane (hip only) is expected to vary less than 4 degrees in absolute magnitude between measurements. Yavucer et al. (2008) investigated intra-observer variability in 20 chronic stroke patients, and observed that gait speed (ICC) (0.99), step length (0.85), single limb

support (0.84), and kinematics (0.91-0.97; hip, knee, and ankle in sagittal plan) was good (ICC >0.75) (94). In summary, this indicates that gait analysis systems are reliable in measuring relevant spatiotemporal parameters, kinematics, and kinetics included in the thesis. This has been shown in healthy subjects and to some extent stroke patients. No studies were found, in which the reliability of kinetic measures were investigated in stroke patients.

Power was calculated as the product of the joint moment and angular velocity and was normalized to body weight (W/kg) (1). An individual power curve is estimated with an amplitude peak for each trial corresponding to the phase of the gait cycle in which muscle generation or absorption is at its greatest (figure 4). In general, each gait analysis is based on several sufficient trials (3 to 5). Usually, standard settings provided a mean power curve (MPC) based on the individual power curves, and defines the mean peak power (MPP) from MPC. Despite this, MPC will underestimate MPP compared to an MPP based on power peaks from each single power curve (SPC), because differences in the time to peak power (TTP) and in configuration are observed in individual power curves (1;21). This will reduce the peak power identified on the MPC (phase cancellation) (figure 6 and 7). This difference in estimation methods is expected to be particularly evident in stroke patients as the gait cycle events (57;63;65) and power curve configurations (9) can differ considerably from healthy subjects. In addition, TTP may also differ from healthy subjects as peak power is described in relation to gait cycle events. Furthermore, the centrally controlled motor programs are impaired due to stroke, and may increase the variability in TTP (decreased motor control). Consequently, this might disqualify an MPP based on MPC even more. Even so, few studies have been identified in which a description of the estimation method is specified (15;19), whereas other studies vaguely describe the estimation method used (1;9;11;13;21). Studies are required to investigate this methodological issue and the interpretation of MMP.

### 3.3.1. Measurements

A brief description of the outcome measures is presented.

*Spatiotemporal parameters.* Gait speed (m/s) was calculated as the gait cycle distance (m) divided by gait cycle time (s), step length as the distance between left and right heel strike (m), stride length as the distance between two consecutive heel strikes in the ipsilateral foot (m) and cadence as steps per minute. Absolute step length ratio (SLR) has been reported elsewhere (85) and is a modification of the step length ratio (61).  $SLR = [1 - (\text{step length in the IE} / \text{step length in the UE})]$  with perfect symmetry defined as 0. Swing time ratio (STR) was defined as;  $STR = \text{swing time in}$

the impaired extremity/swing time in the unimpaired extremity, with perfect symmetry defined as 1. Single limb support time in the impaired extremity (%) (SLS) was defined as the time from contralateral toe-off to contralateral foot strike.

*Power variables.* Power was assessed as proposed by Eng and Winter (1995) (21) at the relevant phases of the gait cycle, and described below (1;95). A2-S was identified in the sagittal plane at the second double stance phase (pre-swing) where ankle plantarflexor generation ensures gait progression. This occurs at 40 to 60 percent at ipsilateral toe-off in the normal gait cycle (figure 4). H1-S was identified in the sagittal plane at the first double stance phase (occurring at approximately 0-25 % of the gait cycle in healthy subjects immediately prior to and following contralateral toe-off) in which concentric work by the hip extensors was important for acceptance of body weight to maintain posture and progression (figure 4). H3-S was identified in the sagittal plane, at the second double stance phase (pre-swing), and early swing (occurring at approximately 50-75 % of the gait cycle in healthy subjects immediately prior to and following ipsilateral toe-off) in which the active concentric work by the hip flexor assisted progression of the leg in the swing phase (figure 4). H3-F was identified in the frontal plane at terminal stance (occurring at approximately 40-60 % of the gait cycle in healthy subjects immediately prior to and following ipsilateral heel strike) in which concentric work by the hip abductors stabilise to produce a level pelvis position, sufficient step length and toe clearance in swing phase of contralateral leg and assisted to maintain posture in the frontal plane (figure 4). K3-S was identified in the sagittal plane at the second double stance phase (pre-swing) and early swing (occurring at approximately 50-70 % of the gait cycle in healthy subjects immediately prior to and following ipsilateral toe off) in which eccentric work by the knee extensors controlled knee flexion during pre-swing in preparation for limb positioning prior to ankle plantarflexor power generation and toe clearance during the swing phase (figure 4). By convention, A2-S, H1-S, H3-S and H3-F were displayed positively (concentric muscle group power generation) and K3-S was displayed negatively (eccentric muscle group power absorption) (1).

### 3.2. The Lokomat<sup>®</sup> gait orthosis



Picture was located at Hocoma's website 7. April 2010  
(<http://www.hocoma.com/en/>)

The Lokomat<sup>®</sup> (Hocoma, Zürich, Switzerland) has been described in detail previously (83). The Lokomat<sup>®</sup> consists of a treadmill, a body weight support system, motor driven orthosis to guide legs of the subjects at the knee and hip (guidance force (GF)), with firm cushions and straps to attach the orthosis to the participant. This enables gait training of non-ambulatory patients with a reduced working load on the physical therapist. A non-motor driven foot lifter system using elastic straps is attached to the ankle with the ability to increase or decrease the amount of voluntary ankle range of motion.

Consequently, concerns have been raised with regards to the patients active plantarflexion work during gait training (85). Findings by Hidler et al. (2005) indicated a lack of plantarflexion activity during gait in the Lokomat<sup>®</sup> compared to treadmill gait in healthy subjects (96), and later (2008) observed a trend towards lower plantarflexion angle velocity in healthy subjects (97) and a reduced internal plantarflexion moment in chronic stroke patients both during the pre-swing phase when practising in the Lokomat<sup>®</sup> (98). This might cause a restriction in the ability of the patient to practise plantarflexion power generation during the pre-swing phase of gait in the Lokomat<sup>®</sup>.

Leg movements are controlled in the sagittal plane, and restricted from direct movements in the frontal and transverse planes, and consequently, the Lokomat<sup>®</sup> induces no hip abductor torques during gait (97;98). Due to this, Hidler et. al (2008) and Neckel et al. (2008) observed an increased activity in hip flexors and an increased hip angle velocity at approximately 60-80 % of the gait cycle (H3-S) during Lokomat<sup>®</sup> training compared to treadmill training (96;97). This might indicate that Lokomat<sup>®</sup> training could facilitate appropriate hip flexor power generation during gait both to secure progression and toe-clearance and probably, to some extent, can compensate for the lack of muscle activity in plantarflexors during Lokomat<sup>®</sup> training. In addition, an increase in ankle and hip extension during healthy subjects' Lokomat<sup>®</sup>-gait seem to be a logical consequence of the limited degrees of freedom in the lower extremity joints (97).



A pre-programmed physiological ('ideal') gait pattern is administered to the participants during gait training in the Lokomat<sup>®</sup>, but adjustments are possible in certain gait parameters, e.g. treadmill speed, level of body weight support (BWS), step length, knee and hip angles and the amount of guiding force. A biofeedback system provides information to the patients regarding their gait performance during training. The biofeedback monitors the performance of the subjects at the knee and ankle joints and compares the subjects expected performance with their actual performance. Thus, the operators may guide subjects to modify the timing of gait cycle parameters and encourage subjects to work actively while training in the Lokomat<sup>®</sup>.

No doubt, the gait pattern practised in the Lokomat<sup>®</sup> does not resemble an overground gait pattern produced by healthy subjects due to the restricted nature of the Lokomat<sup>®</sup> (leg movements in sagittal plane only) (96;97), but symmetrical bilateral hip range of motion (kinematics) can be controlled and are observed in chronic stroke patients during Lokomat<sup>®</sup> training (98). Similarly, chronic stroke patients increased gait pattern symmetry by gait training on a treadmill compared to overground training (99).

In the present thesis (study 4), three parameters were adjusted during gait training in the Lokomat<sup>®</sup>; gait speed, BWS and GF. Our training protocol reduced BWS in week 1, increased gait speed in week 2 and reduced GF in week 3. In every training session patients were encouraged to increase the degree of active participation (increase gait speed or decrease BWS/GF) and continue to maintain safe walking without the risk of stumbling. Similarly the gait training strategy was used by Pohl et al. (2002), in which they observed that enhanced speed treadmill training without BWS in every training session increased gait speed. This was compared to conventional gait training and treadmill training without enhanced speed training in subacute stroke patients (100). Previous studies have used a variety of approaches to conduct Lokomat<sup>®</sup> training (24-26;84-86), which indicates that, studies are required to observe the effect of the Lokomat<sup>®</sup> training parameters (treadmill speed, BWS, and GF) in stroke patients.

