EFFECTS OF PROGRESSIVE RESISTANCE STRENGTH TRAINING ON HEMIPARETIC GAIT

RATAKORN AIMKOSA

A THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR
THE DEGREE OF MASTER OF SCIENCE (SPORTS SCIENCE)
FACULTY OF GRADUATE STUDIES
MAHIDOL UNIVERSITY
2011

Thesis
entitled
EFFECTS OF PROGRESSIVE RESISTANCE STRENGTH TRAINING ON HEMIPARETIC GAIT

Ratakorn Aimkosa
.....
Mr. Ratakorn Aimkosa
Candidate

.....
Asst.Prof. Opas Sinphurmsukskul, M.D.
Major advisor

Vimonwan Hiengkaew
.....
Assoc.Prof. Vimonwan Hiengkaew, Ph.D.
Co-advisor

Kittipong Poonchob
.....
Mr. Kittipong Poonchob, Ph.D.
Co-advisor

Metta Pathong
.....
Mrs. Metta Pinthong, Ph.D.
Co-advisor

B. Mahaisavariya
.....
Prof. Banchong Mahaisavariya, M.D., Dip Thai Board of Orthopedics
Dean
Faculty of Graduate Studies
Mahidol University

Arth Nana
.....
Prof. Arth Nana, M.D.
Program Director
Master of Science Program in Sports Science
College of Sports Science and Technology
Mahidol University

Thesis
entitled

EFFECTS OF PROGRESSIVE RESISTANCE STRENGTH TRAINING ON HEMIPARETIC GAIT

was submitted to the Faculty of Graduate Studies, Mahidol Univerity
for the degree of Master of Science (Sports Science)
on
February 28, 2011

Ratakorn Aimkosa
.....
Mr. Ratakorn Aimkosa
Candidate

Kittipong Poonchob
.....
Mr. Kittipong Poonchob, Ph.D.
Member

Siriporn Sasimotonkul
.....
Asst.Prof. Siriporn Sasimotonkul, Ph.D.
Chair

Metta Pinthong
.....
Mrs. Metta Pinthong, Ph.D.
Member

.....
Asst.Prof. Opas Sinphurmsukskul, M.D.
Member

Weerawat Limroongreongrat
.....
Mr. Weerawat Limroongreongrat, Ph.D.
Member

Vimonwan Hiengkaew
.....
Assoc. Prof. Vimonwan Hiengkaew, Ph.D.
Member

B. Mahaisavariya
.....
Prof. Banchong Mahaisavariya, M.D., Dip Thai Board of Orthopedics
Dean
Faculty of Graduate Studies
Mahidol University

Arth Nana
.....
Prof. Arth Nana, M.D.
Dean
College of Sports Science and Technology
Mahidol University

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude and deepest appreciation to Assistance Professor Opas Sinphurmsukskul, my major advisor. The deepest gratitude also goes to Associate Professor Vimonwan Hiengkaew, Mr. Kittipong Poonchob, and Mrs. Metta Pinthong, my co-advisor. They always supervise me with excellence knowledge, valuable suggestion, proofreading of this manuscript and encouragement throughout the study.

I would like to thank Miss. Worawarun Methesakulkarn who always helped and encouraged me throughout this study.

I would like to give a special thanks to all of my subjects for their good participation. My gratitude also extend to the officers in the Physical Medicine and Rehabilitation section, Charoenkrungpracharuk Hospital for their helpful communication that made this study flowed fluently. Additionally, I would like to thank the staff of College of Sports Science and Technology, Mahidol University for their friendly relationship during working in the laboratory.

This thesis is dedicated with deepest love and affection to my family. It might stressful to complete the study without them. Their love, encouragement and understanding have inspired me to be the best I can be. And I hope this thesis is the best thing that represents my thankfulness to all of them.

Ratakorn Aimkosa

EFFECTS OF PROGRESSIVE RESISTANCE STRENGTH TRAINING ON HEMIPARETIC GAIT

RATAKORN AIMKOSA 4836659 SPSS/M

M.Sc. (SPORTS SCIENCE) MAJOR IN SPORT BIOMECHANICS

ADVISORY COMITTEE: OPAS SINPHURMSUKSKUL, M.D., VIMONWAN HIENGKAEW, Ph.D., Mr. KITTIPONG POONCHOB, Ph.D., Mrs. METTA PINTHONG, Ph.D.

ABSTRACT

Progressive resistance strength training is a well-known method used for increasing muscle strength. However, the effectiveness and safety of progressive resistance strength training for post-stroke rehabilitation are not well known. The purpose of this study was to compare the effects of progressive resistance strength training and conventional strength training by investigating isokinetic average peak torque of lower limb muscles, walking ground reaction force, standing postural sway, physiological cost index, and maximum walking speed of post-stroke hemiparetic patients. Sixteen hemiparetic patients volunteered for this study. The training group (n = 8) participated in supervised progressive resistance strength training intervention in lower limb muscles 3 days/week for 6 weeks, and the control group (n = 8) continued their usual conventional strength training intervention. Both groups were matched by age, weight, height, post stroke duration, motor impairment, muscle tone (Modified Ashworth Scale), Functional Ambulation Classification scores, Postural Assessment Scale for Stroke patients scores, and Barthel index of activities of daily living scores.

Results reveal that progressive resistance strength training intervention could significantly improve both paretic and non-paretic isokinetic average peak torque of lower limb muscles, physiological cost index, and maximum walking speed compared to conventional strength training intervention during the 6-week training period ($p < 0.05$). However, there were no significant improvements in walking ground reaction force and standing postural sway after 6 weeks of both progressive resistance strength training intervention and conventional strength training intervention.

These finding conclude that progressive resistance strength training intervention is an appropriate strength training program for improving lower limb muscle strength and gait performance in hemiparetic patients.

KEY WORDS: PROGRESSIVE RESISTANCE STRENGTH TRAINING /HEMIPARETIC/GAIT

117 pages

ผลของการฝึกความแข็งแรงโดยการเพิ่มแรงต้านแบบก้าวหน้าต่อลักษณะท่าทางการเดินในผู้ป่วยอัมพฤกษ์ครึ่งซีก

EFFECTS OF PROGRESSIVE RESISTANCE STRENGTH TRAINING ON HEMIPARETIC GAIT

นาย รฐกร เอมโกษา 4836659 SPSS/M

วท.ม. (วิทยาศาสตร์การกีฬา)

คณะกรรมการที่ปรึกษาวิทยานิพนธ์: โอภาส สินเพิ่มสุขสกุล, M.D., วิมลวรรณ เหียงแก้ว, Ph.D., กิตติพงศ์ พูลชอบ, ปร.ด., เมตตา ปิ่นทอง, ปร.ด.

บทคัดย่อ

การฝึกความแข็งแรงโดยการเพิ่มแรงต้านแบบก้าวหน้า นั้นเป็นที่รู้จักและนิยมใช้ในการฝึกเพื่อเพิ่มความแข็งแรงของกล้ามเนื้อ อย่างไรก็ตามเนื่องจากประสิทธิภาพและความปลอดภัยของการฝึกชนิดนี้นั้นทำให้ยังไม่เป็นที่นิยมใช้กันในกระบวนการฟื้นฟูผู้ป่วยที่มีอาการอัมพฤกษ์ครึ่งซีก จุดประสงค์ของการศึกษาในครั้งนี้เพื่อเป็นการการเปรียบเทียบผลที่ได้จากการฝึกความแข็งแรงโดยการเพิ่มแรงต้านแบบก้าวหน้ากับการฝึกเพิ่มความแข็งแรงของกล้ามเนื้อแบบดั้งเดิมในผู้ป่วยอัมพฤกษ์ครึ่งซีก โดยการศึกษาการเปลี่ยนแปลงของค่า Isokinetic average peak torque ของรยางค์ส่วนล่าง ค่าแรงปฏิกิริยาของพื้นขณะเดิน ค่าความสามารถในการทรงตัวในขณะยืน ค่า physiological cost index ขณะเดินและ ค่าความเร็วสูงสุดในการเดิน การศึกษาในครั้งนี้มีผู้ป่วยอัมพฤกษ์ครึ่งซีกสมัครใจเข้าร่วมทั้งหมด 16 คน แบ่งเป็นกลุ่มทดลอง 8 คนใช้วิธีการฝึกโดยการเพิ่มแรงต้านแบบก้าวหน้าในรยางค์ส่วนล่าง 3 วันต่อสัปดาห์เป็นระยะเวลา 6 สัปดาห์ กลุ่มควบคุม 8 คน ใช้วิธีการฝึกเพิ่มความแข็งแรงแบบดั้งเดิม ทั้ง 2 กลุ่มไม่มีความแตกต่างกันในเรื่องของ อายุ น้ำหนัก ส่วนสูง ระยะเวลาในการป่วย โทนของกล้ามเนื้อ (Modified Ashworth Scale) คะแนนของแบบประเมินความสามารถในการเลื่อนย้ายร่างกาย แบบประเมินความสามารถในการทรงตัว และดรรชนีความสามารถในการทำกิจกรรมประจำวันของ Barthel

ผลของการศึกษาแสดงให้เห็นว่าการฝึกเพิ่มความแข็งแรงโดยการเพิ่มแรงต้านแบบก้าวหน้าในระยะเวลา 6 สัปดาห์นั้นทำให้ค่า Isokinetic average peak torque ของรยางค์ส่วนล่างเพิ่มขึ้นทั้งข้างที่อ่อนแรงและข้างปกติ เพิ่มขึ้น ค่า physiological cost index ที่ใช้ในการเดินลดลงและความเร็วสูงสุดในการเดินเพิ่มขึ้นอย่างมีนัยสำคัญมากกว่าการฝึกเพิ่มความแข็งแรงแบบดั้งเดิม อย่างไรก็ตามไม่พบการ ปรับปรุงของค่าแรงปฏิกิริยาของพื้นขณะเดินและความสามารถในการทรงตัวขณะยืนหลังจากการฝึก 6 สัปดาห์ในการฝึกทั้ง 2 แบบ

การศึกษาในครั้งนี้สรุปว่าการฝึกความแข็งแรงโดยการเพิ่มแรงต้านแบบก้าวหน้านั้นเป็นวิธีการที่มีความเหมาะสมในการเพิ่มความแข็งแรงของกล้ามเนื้อรยางค์ส่วนล่างและเป็นปรับปรุงประสิทธิภาพในการเดินของผู้ป่วยที่มีอาการอัมพฤกษ์ครึ่งซีก

117 หน้า

CONTENTS

CONTENTS (cont.)

CONTENTS (cont.)

LIST OF TABLES

LIST OF FIGURES

LIST OF FIGURES (cont.)

CHAPTER I
INTRODUCTION

Cerebrovascular accident (Stroke) is the leading cause of adult disability in Thailand (1). Stroke results from cell damage and impaired neurological function that occurs from ischemic or hemorrhage into the brain tissue (2), leading to a sudden, focal neurological deficit. The physical effects of stroke are varies and which include several impairments such as motor, emotion, sensory systems, language, perception, and cognitive function (3). Impairment of motor function involves paralysis or paresis of the muscle on one side of the body contralateral to the site of the brain lesion (3). Damage to the descending neural pathways result in abnormal regulation of spinal motorneurons, causing alterations in postural stretch reflexes and voluntary movement (4). Abnormalities in the temporal and spatial recruitment of motor units slow the ability of muscles to generate tension, leading to prolonged agonist contractions (4).

Gait performances in stroke patients are characterized by slower gait velocity with associated weakness, spasticity, and abnormal central neural pattern of muscle activation, can markedly reduce the gross motor efficiency of ambulation (5). Reduced cardiovascular fitness, along with the increased energy demands associated with hemiparetic gait, may contribute to low ambulatory activity levels and to poor rehabilitation outcomes (6). Empirical data have shown that gait velocity of stroke patients varying depends on severity ranges from approximately 0.18 to 1.03 m/s, (7-10) whereas velocity of healthy adults of similar ages average about 1.4 m/s (11). Olney et al (12) and Wade et al (10) reported that there were increased in mechanical energetic costs during hemiparetic gait when compared with normal gait. Wall and Turnbull (13) and Hill et al (14) reported that the asymmetry ratio of the single support time between the 2 lower extremities (temporal asymmetry) was approximately 0.53 to 0.66 for patients with moderate stroke.

Faster gait velocity and better gait patterns (or appearance) are perceived by many stroke patients as their ultimate goals of rehabilitation (15,16). To enable stroke patients to achieve these goals, therapists designing a gait-training program should first identify the primary underlying impairments that account for the reduced gait velocity and abnormal gait pattern of these patients. Impairments in muscle strength (8,17-28), motor (7,17,22), sensory functions (7,29), visuospatial perception (30), spasticity (8,20), and balance (17,18,22,25,26,29) have been suggested to be related to inability of hemiplegic patients to walk in a normal fashion. However, the impairments that are the most importance factors in determining the gait velocity and gait pattern of these patients remain unknown.

Many researchers have shown that the strength of the paretic hip flexors (22,27,28), knee extensors (18-21,23-26), and ankle plantarflexors (18,27) correlate with comfortable and/or fast gait velocities in patients with stroke of varying severity. By using the isokinetic strength-testing method, Nadeau et al (22), showed that the strength of the paretic hip flexors was associated with comfortable ($r=0.83$) and fast walking speeds ($r=0.88$) in patients with mild to moderate stroke. Suzuki et al (26), also using isokinetic testing, reported that the strength of the paretic knee extensors was associated ($r=0.85$) with fast walking speed in patients with mild to moderate stroke. By using kinetic analysis, Olney et al (27) found that the positive work generated by the paretic ankle plantarflexors and hip flexors in late stance to early swing of the gait cycle correlated ($r=0.66$, $r=0.46$, respectively) with comfortable walking speeds of patients with hemiplegia after mild to severe stroke. However, there is still no consensus regarding the strength of which of these muscle groups is the most important in determining comfortable or fast gait velocity of patients with stroke.

Most researches that found relative relationships between muscle strength and gait velocity in stroke patients used isokinetic peak torque measures of strength (22-26). However, it has been suggested that total work is another functional measure of muscle strength, because total work tasks into consideration the ability of muscle group to generate torque throughout an arc of motion (31). It remains unknown as to

whether total work strength measures would show greater correlation with gait performance than peak torque strength measures.

The kinetic analysis of gait is essential to proper interpretation of human locomotion. The force platform has been used to measure the ground reaction forces. [It consists of a rigid platform suspended on piezoelectric. The force platform is set at the center of walkway]. During walking, the force of stance foot on the force platform is recorded. The electrical output signals produce three spatial planes of forces. The length of vector represents the quantity of applied force which is normalized by body weight (%BW). Ground reaction force is developed which is equal magnitude and opposite direction to the foot which applies on the ground. The ground reaction force is regarded as a representative measurement of gait, because it is the external force involved in walking and it affects the acceleration of the body's center of mass (32). The goal of locomotion is to drive the center of mass stably in the desired direction. It is that the kinetic level that cause of movement rather than at the kinematic level and therefore, ground reaction force may be a more appropriate global parameter to characterize gait than kinematics such as step length or step width. To understand the contribution of lower limb locomotion, it is necessary to analyze vertical, medial-lateral, and anterior-posterior ground reaction forces. The analysis of vertical forces during gait provides information on weight bearing, analysis of medial-lateral forces provides information on balance performance, and anterior-posterior forces provide information on speed control.