In the current study, gait patterns similar to healthy subjects were obtained during training, e.g. sufficient heel strike, knee and hip angle patterns during stance and toe clearance in late pre-swing. Guidance forces were applied to both sides and changes in guidance force were adjusted equally on both sides. Verbal encouragement and temporary physical assistance was applied so that subjects could follow the induced gait pattern. Although it might seem unnatural for some patients, it is assumed that it is safe for gait training in stroke patients (22)

### 3.3. Physical therapy

Based on the recommendations from a systematic Cochrane review by Pollock et al, physical therapy gait training contained a mix of components from different physical therapy approaches, and the intervention was clearly described (74). Gait training was inspired by the neurophysiological approach (101;102) and the motor re-learning approach (81;103). The principle of the gait training was to mimic the three dimensional symmetrical gait patterns of healthy subjects (joint movements in sagittal, frontal, and transverse plane) (42) and to re-learn everyday gait activities using a “hands-on” (101;102) and “hands-off” approach (81;103). The active task-orientated and repetitive nature of the motor-relearning approach was emphasized during gait training. This has been proposed to improve uni-dimensional measures (activity/impairment level) such as gait speed and gait endurance in stroke patients (76;77). In addition, the physical therapist explains and demonstrates the exercises practised by the patients and is followed by self-initiated practise in which the therapist can support the patient. This increases the level of task demands and can be a cognitive challenging in stroke patients. This must be taken into consideration in the rehabilitation of patients (81;102;104).

Gait training was classified into four categories. *Category 1:* Specific components in the gait cycle were practiced; i.e. in standing, transferring from two feet to one foot practising weight acceptance, single limb support, limb advancement (including push and pull off subtasks at pre-swing phase). *Category 2:* Gait training with support, i.e. walking supported by a wall, bench, parallel bars with the opportunity of physical assistance. *Category 3:* Patients walking independently with no physical assistance, with the focus on practising dual tasks, i.e. patients carried a glass of water and prepared the dinner table. *Category 4:* Independent walking in different environments, i.e. shopping and outdoor walking in challenging environments. Focus was on dual tasks as well as distance and endurance practise. In general, practising stair climbing and gait with walking aids were presented in category 2 - 4. The patients gait performance increased with a higher category number and the total amount of assistance and the physical therapists verbal or “hands-on” facilitation reduced with a higher category number. A gait training session could contain one or a mix of more categories depending on the patients’ impairment.

A major limitation in physical therapy gait training is the physical load required by the therapist during gait training in stroke patients with a low walking performance. This may reduce training duration, intensity, and amount of repetitions practised. These limitations were offset by the

high level of task specific gait training in different relevant contexts (i.e. shopping and outdoor walking in challenging environments) (75;76).

## 4. Results

This section gives an overview of the main findings of the four papers included in the thesis. For a detailed description of each study please see appendix.

### 4.1. Gender, age and height are of minor importance in determining power based on gait analysis in healthy subjects (study 1)

This cross-sectional study estimated reference data with intervals for mean peak power of ankle plantarflexors (A2-S), hip extensors and flexors (H1-S, H3-S), hip abductors (H3-F) and knee extensors (K3-S) related to the demographic variables gender, age, and height. Using 3D gait analysis 158 healthy subjects were tested at their preferred walking speed. Predictive models were presented with the standard deviation of the residuals (SD of the predictive muscle power), standard deviation of the measured muscle power (SD) and coefficient of determination ( $R^2$ ) in each model. No significant differences were observed in preferred gait speed and age between genders, but as expected, height, weight, step length and stride length were significantly larger in males than in females, and cadence was higher in females.

Predictive models were estimated for A2-S, H1-S, H3-S, H3-F and K3-S and were related to gender, age, and height (table 1). Only the model for H1-S was able to explain more than 10 % of the measured variation in power (27 %), which was due to a significant positive relation between H1-S, gender (male) and height. Consequently, the variation of the predictive power was not significantly different from the variation of the measured power after adjusting for gender, age, and height in the predictive models ( $RSD \approx SD$ ). Females produced a larger H3-S and K3-S than males ( $p < .05$ ), and the H1-S was higher in males ( $p < .05$ ) when adjusted for age and height (table 1).

Table 1. Predictive models for power variables in the lower extremities in relation to gender, age and height

Power variables (W/kg)	Inter-cept	Regression coefficient (CI)			RSD	SD	$R^2$	*Walking speed (m/s)	$R^2$
		Gender (male=1; female=0)	Age (years)	Height (cm)					
A2-S	2.30	-0.5 (-0.92;0.08)	-0.01 (-0.018;-0.000)	0.015 (-0.01;0.04)	0.84	0.88	0.10	3.5 (2.9;4.2)	0.51
H1-S	-1.44	0.19 (0.075;0.038)	0.002 (0.001;-0.001)	0.01 (0.003;0.020)	0.30	0.35	0.27	0.9 (0.7;1.2)	0.45
H3-S	0.74	-0.21 (-0.370;-0.056)	0.002 (-0.001;0.006)	0.003 (-0.006;0.012)	0.31	0.32	0.08	1.4 (1.1;1.6)	0.53
H3-F	0.91	-0.014 (-0.095;0.067)	-0.002 (-0.004;-0.000)	-0.002 (-0.007;0.003)	0.16	0.16	0.05	0.2 (0.07;0.4)	0.10
K3-S	-0.57	0.17 (0.008;0.326)	-0.002 (-0.005;0.002)	-0.003 (-0.012;0.007)	0.32	0.32	0.05	-1.3 (-1.6;-1.1)	0.49

Abbreviation:  $R^2$  reflects the variation in muscle power that the predictive models explain; RSD= standard deviation of the residuals (SD of the predicted muscle power); SD= standard deviation of the measured muscle power in the subjects (n=158). \* Walking speed was not included in the predictive models.

#### *4.2. Mean power curves underestimate the true muscle power: Studies of subacute stroke patients and healthy subjects (study 2)*

Data from this study is partly described in study 3 and 4. This explorative pilot study investigated estimation methods of the mean peak power of ankle plantarflexors (A2-S), hip extensors and flexors (H1-S, H3-S), hip abductors (H3-F) and knee extensors (K3-S) in subacute stroke patients and healthy subjects. Using 3D gait analysis two subacute stroke patients, a slow walker (SW, 0.33 m/s) and a fast walker (FW, 0.52 m/s), were tested twice during a six week rehabilitation period; at the beginning (test 1) and end (test 2) of rehabilitation. Similarly, 13 healthy subjects were tested at slow and preferred walking speeds, and 12 subacute stroke patients were tested once. Variables were MPP based on MPC and SPC and TTP in A2-S, H1-S, H3-S, H3-F and K3-S.

*Differences between MPC and SPC.* SW and FW experienced a larger estimated MPP based on SPC compared to MPC for each power variable in test 1 and 2. In test 1 the percentage differences were smallest for H1-S, whereas large differences were observed for H3-S in both extremities, and H3-F and K3-S for the UE in both subjects (SW and FW). These differences decreased during rehabilitation for all power variables. Even so, SW experienced a larger percentage difference estimating H3-F (115.4 %) in test 2 (figure 6). A larger difference in TTP was observed in test 1 compared to test 2 in SW and FW, and it was observed in both extremities.

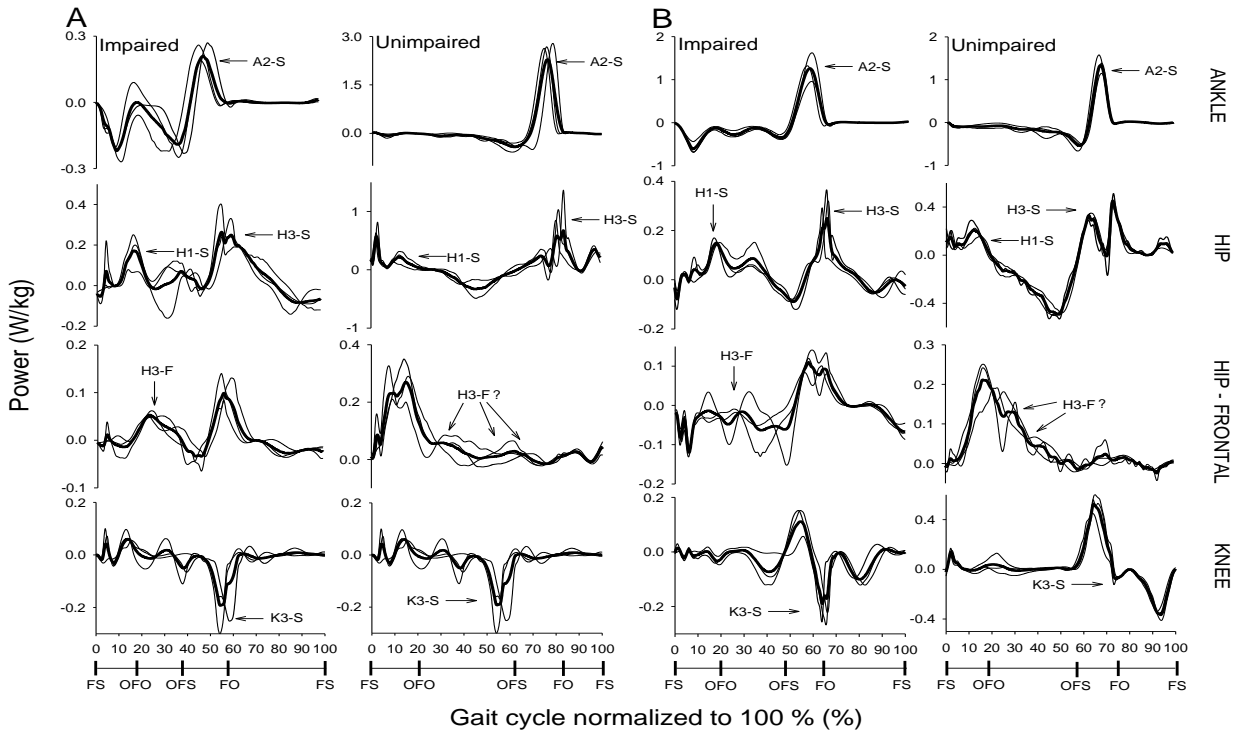


Figure 6. Single power curves (thin lines) and mean power curves (thick line) at the ankle, hip and knee in the impaired and unimpaired extremity for a slow walking stroke patient (63 years old, 0.36 m/s, FIM=62, 39 days post stroke) at test 1 (A) and test 2 (0.46 m/s) (B). Gait cycles are normalised to 100% and displayed with the following gait cycle events: FS, foot strike; OFO, opposite foot off; OFS, opposite foot strike; FO, Foot off.