In normal subjects, Benedetti et al (33) reported that the vertical ground reaction force at loading response was 112±8.9%BW, at midstance was 73±6.8%BW, and at terminal stance 113.8±6.5%BW. The anterior-posterior ground reaction force at loading response was 43±3.4%BW, at midstance -19.3±4.2%BW, and at terminal stance 21.6±4.3%BW. The medial-lateral ground reaction force at loading response was -4.7±2%BW, at midstance 6.6±2.3%BW, and at terminal stance 5.5±1.9%BW. Vachalathiti and Impoolsub (34) showed no significant differences either in force or temporal parameter between left and right lower extremities for all directions, except medial-lateral shear force in the female subjects. For vertical ground reaction force,

two peak forces occurred at approximately 25 and 78 %stance. Mean values of peak vertical ground reaction force were between 106.6 and 117.79%BW, between 18.61 and 22.2%BW for anterior-posterior ground reaction force, and between 4.09 and 5.31%BW for medial-lateral ground reaction force in the case of walking with preferred speed. Nilsson and Thorstensson (35) studied the variation of ground reaction force parameters at different speeds of walking and running found that during walking, time at first peak force of vertical ground reaction force occurred earlier in higher speeds whereas time at second and third peak force of vertical ground reaction force occurred later in higher speeds. The breaking and propulsive peaks of anterior-posterior ground reaction force occurred earlier at higher speeds. During running, reaction force events in vertical, anterior-posterior, and medial-lateral occurred relatively later with increasing speed. During both walking and running, the amplitude of ground reaction force in all directions increased at higher speeds. In contrast, total vertical ground reaction forces and total medial-lateral forces decreased at higher speeds. During walking, propulsive force was higher than the breaking force at higher speeds. However, in running the breaking and propulsive forces of anterior-posterior ground reaction forces were equal at all speed. In hemiparetic subjects, Bowden et al (36) have found that anterior-posterior ground reaction forces were correlated with walking speed and hemiparetic severity. The percentage of total propulsion generated by the paretic leg was calculated and found to be 16%, 36%, and 49% for those with high, moderate, and low hemiparetic severity, respectively.

The association between standing balance ability and gait performance of stroke patients also remains to be determined. Some investigators have shown moderate correlation between standing balance and gait performance of patients with stroke of varying severity (17,22,25,26,29). Nadeau et al. (22), for example, found that the Fugl-Meyer balance sub score was associated with comfortable and fast walking speed ($r=0.51$, $r=0.57$, respectively; $P<0.05$) in patients with mild to moderate stroke. Suzuki et al (26), however, reported no significant correlation ($r=-0.32$, $P>0.05$) between standing balance and maximal walking speed for stroke patients with good standing balance ability, that is, those showing less than 30cm of center of foot pressure path in 10 seconds of standing. Similarly, Winstein et al (37) found that

standing balance training that led to better improvement in standing symmetry failed to result in greater improvement in hemiplegic gait symmetry of patients with moderate stroke.

In this study, the relationship between a subject's standing balance and gait performance were not investigated, the researcher compared the different of the standing postural sway between groups only, because of balance deficit by itself may result from a combination of several direct impairments (such as muscle weakness and sensory impairment) and indirect impairments (such as joint contracture) (38). Thus, investigation of the relationship between standing balance and gait performance in stroke patients may not contribute much to the identification of the primary direct impairments determining hemiplegic gait.

In hemiparetic patients, low endurance to exercise may compound the increased energy cost of movement and contribute to poor rehabilitation outcomes. The measurement of oxygen consumption requires expensive equipment and specialized knowledge in data reduction and analysis, as well as time-consuming procedures in a metabolic testing laboratory. Therefore, it is important to identify a proxy measure of oxygen cost that can be realistically performed in the clinical environment. The Physiological Cost Index (PCI), has been used as a simple, indirect measure of oxygen cost during exercise (39). The PCI is defined as follow: (heart rate during steady-state exercise minus heart rate at rest) divided by walking speed.

The PCI has the potential to serve as an index of oxygen cost for a given individual because of the relationship of both oxygen consumption and heart rate to cardiac output (40). During exercise, greater oxygen demand is created in the muscle. Increased oxygen demand induces sympathetic stimulation of the heart, which increases cardiac output (heart rate × stroke volume). Cardiac output and heart rate are linearly related (except for a plateau effect of cardiac output at maximum heart rates). Greater oxygen demand in the muscle and greater cardiac output result in higher oxygen consumption. These relationships among oxygen consumption, cardiac output,

and heart rate (41) support and investigation regarding weather PCI (based on heart rate) can be useful as an index of oxygen cost in a clinical setting for stroke survivors.

The PCI is a simple measure that can be performed by a clinician using a heart rate monitor. In a standardized treadmill test, the PCI had a high day-to-day reproducibility for healthy adults (a correlation of r=0.868 for steady state condition) (42). Hood et al (43) found that the PCI was more reproducible in repeated trials than oxygen cost. The PCI has been used in studying the gait performance of normal individuals, children, and subjects with spinal cord or head injury (39,42,44-47).

Muscle weakness can be a major factor contributing to disability after stroke (48,49). Studies of pattern of muscle weakness after stroke have indicated that the non-paretic lower limb is weaker than normal, flexors and extensors are similarly affected in the paretic lower limb (50,51). Several stroke studies have shown that weakness is associated with reduced walking speed and endurance (24,52), the need for assistance to enable walking (18) and difficulty independently transferring from one position to another (53). Studies of the relationship between the strength of paretic lower limb muscle groups and locomotors performance have shown that strength of lower limb muscle such as hip flexor, knee extensor and ankle plantarflexor muscles influence gait velocity (21,54,55). Kim and Eng (54) highlighted non-paretic limb muscle strength as a contributor to gait velocity. Variation exists in the relationship between strength of specific lower limb muscles and gait velocity but knee extensors are consistently reported as determinants of gait velocity (21, 55). Progressive resistance strength training refers to progressive increases in resistance to a muscle as training induces greater ability to produce and sustain force (56). The key elements of progressive resistance strength training are to provide sufficient load (resistance) so that only a relative small number of consecutive repetitions (usually less than 12) can be completed before fatigue, to progressively increase the amount of resistance as strength increase, and, to continue the program for a sufficient duration (a minimum of 4 weeks) for benefits to accrue (56). However, some clinicians have suggested that resisted exercises should be avoided after stroke, due to the risk of increasing spasticity (57). Others argue that muscle weakness is a major factor that impairs motor

performance (58-60) and that progressive resistance exercises reduce musculoskeletal impairment and functional disability (59).

The aim of this study is to investigate the effects of strength training programs in hemiparetic patients.

Objectives of the study

1. To compared isokinetic variables (average peak torque) between baseline and 6-week training in each group, and between groups.
2. To compared kinetic variables of gait (vertical ground reaction force, medial-lateral ground reaction force, and anterior-posterior ground reaction force) during stance phase between baseline and 6-week training in each group.
3. To compared standing postural sway (sway velocity, sway area, AP sway, and ML sway) between baseline and 6-week training in each group, and between groups.
4. To compared gait performance (Physiological Cost Index) between baseline and 6-week training in each group, and between groups.
5. To compared maximum walking speed between baseline and 6-week training in each group, between groups.

Hypotheses of the study

The progressive resistance strength training should improve muscle average peak torque, vertical ground reaction force, anterior-posterior ground reaction force, medial-lateral ground reaction force, postural sway, physiological cost index, and maximum walking speed more than conventional strength training.

CHAPTER II
LITERATURE REVIEW

2.1 General aspect of stroke

2.1.1 Definition of stroke

Stroke is a non-traumatic brain injury caused by occlusion or ruptures of cerebral blood vessels that result in sudden neurological deficit characterized by loss of motor control, altered sensation, cognitive or language impairment, disequilibrium, or coma. This definition includes an array of etiological sources but excludes nonvascular conditions that can present with stroke-like symptoms, such as seizure, syncope, traumatic brain injury, or brain tumor (61).

Stroke is one of the most important causes of physical disability in people over 60 years of age. Hemiplegia is the prominent characteristic of stroke. The term of hemiplegia is paralysis of one side of the body. In practice, hemiplegic is loosely applied to features not included in the strict definition. Therefore, a patient who has a paresis (weakness) rather than a complete paralysis is also commonly labeled as hemiplegia (62).

2.1.2 Stroke pathophysiology

2.1.2.1 Ischemic Stroke

The Unifying pathophysiology of thrombotic, embolic, and lacunar stroke is ischemia from compromise of cerebral blood flow. The location and temporal development of cerebral injury vary with the etiology.

Thrombosis

The entire pathophysiology of infarction from cerebral thrombosis remains controversial, but it is strongly associated with atherosclerotic cerebrovascular disease. Atherosclerotic plaque formation occurs frequently at major

vascular branching sites, including the common carotid and vertebrobasilar arteries. The disease occurs often in the presence of chronic hypertension, with injury to the intimal surface followed by macrophage infiltration and cholesterol accumulation within the vascular media. Foam cells develop within the vessel wall and cholesterol streaks appear on the endothelial surface. Eventually, calcification and vessel wall thickening compromise blood flow, leading to turbulence. Cracking of the plaque and further intimal damage can promote initial thrombus formation by stimulating platelet aggregation and activation of the extrinsic pathway of the coagulation system. The loosely attached thrombus, or "white clot," that rapidly forms is composed of platelet cell and fresh fibrin (63).

It is unclear whether symptoms of TIA are caused by transient thrombotic occlusion of major cerebral arteries or by microemboli that break away from a thrombus, but both phenomena might be important. Symptoms of transient monocular blindness, or amaurosis fugax, are likely due to microemboli from the internal carotid artery that cause a branch occlusion of the ispsilateral ophthalamic artery (64). Similarly, other intracranial branch occlusions can result from microemboli arising in the extracranial vessels, leading to injury or infarction in focal regions.

In contrast, a large arterial thrombus can occlude a major extracranial artery, producing a low-flow state that causes ischemic injury to neural tissue supplied by the most distal arterial branches (65). The volume of damage that results from such hemodynamic compromise can be quite large, but it is dependent on the length of time the vessel is occluded, the rate of flow through the occluded site, and the effectiveness of the collateral circulation. Fribinolytic enzymes are released that control acute thrombus formation, potentially dissolving the clot within minutes to hours. However, recanalization might fail or be delayed, permitting the arterial thrombus to completely or partially occlude blood flow. Collateral circulation can support the compromised cortical zone, but it may be less effective in elderly persons or in those with diffuse atherosclerotic disease or diabetes.

Ischemic injury from a cerebrovascular thrombus likely results in simultaneous distal branch occlusions from microemboli and compromise of blood flow proximally. The neurological outcome of cerebral thrombi varies widely and can include brief TIAs, minor strokes without functional compromise, or major strokes resulting in significant impairment and functional disability.

Embolism

Beyond the microemboli produced by cerebrovascular thrombi, the majority of embolic strokes have a cardiac origin. Thrombus formation within the cardiac chambers is generally caused by structural or mechanical changes within the heart. Arterial fibrillation is a significant risk factor for embolic stroke as a result of poor arterial mobility and outflow, with stasis of blood and arterial thrombus formation. Arterial fibrillation is often caused by rheumatic valvular disease or coronary artery disease, but it can be idiopathic. Mural thrombus within the left ventricle after MI, in the presence of cardiomyopathy or after cardiac surgery, is the other major cause of embolic stroke (66,67). Mechanical heart valves universally cause cerebral emboli if anticoagulation is insufficient. Infectious endocarditis can lead to septic emboli.

Paradoxical embolism is a rare cause of embolic stroke that occurs in the presence of a deep vein thrombosis (DVT) and an arterial or ventricular septal defect (68). Typically, the DVT embolizes to the lung, causing a pulmonary embolus and an acute increase in pulmonary arterial resistance and right ventricular pressure. In the presence of a septal defect, the raised pulmonary pressure leads to a right-to-left shunt. Subsequence emboli cross through the septal defect into the left chambers and systemic circulation, resulting in peripheral emboli and stroke. On modern echocardiography, a patent foramen ovale is a frequent finding, indicating that paradoxical embolism might be more common than previously thought (67).

Cerebral emboli lodge within arterial branches of the major arteries, causing single or multiple branch occlusions resulting in sudden focal neurological impairment. These branch occlusions significantly compromise flow

distally, inducing ischemic injury to neural tissue, glia, and vascular endothelium. Reperfusion of the occluded vessel can occur in response to endogenous fibrinolysis, but because ischemic damage to the vascular bed is often significant, the capillaries become incompetent and secondary cerebral hemorrhage ensures.

In contrast to thrombotic stroke, microemboli probably do not precede cardioembolic stroke, as TIAs are uncommon. Frequently cardiac thrombus can be found after the event, and the only clue to an embolic cause is the sudden neurological deficit without previous or progressive symptoms.

Lacunes

Lacunar infarcts are small, circumscribed lesions that measure less than 1.5 cm in diameter and are located in subcortical regions of the basal ganglia, internal capsule, pons, and cerebellum (69). The area of the lacune (meaning “little lake”) roughly corresponds to the vascular territory supplied by one of the deep perforating branches from the circle of Willis or major cerebral arteries. Lacunar strokes are strongly associated with hypertension, and pathologically associated with microvascular changes that often develop in the presence of chronic hypertension. Histological changes such as arteriolar thickening and evenly distributed deposition of eosinophilic material, called lipohyalinosis and fibrinoid necrosis, are commonly seen in the subcortical perforating arteries of hypertensive persons who have had lacunar strokes. Microatheromas within deep perforating arteries are also important causes of lacunar infarction. In addition to hypertension, diabetes mellitus is associated with lacunar stroke as a result of chronic microvascular changes.

2.1.2.2 Hemorrhagic Stroke

Intracerebral Hemorrhage

The deep perforating cerebral arteries are also the site of rupture preceding intracerebral hemorrhage (ICH). However, unlike lacunar strokes, ICH does not obey the anatomical distribution of a vessel but dissects through tissue planes. Such damage can be significant, resulting in increased intracranial pressure, disruption of multiple neural tracts, ventricular compression, and cerebral herniation.

Acute mortality is high, but those who survive ICH often experience rapid neurological recovery during the first 2 or 3 months after the hemorrhage.

Nearly one-half of all ICHs occur within the putamen and the cerebral white matter (70). Sudden hemorrhage into brain parenchyma is related to both acute elevations in blood pressure and chronic hypertension. Microvascular changes associated with hypertensive hemorrhage include lipohyalinosis and Charcot-Bouchard aneurysm (71). The latter are not true aneurysms of the vessel wall but are pockets of extravasated blood or "pseudoaneurysms," a sign of previous microscopic ruptures within the vascular wall. Typically, the bleeding lasts no more than 1 or 2 hours, corresponding to the usual time course of acute symptom development. Late neurological decline is related to posthemorrhagic edema or re-bleeding.

Cerebral amyloid angiopathy is unusual but is gaining recognition as an important cause of ICH in the elderly (72). Superficially located hemorrhage that occur in patients older than 55 years who have some premorbid history of mild dementia are characteristic of this disease, but in the absence of tissue staining for Congo red amyloid deposits within the adventitia of cerebral vessels, diagnostic uncertainly remains.

Other notable causes of ICH include the use of anticoagulants, intracranial tumor, and vasculitis.

Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH), or bleeding that occurs within the dural space around the brain and fills the basal cisterns, is most commonly caused by rupture of a saccular aneurysm or an arteriovenous malformation (AVM). Saccular aneurysms develop from a congenital defect in an arterial wall followed by progressive degeneration of the advantitia, which causes ballooning or outpouching of the vessel. The risk of bleeding from unruptured aneurysms is speculative but appears greatest for aneurysms greater than 10 mm in diameter (73).

Saccular aneurysms often rupture during the fifth or sixth decade of life. When a rupture occurs, the extravasation of blood into the subarachnoid space is irritating to the dura and results in a severe headache often described as the "worst in my life." Because of a sudden drop in cerebral perfusion pressure, acute loss of consciousness is frequent. Focal neurological changes or coma can ensure. As many as one-third of patients with aneurysmal hemorrhage die immediately. Patients who present with coma, stupor, or severe hemiplegia have the worst prognosis for proximate survival. The risk of rebleeding from an unoperated aneurysm is as high as 30% within the first month after hemorrhage and declines thereafter. The risks for long-term re-bleeding remain 3% per year (74).

Saccular aneurysms are most often found in the anterior region of the circle of Willis, particularly near branches of the anterior communicating, internal carotid, and middle cerebral arteries, but they can also be found at the junction of almost any branch site within the cerebral circulation. Early surgical management using neurosurgical clipping technique is as safe as late surgery, and it significantly reduces risk of re-bleeding.

Arteriovenous malformations present with hemorrhage earlier in life than do aneurysms, often in the second or third decade. Although they cause nearly 9% of all SAHs, vascular malformations are also important causes of ICH and intraventricular hemorrhage (75). An AVM is a congenital structure consisting of a tangled web of vascular tissue that contains multiple arteriovenous fistulas, which permit arterial-to-venous shunting of blood. They can be located anywhere in the CNS and may grow quite large, displacing normal neural structures, usually without disruption of function. Seizure, migraine, or hemorrhages are typical presenting symptoms.