The same patterns were observed in healthy subjects, by which significant differences were observed between MPC and SPC at slow and preferred walking speeds. Percentage differences between MPC and SPC reduced at the preferred walking speed, and were significantly reduced for the A2-S, H3-S and K3-S (figure 7). Likewise, differences in TTP reduced at the preferred walking speed, and differences in TTP for A2-S, H3-S, H3-F and K3-S were significantly reduced compared to the slow walking speed. TTP was significantly different from zero in both the slow and preferred walking speeds.

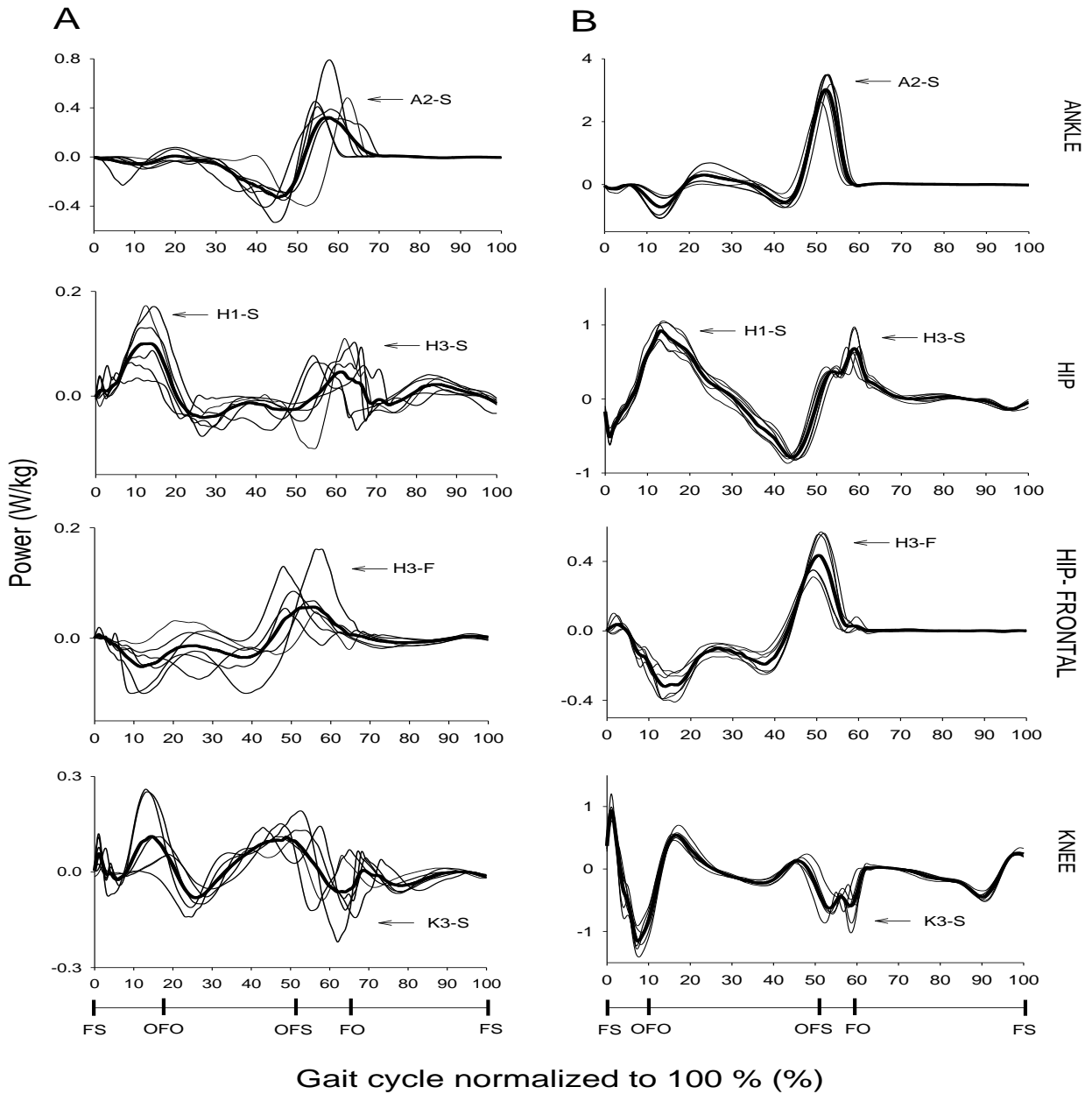


Figure 7. Single power curves (thin lines) and mean power curves (thick line) based on both extremities in the ankle, hip and knee in a typical healthy subject at a slow walking speed (0.36 m/s) (A) and at a preferred walking speed (1.28 m/s) (B). Gait cycles are normalised to 100 % and pictured with the following gait cycle events: FS, foot strike; OFO, opposite foot off; OFS, opposite foot strike; FO, Foot off.

*Correlations between differences in TTP and walking speed.* Differences in TTP for H1-S and H3-F in IE were significantly correlated to walking speed. No other significant correlations were observed (table 3).

Table 2. Correlations between differences in TTP for power variables and walking speed (median: 0.47 m/s (range: 0.27-1.26)) in subacute stroke patients

Power variables	Differences in TTP median (range)	Spearman-Rank correlation (r)	<i>p</i>
Impaired extremity			
A2-S	2 (0-7)	-0.55	.06
H1-S	2.5 (1-14)	-0.58	.047*
H3-S	2.5 (0-6)	0.17	.59
H3-F	3 (0-16)	-0.69	.01*
K3-S	2 (1-7)	-0.32	.31
Unimpaired extremity			
A2-S	1 (0-5)	-0.16	.62
H1-S	4 (0-18)	-0.48	.11
H3-S	1.5 (0-4)	0.15	.65
H3-F	2 (0-13)	-0.26	.42
K3-S	2 (0-4)	-0.07	.82

\* Statistic significant at 5 %;  $p < .05$ ;  $n=12$

### 4.3. Increased power generation in impaired lower extremities strongly correlates to changes in walking speed in subacute stroke patients (study 3)

Power data was extracted and pooled from all participants in study four independent of intervention groups (LGO-PT or PT-LGO), and used in present study. Thirteen subacute stroke patients (time from stroke onset < 3 months, walking speed < 0.5 m/s with or without a walking stick at baseline, and walking speed improved during rehabilitation) and thirteen healthy subjects matched to patients paired for gender, age ( $\pm 10$  years), height ( $\pm 6$  cm) and baseline walking speed ( $\pm 0.1$  m/s) were tested using 3D gait analysis. Patients were tested at baseline and after six weeks of gait rehabilitation and healthy subjects were tested at slow matched walking speed and at preferred walking speed. The following gait parameters were estimated; (1) walking speed (m/s) and (2) A2-S, H1-S, H3-S, H3-F and K3-S (W/kg).

No differences were observed between stroke patients and healthy subjects at baseline regarding anthropometric and demographic data however, five patients used a walking stick and one patient was assisted by a physical therapist to minimize the risk of falling during the tests. A significant improvement was observed in mean walking speed from baseline (0.29 m/s (SD:0.14)) to post-training (0.57 m/s (SD:0.3)) in patients ( $p < .001$ ). The healthy subjects self selected slow walking speed was 0.39 m/s (SD:0.12) and attained, on average, a preferred comfortable walking speed at 1.45 m/s (SD:0.19), ( $p < .001$ ). Patients improved A2-S and K3-S in IE and H1-S in UE with no other improvements attaining significance (table 3).