The incidence of lifetime hemorrhage with AVM is 40% to 50% (76) with a re-bleeding rate of 4% per year and a mortality rate of 1% per year (77). Treatment options include neuroradiological embolization, surgical resection, and radiotherapy (gamma knife)

Hydrocephalus

Acute and chronic hydrocephalus can complicate both SAH and intraventricular hemorrhage by obstructing cerebrospinal fluid (CSF) outflow. Blood coagulum within the ventricular system can block the foramen of Sylvius or the fourth ventricle, causing acute obstructive hydrocephalus over minutes to hours after hemorrhage, leading to lethargy, coma, or death if not treated. Placement of an external ventricular drain can be life-saving, but if the obstruction does not resolve, a ventriculoperitoneal shunt is placed for long-term decompression.

Normal pressure hydrocephalus is very common after SAH and often develops during rehabilitation care. The pathophysiological cause is a functional disruption of CSF resorption due to fibrosis of the arachnoid granulations from subarachnoid blood (78). The classical symptoms of subcortical dementia, incontinence, and gait disorder are clues to the presence of hydrocephalus, but the physiatrist should also have a high level of suspicion when a patient with recent hemorrhage is not making expected functional gains is a rehabilitation program. That suspicion is often confirmed when the patient makes a remarkable recovery after shunting.

2.2 The gait cycle

2.2.1 Heel contact

General

Heel contact (Figure2.1) is the beginning of the stance phase. It is frequently call ‘heel strike’, since there is often a distinct impact between the heel and the ground, known as the ‘heel strike transient’. In pathological gait, where some other part of the foot may contact the ground first, the term ‘initial contact’ is to be preferred. There is considerable variation between individuals as to how much force is applied to the ground at heel contact, some people gliding the foot on the ground, and others digging it in. Figure2.2 shows a trace of the vertical component of the ground reaction force measured from an individual with marked heel strike (79). There have been suggestions that the transient forces in the joints resulting from the heel strike

may lead to degenerative arthritis. The heel strike transient is fairly short, typically lasting between 10 and 20 ms, and it can only be observed using measuring equipment with a fast enough response time. The direction of the ground reaction force changes from upwards and forwards during the heel strike transient (Figure2.1), to upwards and backwards immediately afterwards (Figure2.3).

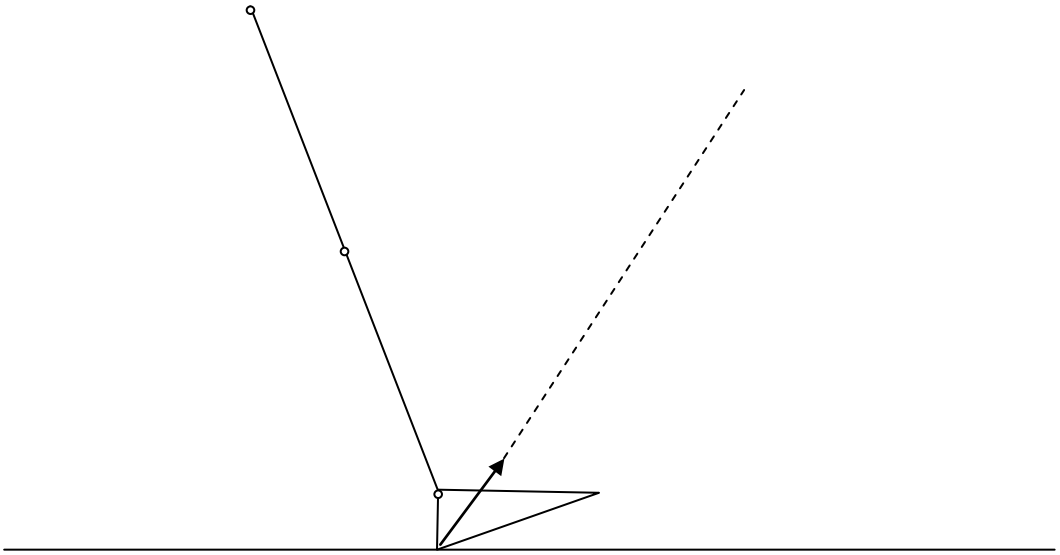


Figure 2.1 position of limb and ground reaction force vector at heel contact (during heel strike transient).

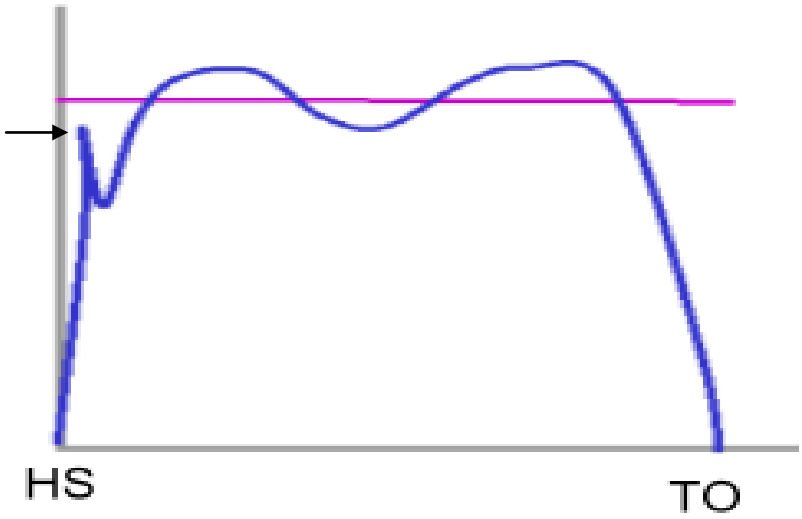


Figure 2.2 vertical ground reaction force, show the heel strike transient (arrowed)

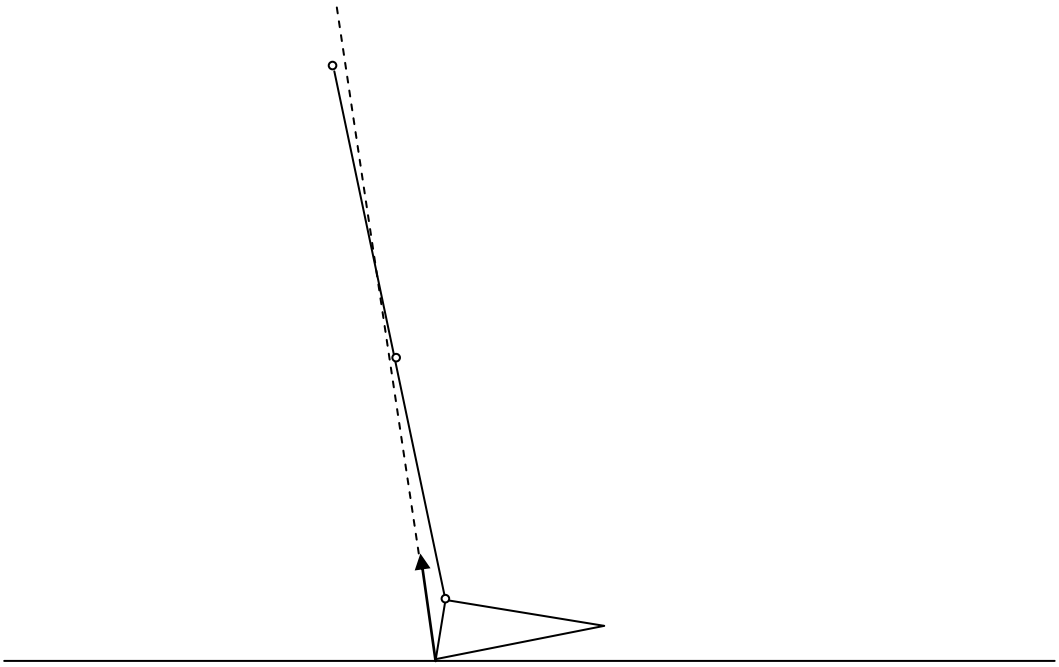


Figure 2.3 position of limb and ground reaction force vector after heel contact.

Upper body

The trunk is about half a strike length behind the level of the foot at the time of heel contact. It is at its lowest vertical position, about 25 mm below its average level for the whole cycle, and its instantaneous forward velocity is at its highest. In the side to side direction, the trunk is crossing the midline of its range of travel, moving towards the side of the leg which has just made contact. The trunk is twisted, the left shoulder and the right side of the pelvis being at their furthest forwards. The left arm is at its most advanced when the right foot undergoes heel contact. The amount of arm swinging varies greatly from one person to another, and it also increases with the speed of walking. At the time of heel contact, Murray et al (80) found that the mean elbow flexion was 8 degrees and the mean shoulder flexion 45 degrees.

Hip

The attitude of the legs at the time of heel contact is shown in Figure 2.1. the maximum flexion of the hip (generally around 30 degrees) is reached around the middle of the swing phase, after which it extends again only slightly before heel contact. The hip then extends more rapidly, through contraction of the hip extensors,

the hamstrings being active during the latter part of the swing phase, and gluteus maximus starting to contract around heel contact.

Knee

During the early swing phase, the knee flexes to 60 or 70 degrees. It then extends again, becoming more or less straight just before heel contact. Except in very slow walking, the knee flexors contract eccentrically at the end of the extension, to act as a breaking mechanism to prevent hyperextension. The hamstrings usually provide this function, in addition to their role in initiating extension of the hip.

Ankle and foot

The ankle is close to its neutral position during the latter part of the swing phase. If the foot is moving backwards as the heel contacts the ground, the ground reaction to the heel strike transient is directed forwards. The direction of the force vector changes to that shown in Figure 2.3 within 10 – 20 ms, and an external plantarflexing moment is produced. The foot would come down much too quickly, in a foot-slap, if this moment were not resisted by the anterior tibial muscles, which contract eccentrically to lower the foot gently to the ground. The heel is usually slightly inverted and the foot slightly supinated at the time of heel contact, and most people show a wear pattern on the lateral side of the heel of the shoe.

2.2.2 Foot flat

General

After heel contact, the rest of the foot comes down onto the ground at foot flat (Figure 2.4), which generally occurs at around 8% of gait cycle, just before toe off on the other side. During the interval between heel contact and foot flat, the ground reaction force increases rapidly in magnitude, its direction being upwards and backwards. This is generally known s the weight acceptance phase of gait, although the term load acceptance is to be preferred, since inertia is involved as well as weight. Although the ground reaction force has a backward directed component, this maybe cancelled out by the forward directed component of the ground reaction force from the other foot, while it is still on the ground.

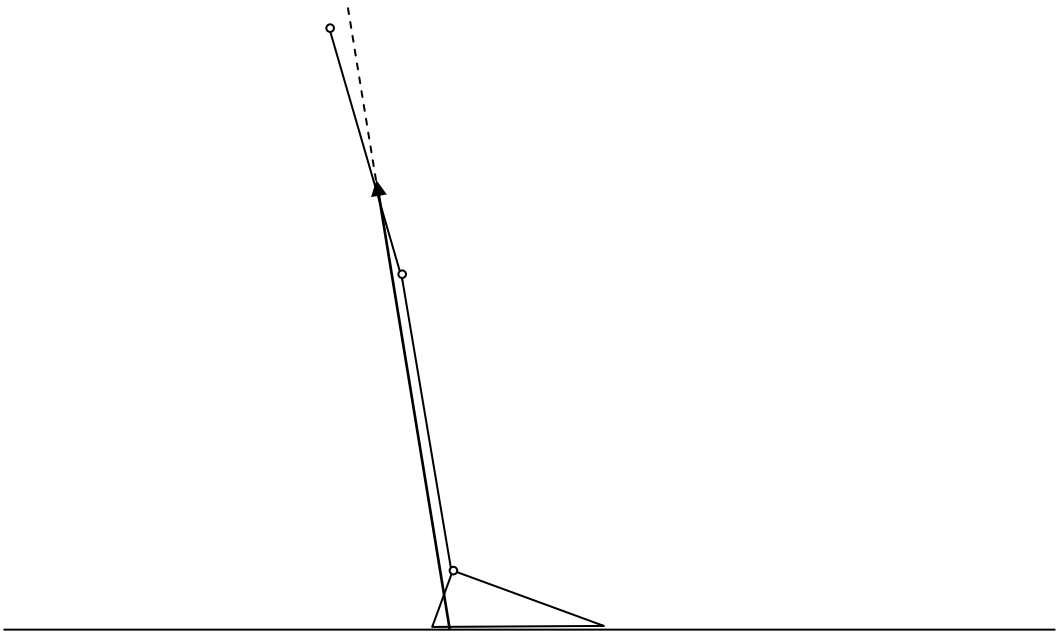


Figure 2.4 position of limb and ground reaction force vector at foot flat.

Upper body

The shoulder and arms, having reached their most advanced position on the contralateral side, are now moving back again. Similarly, the pelvis on the side of the stance phase leg now starts to twist back towards the neutral position. The trunk reaches its lowest position between heel contact and foot flat, and starts to lift upwards, slowing its forward motion slightly as it does so.

Hip

The hip is 20 to 25 degrees flexed at the time of foot flat, and continuing to extend, by contraction of the gluteus maximus and the hamstrings. This extension is opposed by an external flexion moment. In Figure 2.3 and 2.4, the projected ground reaction force vector can be seen to pass in front of the hip.

Knee

The knee flexes following heel contact, and in doing so it acts as a spring, preventing the vertical force from building up too rapidly (81). The knee moment can be estimated from the projected force vector in Figure 2.4, rather more reliability than

it can for the hip, since only the shank and foot are between the knee joint and the ground. The vector passes behind the knee, producing an external moment. This is opposed by an internal extension moment generated by the quadriceps muscles, which contract eccentrically to permit a small amount of controlled flexion. Knee flexion continues beyond foot flat, reaching the peak of stance phase flexion at 15 to 20% of the gait cycle. The magnitude of the stance phase flexion is variable, but is usually between 10 and 20 degrees. The external flexion moment also reaches a peak at this time.

Ankle and foot

The stage of the gait cycle from heel contact to foot flat, involves a plantarflexion at the ankle of around 15 degrees, which is achieved by an external plantarflexion moment, opposed by the anterior tibial muscles contracting eccentrically. As soon as the foot is flat on the ground, the line of the ground reaction force begin to move forwards along the foot, causing the moment to become smaller and then to reverse. The movement into plantarflexion is accompanied by pronation, because the tibial is internally rotated at the beginning of the stance phase (82).

2.2.3 Mid stance

General

The term mid stance may be used either to describe the period of time between foot flat and heel off, or to define a particular event occurring during that period of time. Mid stance (Figure 2.5) is close to the midpoint of the stance phase, and is about 30% of the way through the whole gait cycle.

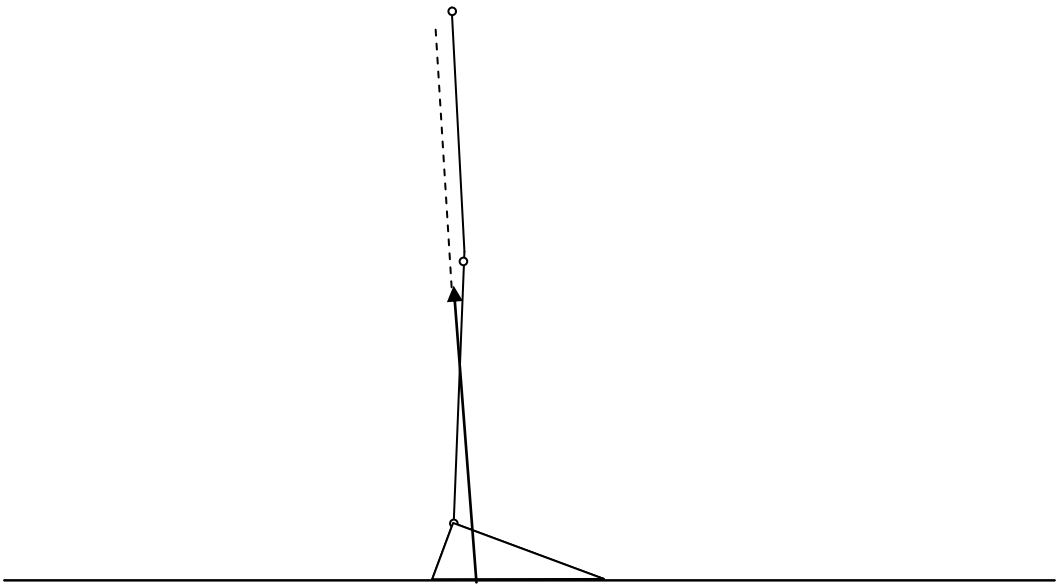


Figure 2.5 Position of limb and ground reaction force vector at mid stance.