Table 3. Power variables in patients impaired and unimpaired lower extremity at baseline and after six weeks of gait rehabilitation

Power (W/kg)	Impaired extremity				Unimpaired extremity			
	Baseline Mean (SD)	Six weeks Mean (SD)	Difference Mean (SD)	<i>p</i>	Baseline Mean (SD)	Six weeks Mean (SD)	Difference Mean (SD)	<i>p</i>
A2-S	0.43 (0.5)	1.04 (1.04)	0.61 (0.62)	.004*	1.28 (0.64)	2.05 (0.8)	0.77 (1.06)	.02
H1-S	0.13 (0.13)	0.20 (0.24)	0.07 (0.23)	.30	0.23 (0.15)	0.44 (0.26)	0.21 (0.2)	.002*
H3-S	0.21 (0.01)	0.37 (0.2)	0.15 (0.23)	.03	0.45 (0.22)	0.63 (0.26)	0.17 (0.31)	.06
H3-F	0.08 (0.07)	0.11 (0.1)	0.03 (0.08)	.21	0.12 (0.06)	0.18 (0.08)	0.07 (0.08)	.01
K3-S	-0.16 (0.1)	-0.31 (0.17)	0.15 (0.14)	.002*	-0.29 (0.17)	-0.45 (0.26)	0.16 (0.27)	.05

\* Statistic significant difference at 5 % level after Bonferroni adjustment;  $p < .005$ ;  $n=13$

All changes in power variables were highly significant for healthy subjects when increasing walking speed from a self selected slow speed to a comfortable preferred speed ( $p < .001$ ). A significantly strong correlation between the change in walking speed and improvements in muscle power generation were observed in the impaired A2-S, H1-S, H3-S and H3-F, and the unimpaired A2-S and H3-F (table 4). The change in the walking speed of healthy subjects was not correlated to H1-S or H3-F, but was correlated to the change in A2-S, H3-S and K3-S (table 4).

Table 4. Linear dependence between changes in walking speed and changes in power variables in healthy subjects and the patients impaired and unimpaired lower extremity

Power variables	Pearson product-moment correlation (r)	<i>p</i> - value
Impaired extremity		
A2-S	0.85	<.001*
H1-S	0.81	<.001*
H3-S	0.89	<.001*
H3-F	0.88	<.001*
K3-S	-0.53	.06
Unimpaired extremity		
A2-S	0.86	<.001*
H1-S	0.41	.16
H3-S	0.53	.06
H3-F	0.67	.01*
K3-S	-0.33	.26
Healthy subjects		
A2-S	0.77	.002*
H1-S	0.35	.24
H3-S	0.7	.007*
H3-F	-0.1	.77
K3-S	-0.64	.02*

\* Statistic significant at 5 %;  $p < .05$ ;  $n=13$

#### 4.4. The order of gait training, including Lokomat<sup>®</sup> and physical therapy, do not influence gait quality in subacute ambulatory stroke patients – A pilot study (study 4)

This cross-over designed pilot study compared the effect of the Lokomat<sup>®</sup> gait orthosis training (LGO) to a task-specific gait training conducted by a physical therapist (PT) in ambulatory subacute stroke patients. Thirteen patients (time from stroke onset < three months and walking speed < 0.5 m/s) were randomised by sealed envelopes into the two gait intervention groups; Lokomat<sup>®</sup> (LGO) - Physical therapy (PT) and Physical therapy (PT) - Lokomat<sup>®</sup> (LGO). The interventions consisted of three weeks of LGO and three weeks of PT, with a total of 30 gait training sessions (2 × 15 sessions) of 30 minutes each were scheduled. Patients participated in no other specific gait training during the six weeks of gait intervention. Gait parameters were collected in a 3D gait analysis system, and consisted of the primary outcome measures; gait symmetry expressed as (1) absolute step length ratio (SLR), (2) swing time ratio (STR), and (3) single limb support time (SLS) in the IE. The secondary outcome was walking speed.

Patient characteristics are presented at baseline in table 5. Seven patients completed the full gait training protocol, four completed 29 training sessions, and two completed 28 sessions. Patients with protocol differences were equally distributed between groups.

Table 5. Subject characteristics at baseline

	Lokomat - Physiotherapy (n=7) (median (range))	Physiotherapy - Lokomat (n=6) (median (range))	<i>p</i>
Gender (M/F)	5/2	4/2	1.00
Age (years)	61 (38-71)	59 (38-64)	.52
Days post stroke	56 (20-79)	21 (9-39)	.05
Hemiparetic lower extremity (L/R)	2/5	5/1	.10
FIM (min.18 - max.126)	88 (59-109)	96 (59-113)	.57

Abbreviations: M, Male; F, Female; L, Left; R, Right; FIM, Functional Independence measure.

No significant changes existed between the outcome measures of the intervention groups (table 5). Additionally, no significant differences were observed between groups measuring primary and secondary outcomes after adjusting for days post stroke with an ANCOVA analysis.

*Absolute step length ratio (SLR), swing time ratio (STR), and single limb support time (SLS).* Regarding primary outcomes no differences were observed (table 6). A near significant decrease in STR was observed in patients practising PT following three weeks of training (median: -0.56 (range: 0.07;0.99)). SLS was improved in both intervention groups during the first three weeks but no significant difference was observed, however PT-LGO had a trend towards improvement when compared to LGO-PT after three weeks (median: 9.6% (PT) vs. 4.1% (LGO)) (table 6).

*Self-selected walking speed (SWS).* Within both intervention groups, a significantly improved walking speed was observed following three weeks (PT-LGO, median: 0.33 m/s to 0.54 m/s; LGO-PT, median: 0.24 m/s to 0.29 m/s) and after six weeks (PT-LGO, median: 0.54 m/s to 0.64 m/s; LGO-PT, median: 0.29 m/s to 0.36 m/s). A trend towards a larger improvement in PT practise following three weeks intervention was observed (median: 0.26 m/s (PT) vs. 0.08 m/s (LGO);  $p=0.06$ ) (table 6).

Table 6. Step length ratio, swing time ratio, single support stance time and walking speed determined at baseline and changes in outcome determined after three and six weeks of intervention

	Lokomat - Physiotherapy (n=7) (median (range))	<i>p</i> (within group)	Physiotherapy - Lokomat (n=6) (median (range))	<i>p</i> (within group)	<i>p</i> (between group)
SLR					
Baseline	0.19 (0.04;1.40)		0.31 (0.03;2.25)		.89
Δ Three weeks	0.04 (-0.73;0.18)	.61	-0.05 (-2.07;0.11)	.35	.57
Δ Six weeks	-0.10 (-0.41;0.11)	.13	-0.04 (-0.22;0.2)	.46	.25
STR					
Baseline	1.99 (1.20;6.60)		1.65 (1.20;2.44)		.67
Δ Three weeks	-0.05 (-3.17;0.23)	.18	-0.56 (-0.99;-0.07)	.028	.39
Δ Six weeks	-0.01 (-1.56;0.21)	.31	-0.03 (-0.12;0.34)	.75	.39
SLS (%)					
Baseline	20.6 (5.2;28.3)		23.2 (10.6;25.9)		.25
Δ Three weeks	4.1 (-0.2;11.5)	.05	9.6 (5.1;13.0)	.028	.028
Δ Six weeks	1.3 (-0.7;6.4)	.13	0.85 (-6.3;2.5)	.46	.77
SWS (m/s)					
Baseline	0.24 (0.06;0.47)		0.33 (0.15;0.53)		.22
Δ Three weeks	0.08 (0.02;0.22)	.018*	0.26 (0.07;0.53)	.028*	.06
Δ Six weeks	0.10 (-0.01;0.31)	.028*	0.12 (0.03;0.22)	.028*	.39

Abbreviations: SLR, absolute step length ratio; STR, swing time ratio; SLS, single support stance in impaired extremity; SWS, self-selected walking speed; Δ Indicates change in outcome measure from baseline to three weeks (Three weeks-Baseline) and from three weeks to six weeks (Six weeks-Three weeks). \* Significant changes within and between intervention groups were set at  $p<.017$  following a Bonferroni adjustment for primary outcomes (0.05/3) and at  $p<.05$  for walking speed.

## 5. Discussion

3D gait analysis systems are used in clinical practise and research. Gait analysis is based on biomechanical models, in which a detailed description is given regarding a patients movements (kinematics) and the forces applied to produce these movements (kinetics). Kinetic variables, such as power and work, explain kinematics such as gait speed and consequently pathological movement patterns may be identified (1). In chronic stroke patients, the muscle group peak power of ankle plantarflexors (A2-S), hip extensors (H1-S), hip flexors (H3-S), hip abductors (H3-F), and knee extensors (K3-S) have been suggested to be strongly correlated to walking speed (7-13). This is also well established in healthy subjects (14;15). Suitable reference data of these power variables may be a valuable tool to compare impaired values to healthy values during gait rehabilitation. In addition, it might assist therapists in choosing appropriate gait training strategies when treating pathological gait patterns; e.g. training plantarflexor muscles to increase the power of ankle plantarflexors and gait speed (if this is the problem).

Reference data was established in muscle group power of the ankle plantarflexors (A2-S), hip extensors (H1-S), hip flexors (H3-S), hip abductors (H3-F) and knee extensors (K3-S) in a comprehensive study population (n=158; study 1). The predictive models were explained by gender, age, and height in which the standard deviation of the residuals (RSD) was presented. Calculating the RSD enabled a calculation of a 95 % reference interval. Below is a clinically relevant example to illustrate the intended use of predictive models. For example, if the intention was to predict a reference value and an interval for the plantarflexion power (A2-S) during gait for a 178 cm tall, 65 year old stroke patient (male). The reference value (predicted power) for A2-S was:  $2.30 - 0.5 \times 1 - 0.01 \times 65 + 0.015 \times 178 = 3.82 \text{ W/kg}$ . The 95 % reference interval (predictive interval) for A2-S with a RSD of 0.84 was: predicted power  $\pm 1.96 \times \text{RSD} \Rightarrow 3.82 \text{ W/kg} \pm 1.96 \times 0.84 = \text{range: } 2.17 \text{ to } 5.47 \text{ W/kg}$  (table 1).