Upper body

The period from foot flat to mid stance sees the trunk climbing to its highest point, about 25 mm above the mean level, and slowing the forwards velocity, as the kinetic energy of forward motion is converted to the potential energy of height. The side-to-side motion of the trunk also reaches its peak at mid stance, the trunk being displaced about 25 mm from its central position, towards the side of the stance leg. Like the feet, the arms are side by side at mid stance, as each follows the motion of the opposite leg. The twisting of the trunk has now disappeared, as both the shoulder girdle and pelvis pass through neutral before twisting the other way.

Hip

At mid stance, the hip is more than half way through its movement into extension, with a typical angle close to zero. The external flexion moment, and the opposing contraction of the extensor muscles, declines and disappears during te middle of the stance phase. It is replaced by a moment in the opposite direction, an external extension moment, which is opposed by an internal moment generated by the ip flexors, psoas major and iliacus, contracting eccentrically. In the coronal plane, as soon as the opposite foot has left the ground, the pelvis is supported only by the stance

phase hip. Although dipping down slightly on the side of the swinging leg, its position is maintained by contraction of the hip abductors, especially gluteus medius.

Knee

By mid stance, the knee has started to extend again. The external flexion moment reduced the magnitude, but is still present, since the ground reaction force is still behind the knee. As the quadriceps muscles have finished contracting by this time, there is, at the first glance, nothing to generate an internal moment to resist flexion at the knee. However, such a moment is generated by the dual effects of the contraction of the soleus and the forward motion of the upper body. If the ankle joint were totally free, the forward motion would simply dorsiflex the ankle, but contraction of the soleus slow down the forward motion of the tibial, and as the femur continues to move forwards, the knee extends.

Ankle and foot

The period from foot flat to heel off is characterized by forward rotation of the tibia about the ankle joint, from about 15 degrees of plantarflexion to about 10 degrees of dorsiflexion. At mid stance the ankle is generally between the neutral position and 5 degrees of dorsiflexion. The ground reaction force moves forward along the foot from the time of foot flat onwards, the initial external plantarflexion moment being replaced by an external dorsiflexion moment of increasing magnitude. As mention above, this is resisted by contraction of the soleus, which prevents the tibia from moving forward too quickly. As the stance phase progresses, the tibia rotates externally, and the subtalar joint causes the foot to move out of pronation.

2.2.4 Heel off

General

Heel off (Figure 2.6) is the time at which the heel begins to lift from the walking surface. Its timing varies considerable, both from one individual to another and with the speed of walking. The descriptions which follow are based on a typical heel off at 40% of the gait cycle. Heel off normally occurs before heel contact by the other leg, which occurs at around 50%.

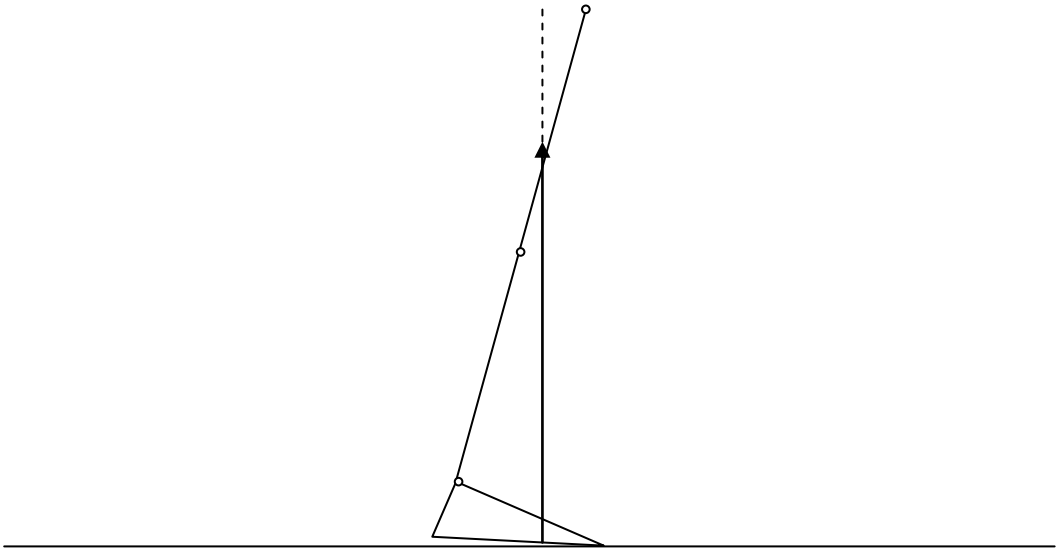


Figure 2.6 Position of the limb and ground reaction force vector at heel off.

Upper body

Once mid stance has passed, the trunk begins to lose vertical height, on its way down to its lowest point in the double support phase. The lateral displacement over the support leg also begins to reduce, in preparation for transfer of weight back to the other leg. As hip extends and stance leg moves backwards, the pelvis twisted backwards with it, and the arms and shoulder girdle on that side move forwards.

Hip

The hip continues to extend, reaching an angle of between 10 and 15 degrees of extension by the time of heel off. The external extension moment continues to act, and the hip flexors, mainly psoas and iliacus, contract eccentrically to resist this. Between heel off and toe off, the hip reaches the peak of extension and starts to flex again. The activity of the hip abductors in the coronal plane is still required, until heel contact by the other foot.

Knee

The knee has an extension peak close to the time of heel off, the angle at this time being between zero and a few degrees of flexion. Between mid stance and heel off, the ground reaction force vector moves in front of the knee joint, producing

an external extension (or hyperextension) moment. The role of the soleus in opposing the external flexion moment around mid stance was described previously. Once the direction of the external moment changes, so that it attempts to extend the knee, the gastrocnemius comes into play. This muscle augments the action of the soleus as far as the ankle joint is concerned, but it also acts as a flexor at the knee, and so prevents hyperextension.

Ankle and foot

The peak of ankle dorsiflexion is reached around the time of heel off. The ankle angle at this time is usually between 15 and 20 degrees. As soon as the heel leaves the ground, the knee begins to flex and the ankle to plantarflex. As the force vector moves forwards along the foot, the external dorsiflexion moment increases, and first the soleus and then the soleus and gastrocnemius together contract to oppose it, the peak activity of these muscles coinciding with the peak external moment, just after heel off. As the heel rises it also inverts, and the foot supinates.

2.2.5 Toe off

General

Toe off (Figure 2.7) occurs about 60% of the gait cycle. It is the point at which the stance phase ends and the swing phase begins. It also marks the end of the second double support phase of the cycle, since the other foot makes heel contact at about 50% of the cycle.

Upper body

The position of the body is roughly a mirror image of that described around foot flat, which is taking place on the other side. The extremes of arm and shoulder advancement, and the second low point for the trunk, all occurs following heel off, and are beginning to reverse again by toe off.