This indicates that even though the reference value is 3.82 W/kg, the “normal” power value will be at an interval from 2.17 W/kg to 5.47 W/kg in 95 % of the time for a healthy male with the above characteristics. The range of reference intervals represents the size of the variation in the predicted power (RSD), which is primarily based on the variation in the measured power (SD) and explainable variables. If the A2-S is outside (below the lower limit) the reference interval, it might indicate that gait training should involve strengthening of the ankle plantarflexors. In comparison, the average A2-S produced by the subacute stroke patients in the thesis was in the range of 0.43 W/kg to 1.04 W/kg in hemiparetic extremity and from 1.28 W/kg to 2.05 W/kg in unimpaired

extremity during rehabilitation (table 3). These examples were outside the reference interval for A2-S, and indicate that the interval may be useful in early stroke rehabilitation. Furthermore, if we incorporate findings from study two regarding an underestimated mean peak power based on a mean power curve an approximately 20 % decrease of the MPP of A2-S was observed in one stroke patient. Therefore, in theory this would incorrectly reduce the above A2-S from 0.43 W/kg to approximately 0.35 W/kg. In this case, there is no consequence on the interpretation of A2-S in relation to the reference interval, but it might have consequences in other estimations near the limits of the intervals.

As emphasised in the introduction A2-S, H1-S, H3-S, H3-F and K3-S are related to walking speed in stroke patients, and by consequence, specific training of these are important during gait rehabilitation. Additionally, in stroke patients and healthy subjects a trade off mechanism is present during the stance phase between the hip and knee joint kinetics in which knee and hip muscles compensate to preserve an upright body position (9;95). Consequently, clinicians are recommended to evaluate current power variables together to reach a completed overview of the power generation of muscle groups in the lower extremity. Hopefully, unrealistic rehabilitation goals might be prevented by comparing the power of the patient to these reference intervals.

In the calculated models, gender determined H3-S, K3-S (women>men), and H1-S (men>women), age determined A2-S and H3-F, and height determined H1-S. In addition, independent variables explained less than or equal to ten percent of the variation in the predictive models for A2-S, H3-S, H3-F and K3-S, and consequently, small differences were observed between RSD and the SD (table 1). By contrast, gender, age, and height explained 27 % of the variation in the model for H1-S. No explanations were established for this difference in  $R^2$  magnitude between the model of H1-S and the other models. Obviously, the easily accessible explainable variables used in the present study cannot account for the variation in power alone, but if preferred walking speed is included into the model,  $R^2$  is expected to increase. In the current study,  $R^2$  ranged from 0.45 to 0.53 in A2-S, H1-S, H3-S, and K3-S and the model for H3-F attained an  $R^2$  of 0.1 after adjusting for gender, age, height, and walking speed (table 1). This is in accordance with the findings of Lelas et al. (2003) in which power (A2-S, H1-S, H3-S, and K3-S) was explained by walking speed (76 %-92 %) (16). In the current study, walking speed was not included into the predictive models because subjects with gait pathologies are not speed-matched with healthy subjects when walking at their preferred speed e.g. stroke patients (27). Consequently, to include the patients walking speed into a

predictive model, which is based on preferred walking speed of healthy subjects, would not predict the correct power reference data.

Gender-specific differences were observed in H1-S, H3-S and K3-S (table 1). Even so, the preferred walking speed did not differ between genders. The larger power observed in H3-S in women may be explained by an increased cadence to reach the same preferred walking speed as men. Consequently, the angular velocity will increase along with the increase in cadence, as power increases proportionally with angle velocity (1). These findings are partly in agreement with Kerrigan et al. (1998). Kerrigan et al. (1998) reported a trend towards larger hip extensor power (H1-S) in female compared to males (20). By contrast, in the present study, males had higher H1-S after adjusting for gender, age, and height, and are probably explained by gender differences in height. In addition, males attained a longer step and stride length compared to females indicating that males utilized this strategy to reach a preferred walking speed similarly to females, as walking speed is dependent on stride length and cadence (41;105). This concurs with the laws of biomechanics as the ground reaction force will be at a larger distance from the joint centre (longer lever arm => increased hip moment) (1). Furthermore, age was significantly and negatively related to H3-F, which indicates that hip abductor power decreases with age. This was also observed in A2-S, whereas H1-S and H3-S were positively related to age. These findings might indicate that a redistribution of power variables is present and related to age in order to attain the same walking speed. De Vita et al. (2000) reported a similar trend in power between A2-S and H1-S (17). Also, intra-limb “trade off’s” have been identified previously and is used to secure lower limb support during preferred gait in healthy subjects and patients (95;106). In addition, no gender difference was found in the model for A2-S after adjusting for gender, age, and height. This may be explained by the negative relationship between A2-S and age, and concurs with previous work performed by Kerrigan et al. (1998) (20).

A limitation of the findings was the method used for subject recruitment. This might introduce bias to the study by the inclusion of a larger number of highly motivated healthy subjects when compared to the general population, and findings are only representative for subjects aged between 20 and 79. Although the explainable variables were chosen in respect to gait impairments in stroke patients, it is believed, that the presented reference data are useful in gait rehabilitation when indicated; e.g. in movement or orthopaedic disorders.

### *5.1. Calculation and interpretation of power in major muscle groups in healthy subjects and subacute stroke patients during gait*

Experiences from the first study indicate that appropriate estimation methods are needed in gait analysis, when power variables are calculated. Usually, standard settings in gait analysis systems provide a mean power curve (MPC) based on the individual power curves, and defines the mean peak power (MPP) from MPC. Even so, clinicians and researchers involved in gait analysis must be aware of estimation methods for a mean power peak (MPP) (mean power curve, MPC, and single power curve, SPC) and the potential differences in MPP derived from these estimations.

In the current study (study 2), an underestimation of MPP was observed in 36 out of 40 analyses for a slow walking stroke patient (SW) and a fast walking stroke patient (FW), when based on MPC compared to SPC. This strongly indicates a difference between the estimation methods for MPP in impaired gait patterns. These underestimations were caused by variability in TTP and in curve configuration (figure 6 and 7), and consequently, a phase cancellation of the MPP was observed in MPC in which the sum of single power curve peaks were reduced. Underestimations were most prominent for SW (figure 6) and FW at test one (approx. walking speed of 0.3 m/s and 0.5 m/s) in both extremities. This indicated a large variability in the TTP in early subacute stroke (test 1) and is probably related to a reduced motor control in the IE (27;28;70). The degree of underestimation of power peaks reduced for SW and FW during rehabilitation (test 2), and were most prominent for FW as the patient attained a walking speed similar to the preferred speed of healthy subjects (approx. 1.3 m/s). A consistent TTP in powers were observed in healthy subjects at the preferred walking speed, and similar findings were reported in previous work by Winter (1983). It was suggested that findings were the result of a central controlled motor system dependent on afferent input rather than just the spinal central pattern generator (CPG) (51), which is in agreement with current understanding of neural control in the locomotion of healthy subjects (31;33). In SW and FW, TTP appeared more consistent later in the subacute rehabilitation (test 2), and might indicate that the timing of the work of specific muscle groups became integrated in a more consistent motor pattern. This concurs with the gait pattern of subacute and chronic stroke patients' when the gait speed paralleled that of healthy subjects (9;56;65).

In healthy subjects, differences in the TTP and percentage differences in MPP (MPC vs. SPC) decreased as subjects achieved their preferred walking speed (figure 7). Despite this, the TTP of the healthy subject varied, indicating that variability in the TTP is a normal feature in

healthy subjects. Therefore, defining power variables related to a specified interval in a normalised gait cycle seems reasonable as proposed in previous studies (1;21). Additionally, an MPP based on MPC does underestimate the true MPP in the healthy subjects walking at their preferred speed (figure 7). In the current study, data indicates that walking at a very slow speed (25 % of the preferred speed) was difficult and resulted in a large variability in TTP in combination with variability in curve configuration (figure 7). Variability was comparable to patients (SW and FW), indicating that it was not entirely a pathological feature. Consequently, speed-matched healthy subjects compared to patients seemed relevant if studies want to distinguish pathological gait characteristics from healthy gait characteristics, and such study designs have been reported (7;8;60). By contrast, testing healthy subjects at their preferred walking speed would be inappropriate. Secondly, the results indicated that a very slow walking speed is not normal walking in healthy subjects, and thus induces walking variability in healthy subjects. This variability in TTP is a consequence of a new uncommon locomotion task to be performed, and requires participation of the motor cortical system to initiate a new locomotion plan (figure 1). If a new locomotion plan does not resemble the current locomotion plans more time and practise is required to coordinate the task (31;33;104). One approach to reduce this variability might be to practice very slow walking before subjects are tested in the gait lab. This might allow speed-match comparisons to be more valid.

Consequently, researchers and clinical staff involved in gait analysis need to be aware of the underestimation of MPP using MPC, and clearly define how MPP is estimated in papers. In the present study, MPP was used to exemplify possible problems with the estimation of the MPP based on MPC. Same problems would be in measuring kinematics such as peak angles and maximal range of motion if based on a mean curve.

Differences in TTP for H1-S and H3-F in the IE were negatively correlated to walking speed. A possible explanation might be that hip extensor work is thought to be responsible for stabilisation of the trunk and preventing the lower extremity to collapse with initial contact and weight acceptance during gait (106). Similar positive hip abductor work is thought to ensure frontal stabilisation of the body during gait (1). Consequently, a consistent timing of the impaired hip extensor and abductor peak power generation during gait might be a precondition for an increase in walking speed for subacute stroke patients. Additionally, strong correlations between lower extremity power generation, absorption and walking speed has been observed in previous studies (9;11;13). If so, physical therapists involved in gait rehabilitation should focus training on the consistent timing of hip extensors and hip abductors during weight acceptance and single limb support



(figure 3). Primarily, to ensure body stabilisation, and secondly, to secure a sufficient basis for muscle power generation and absorption in the lower extremity and by this increase walking speed. No correlation was observed between differences in TTP for power variables in the UE and walking speed. An explanation might be that the UE does not determine walking speed in subacute stroke patients and compensates for the IE (9;10;12;13;107). Consequently, differences in the timing of power in the UE is not a major premise for increasing walking speed, which appears to be controlled by the less functioning IE.

An additional finding was the differences in TTP and in curve configuration for SW's hip abductor power observed in the frontal plane (H3-F) (figure 6). Difficulties in defining H3-F may provide a reason for this. The SW power curves showed the characteristic two peaks during stance (figure 4), but the second peak on the curve did not represent the H3-F. Instead, it represented the large amount of work by hip abductors to generate muscle power for hip circumduction to secure toe clearance in the IE. This difficulty was observed in SW during test one when defining H3-F. Equally difficult was defining the H3-F for the UE in both test one and two. Only one peak on the power curve was observed at weight acceptance during early stages in the gait cycle. This may represent an enhanced muscle work produced by hip abductors as it supports the lifting of the opposite pelvis above level position to ensure toe clearance of the impaired leg the initial swing phase. These abnormal characteristics are not unusual in hemiparetic patients and can vary individually between subjects (9). This must be recognized when interpreting power variables. Consequently, standard peaks (i.e. A2-S, H1-S, H3-S, H3-F and K3-S) originally based on healthy subjects must be critically evaluated in relation to the impairments observed in patients (1;21). This is particularly important in H3-F which was difficult to define in the described compensated gait pattern, and further investigations are still required.

Finally, caution must be undertaken when interpreting the study results as only 2 out of 13 stroke patients completed the full test protocol for test one and two during rehabilitation. Although, less pronounced findings suggest the same trend in 13 healthy subjects and 12 subacute patients later in their subacute rehabilitation, the findings are mainly hypothesis generating. Larger studies are required to confirm study findings in addition to investigations in more specific groups of stroke patients; i.e. state (chronic or subacute), type (haemorrhage vs. ischemic or cortical vs. subcortical), and perhaps even pathological gait patterns.

## *5.2. Hemiparetic lower extremity muscle groups may be target in early gait rehabilitation*

Lower extremity muscle group power is related to walking speed in chronic stroke patients (7-13), and achieving independent walking and “normal” gait appearance are of a high priority for stroke patients (6). Reference data on muscle group power may be one valuable tool to evaluate gait rehabilitation in stroke patients (progression/regression). In addition, it is important that future work seeks to explain changes in the gait speed of patients, and knowledge like this may assist clinicians to identify pathological gait patterns and to target gait rehabilitation. Even so, power analyses of subacute stroke patients with low gait performance have not been investigated in this manner, and are required to target gait training in this subgroup during early gait rehabilitation.

Only two trials have investigated changes in power and work variables (A2-S, H1-S, H3-S and K3-S), and was estimated following muscle strength and aerobic exercise training in chronic stroke patients (12;13). Teixeira-Salmela et al. (2001) reported no increase in A2-S, H1-S, H3-S and K3-S on the IE and UE with increases in gait speed (0.6 m/s to 0.76 m/s) (13). By contrast, Parvataneni et al. (2007) reported an increase in work variables A2-S on the IE and UE and in H3-S on the UE (integral of the power curve) when walking speed improved from 0.69 m/s to 0.83 m/s in 28 chronic stroke patients. An increased walking speed correlated to changes in the impaired A2-S and H1-S, only, and one regression model reported these to be predictors of improvements in walking speed. The second reported that improved A2-S in the IE and H1-S in UE extremity predicted improvements in walking speed (12). The improvements in IE and UE A2-S were comparable to the results in the present study (study 3) and the improvements were also strongly correlated to the change in walking speed ( $r=0.86$  and  $0.88$ ). This indicates that plantarflexion power in subacute and chronic patients is important for the generation of walking speed. In addition, K3-S in the IE significantly increased following six weeks of rehabilitation in present study, but more surprisingly, it did not correlate to changes in walking speed (table 4). As the patients in the present study were slow walkers, a possible explanation might be that the eccentric work of the knee extensors is prolonged during gait. This might increase the knee extensor eccentric work to control and stabilize the knee, and would partly modify its function, from being an important component at pre-swing contributing to body progression, to additionally being a stabiliser and controller of the lower limb.

The UE compensated for the IE by producing higher muscle group power during gait, but the improvements were not correlated to changes in walking speed. This indicated that a large

proportion of the muscle work of the UE was for body support to ensure safe ambulation. These patterns were identified in the H1-S, H3-S and K3-S of the UE. A possible explanation was proposed by Parvataneni et al. (2007) who stated that the IE was the “weak link” and responsible for the reduced gait speed. If so, improvements in walking speed are influenced by the “weak link”, and the UE, consisting of a residual muscle power capacity, easily follow the muscle power improvements in the IE, but does not correlate to walking speed. A second explanation for the increase in the H1-S in the UE could be that the hip extensor muscles compensate for the lack of power generation achieved by the ankle and hip in paretic extremity during pre-swing. Consequently, the H1-S of the UE is forced to generate a greater muscle power during weight acceptance to regain and secure body position (11). Unlike H1-S, H3-S and K3-S the improvement in H3-F on the UE was correlated with changes in walking speed. This inter-limb compensation secures foot clearance allowing the paretic limb to swing freely. Additionally, it secures an upright posture during gait in the frontal plane, as the increase in work by the hip abductors on the non-paretic side levels the paretic pelvis. This compensatory strategy is well-known in clinical practise and has been described previously (9). Furthermore, power curve configurations and time to peak power might be influenced by this compensating strategy (see 5.1.).

Even though no significant improvements were reported in H1-S, H3-F, and H3-S in the IE following gait rehabilitation, a strong relationship ( $r > 0.75$  (108)) was observed between improvements in these and the changes in walking speed ( $r = 0.81-0.89$ ). This indicates that in the present study the combined six weeks of gait intervention did not sufficiently affect hip muscle power in the IE and secondly, that patients might improve gait speed if these hip muscles are strengthened during gait rehabilitation. The same trends have been identified in a study by Teixeira-Salmela et al. (2001).

The healthy subjects improved in all investigated power variables following an increase in walking speed. Improvements in A2-S, H3-S and K3-S were only correlated to changes in walking speed, indicating that these variables are responsible for increased gait velocity in healthy subjects. By contrast, improvements in hip extensor muscle generation at weight acceptance (H1-S) and hip abductor concentric work during the single support stance (H3-F) appeared to be more involved in supporting the upright body position in the sagittal and frontal planes (1;95). Power strategies in healthy subjects were only partly observed in patients, as only A2-S and K3-S in the IE and H1-S in the UE increased following gait training. This may be due to the minor increase in walking velocity observed in patients, resulting in a diminished potential for power production de-

velopment in patients compared to healthy subjects. Although, H1-S and H3-F in the paretic extremity was observed to determine walking speed in patients, it was not observed in healthy subjects. This indicates that H1-S and H3-F compensates for the diminished muscle group power in A2-S, H3-S and K3-S in the paretic limb to ensure gait progression. Even so, improvements in A2-S and H3-S in the paretic extremity and A2-S in non-paretic extremity correlated to changes in walking speed when compared to healthy subjects.

Although power strategies in healthy subjects were partly adopted by patients, common determinants of walking speed improvements for subacute patients and healthy subjects were observed, and only few compensating power strategies were identified, i.e. hip abduction power in the UE. This could indicate that gait rehabilitation based on power strategies similar to healthy subjects seems reasonable for subacute patients.

Observed signs of an adopted healthy power strategy in the current study deviate from a previous study investigating power strategies in six patients with chronic stroke instructed to walk at a self selected comfortable walking speeds (approx. 0.38 m/s) (7). No improvements were detected in power variables (A2-S, H1-S, H3-S and K3-S) as walking speed improved. Differences in these results could be due to the different time post stroke (subacute vs. chronic) which could have induced a compensatory rigid pathological gait pattern in the chronic patients (9;56;57;109). In the present study, healthy subjects were not speed-matched to the final walking speed of the patients following gait rehabilitation. This might bias the comparison of power strategies between healthy subjects and patients. Even so, as observed in a previous study, improvements in power strategies were comparable with healthy subjects improving walking speed from very slow to slow and from slow to preferred walking speed (7). Therefore, we believe that this did not influence the study's findings.

Considerations with regards to the interpretation of the results are required, as six out of thirteen patients were tested using a walking aid (walking cane and support) at baseline and after training, and consequently, the support of the IE may have reduced the power magnitude. Therefore, it might be assumed, that the impaired H1-S and H3-F would be reduced in patients with support as the base of support is larger, and postural control is supported in frontal and sagittal plane during gait. A post study data analysis showed that patients without support improved walking speed more than patients with support (0.19-0.39 m/s vs. 0.36-0.73 m/s;  $p=0.02$ ). Not surprisingly, this is reflected in a general trend towards a larger muscle group power (A2-S, H1-S, H3-S, H3-F, and K3-S) and larger Functional Independence Measure (FIM at baseline; 91 vs. 81) in the patients

without support. This indicates that no change was present in the overall trends of the results, but changes were identified in magnitudes (power and speed). Obviously, it is difficult to determine whether or not the support influenced the muscle group power, and no gait analysis was undertaken to measure the patients muscle group power with or without a walking aid or support. Future studies should quantify the influence of support during gait in muscle group power of stroke patients.

Generally, biomechanical models do not account for muscle group power contributions from co-contracting antagonist muscles, and as a result, the muscle power generation and absorption observed might be underestimated compared to the true power value in current study.

### *5.3. Task specific gait interventions: The Lokomat<sup>®</sup> versus physical therapy*

Several studies have investigated the effectiveness of the Lokomat<sup>®</sup> on gait parameters such as walking independence, speed, and endurance (24-26;84-86), which are important rehabilitation outcome measures, as these may predict the ambulatory independence in community settings (110). However, little focus has been on improvements in gait quality in stroke patients attending robotic gait rehabilitation.

The Lokomat<sup>®</sup> is designed to produce an equal time in swing phase, mimicking symmetrical kinematic gait patterns comparable to those of healthy subjects (97;98). In addition, it might facilitate neural locomotion control systems in the midbrain and spinal cord as well as stimulating afferent input (tactile and proprioceptive) in a highly repetitive manner during training (33;35;37;78;79). Even so, no improvements were detected in step length ratio (SLR) and swing time ratio (STR) for patients with subacute stroke. The result is consistent with a previous study on ambulatory chronic stroke patients evaluating the SLR after Lokomat training and BWSTT (26), but differ from another study in which the Lokomat<sup>®</sup> improved SLR in chronic ambulatory stroke patients (85). Additionally, McCain et al. (2008) investigated the effect of BWSTT on STR and showed a distinct improvement in gait symmetry for subacute stroke patients with a small sample size (n=14) (111), and similar improvements were seen in gait symmetry of chronic stroke patients when practising treadmill training compared to overground training (99). Furthermore, Neckel et al. (2008) reported that chronic stroke patients following a one time Lokomat<sup>®</sup> session produced bilateral symmetrical hip range of motion (98). In addition, the current findings did not observe any significant differences in SLS between gait interventions at three weeks (LGO: 4.1 % vs. PT: 9.6 %) and six weeks (LGO: 1.3 % vs. PT: 0.85 %) or within intervention groups. Even though non-significant, PT showed an indication to a larger improvement in SLS following three weeks of in-

intervention compared to LGO, and might indicate that PT is more efficient in improving SLS in ambulatory stroke patients. The trend was observed by Hornby et al. (2008) who showed that BWSTT was more effective than Lokomat training for SLS following 12 training sessions (26). This indicates that a task-specific gait training with many repetitions improved time spend on the impaired leg for ambulatory subjects with chronic stroke. Even so, non-ambulatory stroke patients improved SLS following Lokomat<sup>®</sup> gait training (84).

A plausible explanation for lack of improvement in gait symmetry observed in the current study might be due mechanical limitations of the Lokomat<sup>®</sup> which imposes little or no leg movements in the frontal and transverse plane. Consequently, the Lokomat<sup>®</sup> compensates by increasing the maximal hip and ankle extension as well as maximal ankle and hip range of motion (97). These compensations might be difficult for subjects to transfer to symmetrical overground walking. These limitations of the Lokomat<sup>®</sup> combined with the study results might suggest a limited task-specific nature of Lokomat<sup>®</sup> training in measuring gait symmetry in ambulatory patients. Although PT was thought to overcome the abnormal gait pattern imposed by Lokomat<sup>®</sup> training, no differences were observed between intervention groups in the measured gait symmetry.

The findings of the current study, to some extent, confirmed the existing findings on the effectiveness of the Lokomat<sup>®</sup> on self-selected walking speed (SWS). In previous studies comparing the Lokomat<sup>®</sup> with PT or BWSTT, robotic training improved walking speed from 0.06 m/s to 0.12 m/s (0.08 m/s in our study) for dependent and independent walkers with stroke (25;26;84;85). In the current, PT increased walking speed by 0.26 m/s compared to 0.08 m/s in the Lokomat<sup>®</sup> during the first three weeks of training. Although not significant it indicated a larger effect of PT. In addition, the size of the changes in gait speed were clinically meaningful ( $\Delta$  speed > 0.1 m/s) during the six weeks of rehabilitation in both gait intervention groups (PT-LGO and PT-LGO) (112). Furthermore, participants improved their ambulation level from household ambulation (gait speed < 0.41 m/s) to limited community ambulation (gait speed > 0.41 m/s) in the intervention group PT-LGO and slightly below the limit for the intervention group LGO-PT (110). This indicates a clinical meaningful effect for both intervention groups, and demonstrates gait speed improvements of other gait interventions in subacute stroke rehabilitation (80;113-118).

In concurrence to previous studies (25;26;85), no differences were shown between gait interventions for ambulatory patients in the current study. As previously described, the combination of gait robotic and conventional gait training improved gait independence in non-ambulatory stroke patients (24), and Husemann et al. (2007) demonstrated that the Lokomat<sup>®</sup> alone increased

single limb support time on hemiparetic side in this subgroup of patients (84). This might suggest that, the Lokomat<sup>®</sup> is indicated in low walking stroke performers. In the current study, the same trend was recognised in this subgroup of participants. Two outliers, able to walk independently with walking aids, were randomized to intervention group LGO-PT. Both had an initial walking speed of approximately 0.06 m/s, a single support stance time below 8 % and a swing time of the IE six times larger than the UE. They improved similarly to the remaining patients, which indicate a possible benefit of Lokomat<sup>®</sup> training for patients that walk poorly. In addition, this observation supports the findings of Visintin et al. (1998) in which non-ambulatory stroke patients improved walking speed and endurance following BWSTT (80). These patients are a major challenge in conventional physical therapy as training duration and intensity are diminished due to the working load applied to the physical therapist. Consequently, more than one person is needed to assist during gait training.

Importantly, the Lokomat<sup>®</sup> is limited to movements in the sagittal plane and produces abnormal joint torque patterns during gait training in subjects with chronic stroke (97). In addition, the ankle joint are passively moved in the Lokomat<sup>®</sup> during training, and consequently little ankle plantarflexor activity is present (96;98). Furthermore, in the present study additional post study data analysis was performed, in which estimation of plantarflexion power revealed a larger improvement in the intervention group PT-LGO (0.76 W/kg) when compared to LGO-PT (0.15 W/kg) within the first three weeks of training (not shown in results 4.4.). Consequently, it might be reasonable to compensate these limitations during overground walking practice to improved gait parameters in ambulatory stroke patients. Therefore, Lokomat<sup>®</sup> gait training in addition to task-orientated PT (73;74;76) in which specific components of gait are targeted (e.g. A2-S, H1-S, H3-S, H3-F, and K3-S), might positively influence gait speed and gait symmetry in ambulatory subjects early after stroke. This is supported partly by the results of study three (see discussion in section 5.3.)

Some limitations were apparent in the current pilot study and must be addressed. First, the combination of restricted inclusion criteria (walking speed < 0.5 m/s; middle cerebral artery infarction), and limited recruitment time in the PhD project explain the small sample size. This limits the power of the study to detect a true difference between gait interventions (type 2-error). This was obvious within the first three weeks in which patients trained in the intervention group PT-LGO and experienced an improvement in walking speed of 0.26 m/s while patients in the LGO-PT group only experienced an improvement in walking speed of 0.08 m/s. The between group *p*-value was 0.06 borderline. If more subjects were included, statistical significance may have been reached

(e.g.  $p$ -value < 0.05). The same trend was present for SLS within the first three weeks. A second study limitation was the difference in time from stroke onset to intervention as the subjects in the intervention group LGO-PT were allocated to the study one month later (56 days vs. 21 days) than the group PT-LGO. The statistical data analysis for covariance (ANCOVA) was applied and an adjustment for time from stroke onset to intervention start was estimated for all outcome measures, and as expected differences between intervention groups became smaller and remained non-significant. This indicates that the time post stroke influenced the results, and might be one reason for the trend in differences observed between interventions at three weeks. The risk of an uneven distribution of patient characteristics (e.g. time from stroke onset to intervention, walking speed) is present in small sample sizes, and can be adjusted for by a block randomisation. In the current study, this might explain some of the positive effect of the PT-LGO group during the first three weeks with the measured parameters of SWS and SLS (table 6).

Although other rehabilitation studies have used a cross-over design to investigate the effect of gait interventions in stroke patients (86;88;119), it might not be the most appropriate study design. The cross-over design may introduce a carry-over training effect between gait interventions as no “wash-out” period was present. We tried to reduce this bias by randomizing patients into the two intervention groups, so that training effects between gait interventions would be equally distributed in the gait interventions. Even so, it is likely that both groups are not comparable with each other after the point of cross-over due to the accumulation of treatment effect in the intervention already received. Consequently, the change in outcome measures should be interpreted with caution after the first three weeks that show differences in effects between groups.



## 6. Conclusion and application of findings

Reference data with reference intervals were calculated in plantarflexion, hip extension, hip flexion, hip abduction and knee extension power in healthy subjects walking at their preferred speed, in which the demographic variables of gender, age, and height partly determined power. Reference power intervals may serve to guide and support realistic goal setting in gait rehabilitation as pathological gait peak power can be compared to healthy values. In the present thesis, the average mean A2-S in patients was approximately 0.43 W/kg and below the lower limit of the reference interval of a 178 cm tall, 65 year old healthy man (2.17 W/kg to 5.47 W/kg). This indicates that the reference intervals are useful in evaluating power variables in subacute stroke patients.

Mean power curves underestimated mean peak power compared to single power curves due to variability in the time to peak (TTP) and curve configurations in subacute patients and healthy subjects. Results of this pilot study suggest that MPP should be calculated by SPC. Additionally, variability in TTP in hip flexion and hip abduction power correlated negatively with walking speed in subacute stroke patients. This indicates that a consistent timing of impaired hip extensor and abductor peak power generation during gait might be a precondition for an increased walking speed in subacute stroke patients. Furthermore, the definition of hip abduction power (H3-F) must be critically evaluated, as H3-F was difficult to define in one low performing stroke patient. Larger studies are warranted to investigate this methodological issue in-depth.

Concentric ankle plantarflexion at push-off (A2-S) and eccentric knee extension power at pre-swing (K3-S) in the paretic extremity and hip flexion generation in non-paretic extremity at pull-off (H3-S) improved significantly following six weeks of gait rehabilitation. Improvements in A2-S, H1-S, H3-S and H3-F in the IE and A2-S and H3-F in the UE correlated with changes in walking speed. Consequently, it is recommended that the timing of hip extensors and hip abductors during weight acceptance and single limb support (study 2) in addition to practice muscle power of impaired ankle plantarflexors, hip extensors, hip flexors, hip abductors, and knee extensors to increase walking speed in low ambulatory subacute stroke patients (study 3).

In addition, healthy matched subjects increased power variables at the ankle, hip and knee significantly and improvements in A2-S, H3-S and K3-S were correlated to changes in walking speed. It has been debated whether or not that gait rehabilitation should target compensating walking strategies of stroke patients to reach a higher walking performance (9;13;56;107;120-122) instead of practising symmetrical gait patterns similar to healthy subjects (107;122). The results of

the present study indicate that subacute stroke patients use power strategies that are partly similar to that of healthy subjects (study 3). This indicates that gait training might include exercises supporting power strategies similar to healthy subjects in early stroke rehabilitation.

The order of gait training interventions, including Lokomat<sup>®</sup> and physical therapy, did not appear to influence gait qualities in subacute ambulatory stroke patients. Additionally, no improvements were observed in gait qualities within the intervention groups, however all subjects improved their overground walking speed following three and six weeks of intervention. Results of previous studies reported, that the Lokomat<sup>®</sup> did not induce active ankle plantarflexion or hip abduction torques during gait in healthy subjects or chronic stroke patients, respectively (96;98). This might indicate that the Lokomat<sup>®</sup> is inadequate to increase gait speed alone, as it was observed, that an increase in plantarflexor and hip abductor power was important to increase walking speed in ambulatory patients (study 3). Consequently, in search of additional effects of the Lokomat<sup>®</sup> for low ambulatory subacute stroke patients an interesting randomised control trial could compare the highly repetitive BWSTT to the Lokomat<sup>®</sup> in which task specific physical therapy targets relevant hemiparetic muscle group power (A2-S, H1-S, H3-S, H3-F, and K3-S) are added in both intervention groups. Gait analysis could be performed to evaluate the effect (i.e. gait symmetry, gait speed, and muscle group power).

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## 8. Summary in English

The present thesis deals with 3D gait analysis in healthy subjects and subacute stroke patients. The overall aim of this PhD thesis was to utilize gait analysis to estimate reference power data, and to estimate changes in lower extremity major muscle group power during an increase in gait speed in healthy subjects and subacute patients. In addition, task specific gait interventions (Lokomat<sup>®</sup> and physical therapy) were evaluated in slow walking subacute stroke patients. Hopefully, findings will contribute to target gait training in a subgroup of stroke patients during early rehabilitation. The thesis is based on four papers which are submitted to peer-reviewed journals.

In present thesis, muscle group power generation and absorption of plantar flexors, hip extensors, hip flexors, hip abductors, and knee extensors were estimated during preferred walking speed (study one to three). In addition, gait parameters consisted of gait speed and gait symmetry expressed as an (1) absolute step length ratio, (2) swing time ratio, and (3) single limb support time in the impaired extremity (study four).

In short, reference data including reference intervals were estimated and seemed suitable as a clinical evaluating tool for comparison to stroke patients' power generation during rehabilitation. Estimation of a mean peak power was underestimated based on peaks derived from mean power curves compared to estimations based on peaks derived from single power curves. Changes in hemiparetic muscle group power correlated strongly to changes in walking speed in subacute stroke patients and resembled in part healthy subjects' power strategies, whereas no differences were found between gait interventions during six weeks of rehabilitation measured on gait symmetry.

In summary, reference power intervals determined by gender, age, and height are useful in evaluating power variables during gait rehabilitation, and the estimation of peak power variables are recommended to be based on individually peaks in single power curves. Further, it is recommended, that timing of impaired hip extensors and hip abductors during weight acceptance and single limb support is practised in addition to target muscle group power of impaired ankle plantarflexors, hip extensors, hip flexors, hip abductors, and knee extensors in gait rehabilitation of subacute stroke patients. In addition, no additional benefits of the Lokomat<sup>®</sup> measured on gait symmetry and gait speed was identified in low ambulatory stroke patients, but larger randomised controlled trials are warranted to confirm these findings.

## 9. Summary in Danish

Denne ph.d. afhandling omhandler 3D ganganalyse af raske og apopleksipatienter. Formålet med afhandlingen var at estimere referenceværdier for power i et udvalg af underekstremitetens muskelgrupper, samt at estimere ændringer i muskelgruppers power, når ganghastigheden øges blandt raske og apopleksipatienter. Ligeledes blev effekten af to opgave-specifikke ganginterventioner (Lokomat<sup>®</sup> og fysioterapi) vurderet i relation til langsomt gående subakutte apopleksipatienter. Forhåbentligt kan resultaterne bidrage til at målrette gangtræning til denne gruppe af apopleksipatienter. Ph.d. afhandlingen er baseret på fire artikler, som alle er indsendt til ”peer-review” tidsskrifter.

Power af plantarfleksorerne, hofteekstensorerne, hoftefleksorerne, hofteabduktorerne og knæekstensorerne blev vurderet i et 3D ganganalysesystem ved en foretrukken ”normal” ganghastighed (studie et til tre). Ligeledes blev ganghastighed og gangsymmetri, målt som (1) absolut skridtlængde ratio, (2) en svingfase ratio og (3) enkeltstandfase tiden på hemiparetiske ben (studie fire), vurderet ved hjælp af ganganalyse.

Resultaterne viste, at reference power værdier beskrevet med et forventet ”normalområde” var et anvendeligt klinisk redskab til vurdering apopleksipatienters muskelgruppe power under gangrehabilitering. Bestemmelsen af den gennemsnitlige maksimale power værdi blev underestimeret, hvis denne var udtrukket fra en gennemsnitskurve sammenlignet med en gennemsnitlig maksimal power værdi baseret på maksimale værdier fra flere individuelle enkeltkurver vurderet på samme person. Ændringer i hemiparetiske muskelgruppers power korrelerede stærkt med en øget ganghastighed blandt subakutte apopleksipatienter, hvilket til dels lignede raske personers power strategier. Derimod var der ikke forskel mellem ganginterventionerne efter seks ugers intensiv gangtræning vurderet på gangsymmetri og ganghastighed.

Et ”normalområde” for power værdier i underekstremitetens muskelgrupper baseret på de forklarende variabler køn, alder og højde synes at være et brugbart klinisk redskab, og beregningerne af en persons gennemsnitlige maksimale power værdi bør baseres på individuelle enkeltkurver. Endvidere anbefales det at minimere variationen i timing af hofteekstensorernes og hofteabduktorerne maksimale arbejde i gangens initiale fase samt enkeltstandfase gennem træning, samt at målrette træning af plantarfleksorerne, hofteekstensorerne, hoftefleksorerne, hofteabduktorerne og knæekstensorerne i det hemiparetiske ben under gangrehabilitering af subakutte patienter. Der var ingen indikationer på en effekt af Lokomat<sup>®</sup> træning vurderet i relation til gangsymmetri eller

ganghastighed til langsomt gående apopleksipatienter, men større randomiserede kontrollerede studier bør iværksættes til at bekræfte dette resultat.

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