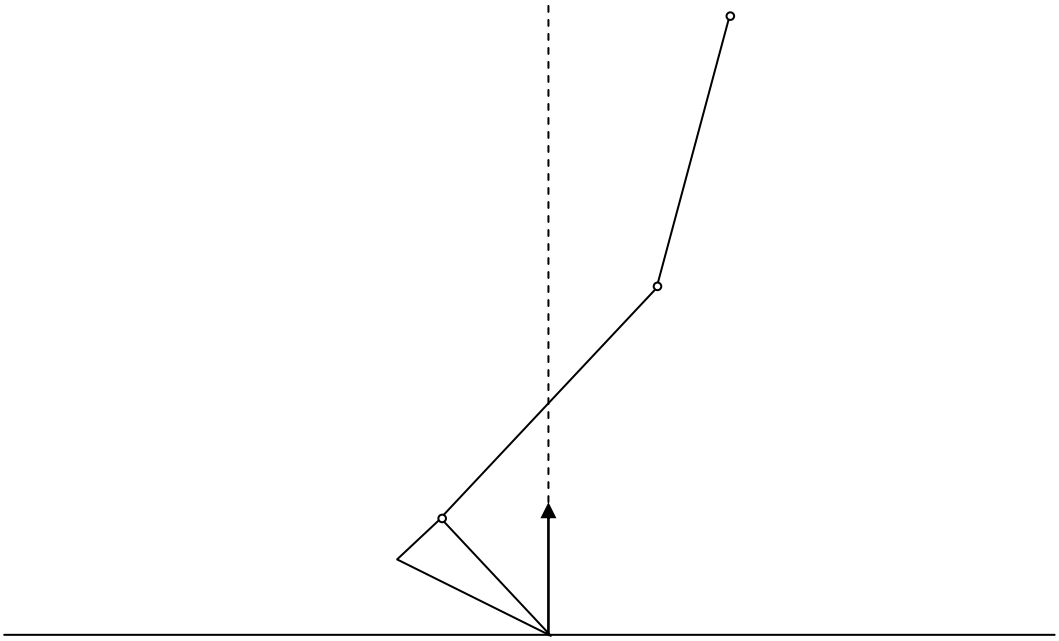


Figure 2.7 Position of limb and ground reaction force vector at toe off.

Hip

The peak extension of the hip, which is between 10 and 20 degrees, is reached before toe off, by which time the hip is flexing again. Illiopsoas activity ceases before toe off, and the moment which flexes the hip is provided partly by gravity, partly by rectus femoris, in its dual role as hip flexor and knee extensor, and partly by the hip adductors, which also act as flexors when the hip is extended.

Knee

The knee starts to flex even before heel off, and by the time of toe off it is flexed to an angle of 40 to 50 degrees. Before the foot leaves the ground, the force vector moves from in front of the knee to behind it, and in the final part of the stance phase the knee is subjected to an external flexion moment. The knee is permitted to flex, but the rate is controlled by eccentric contraction of the rectus femoris, which flexes the hip at the same time that it controls knee flexion. The other elements of the quadriceps may also assist in controlling knee flexion. Knee flexion continues even after the foot has left the ground, partly as a result of the continued forward movement of the femur. During the swing phase the leg acts as a double pendulum, and knee flexion followed by extension takes place purely passively.

Ankle and foot

In the period between heel off and toe off, the ankle moves from dorsiflexion into plantarflexion. The total range of this movement varies between about 20 and 35 degrees. Extension take place at the metatarsophalangeal joints, as the heel and hindfoot lift up while the phalanges of the toe stay on the ground. The major force transmission to the ground is through the metatarsal heads. During this period there is a substantial external dorsiflexion moment about the ankle, which is resisted by powerful contraction of the soleus and gastrocnemius. The force transmitted up the leg flexes the knee and helps to initiate flexion of the hip. While load is being transmitted through the forefoot, the peronei, tibialis posterior and the long toe flexors contract to stabilize the foot and to permit the toe, as well as the metatarsal heads, to transmit force to the ground. As toe off approaches, the ground reaction force diminished rapidly, and it disappears as the foot leaves the ground and the leg enters the swing phase. The foot is supinated at toe off, but this is largely lost during the first part of the swing phase.

2.2.6 Mid swing

General

Mid swing on one side (Figure 2.8) corresponds to mid stance on the other: it is the time when the swinging leg passes the stance phase leg, and the two feet are side by side. The swing phase occupies about 40% of the gait cycle, and mid swing generally occurs close to the midpoint of this period. The swing phase is divided into two parts: an acceleration phase, before mid swing, and a deceleration phase following it. Alternative names are initial swing and terminal swing. The walking velocity depends to a large extent on the efficiency of the swing phase, since the stride length is the amount by which the foot can be moved forward during this time. If the foot catches on the ground, this will slow or even terminate the swing, and thereby reduce the stride length.

Upper body

At mid swing, as at mid stance, the trunk is at its highest position, and is maximally displaced over the stance phase leg. The arms are level with each other, one moving forward and one moving back.

Hip

The hip starts to flex even prior to toe off, and by the time of mid swing it has almost reached its most flexed position. Iliopsoas is active around heel off. It stops contracting briefly until toe off, and then contracts powerfully to flex the hip until mid swing. Hip flexion is aided by gravity, rectus femoris and the adductors. After mid swing, in the deceleration phase, the hamstrings contract, to slow down, stop and then reverse the flexion of the hip.

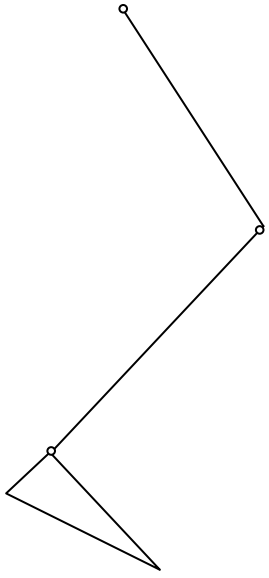


Figure 2.8 Position of limb at mid swing.

Knee

The flexion of the knee during the swing phase results largely from the flexion of the hip. The leg acts as a jointed pendulum, and no muscular contraction around the knee needs to be involved. The peak of swing phase flexion is usually between 60 and 70 degrees, and occurs before mid swing, by which time the knee has started to extend again. Muscular activity is required at the end of the swing phase, when the knee flexors, in particular the hamstrings, contract to prevent hyperextension

of the knee, which would result from an uncontrolled continuation of the pendulum-like movement.

Ankle and foot

In normal walking, the toes clear the ground by very little. Murrey et al (80) found a mean clearance of 14 mm with a range of 1 to 38 mm. most of the shortening of the leg required to achieve this comes from flexion of the knee, but the ankle is also needs to move from its position of plantarflexion, at the end of the stance phase, to an approximately neutral position, to help with clearance. This movement requires contraction of the anterior tibial muscles, although the force of contraction is much less than that required to control foot lowering following heel contact. The foot is supinated at toe off, and remains so until the following heel contact.

2.3 Hemiparetic Gait

The pathophysiological basis of stroke is damage to motor cells and pathways of the central nervous system caused by hemorrhage or thrombus affecting the arterial supply of the brain, usually of one side, typically occurring with little or no warning. The two immediate impairments of most significance to gait performance are diminished strength, or the inability to generate voluntary muscle contractions of normal magnitude in any muscle groups, and inappropriately time or inappropriately graded muscle activity. After a few weeks, there may also be two further impairments: spasticity and changes in the mechanical properties of the muscle causing abnormal extensibility of muscle groups (83).

The individual’s gait depends upon the severity of the cerebral involvement and the particular adjustments utilized. Two fundamental motor problems beset most hemiparetic; interference by gross limb synergies and the slow increase and decrease of muscular tension. Ironically, the phasic action of muscles on the non-paretic side resembles the paretic side. Perceptual difficulties add to the abnormally of ambulation.

The primitive patterns that appear in hemiparetic gait are; the flexion synergy includes hip flexion, abduction, and external rotation, knee flexion, and dorsiflexion, and inversion of the ankle and foot. The extension synergy combines extension, adduction, and internal rotation of the hip, extension of the knee, and plantarflexion and inversion at the ankle and foot.

The foot often exhibits inadequate dorsiflexion control, insufficient push off, and excessive lateral contact. Weight bearing activates the extension synergy; calf tension prolongs plantarflexion. Thus, patient strikes on heel and forefoot simultaneously or, if spasticity is severe, patient contacts only with the ball. When inversion is marked, patient stands initially on the lateral border of the foot.

Later in stance, patient cannot regulate the extensor tension initiated by weight bearing. Patient does not maintain an effective foot lever for push off, nor does patient flex hip and knee forcefully. Throughout stance, triceps surae activity is low, neither decreasing nor building up quickly in the normal manner.

During swing phase, inadequate dorsiflexion control is still apparent. The dorsiflexors fail to contract, so the foot slaps the floor and simultaneously inverts, sometime it everts as it is drag. Others patients display exaggerated uncontrolled dorsiflexion as part of the flexion synergy. These patients may intentionally flex the hip abruptly to begin the climbing (flexion) reflex which terminates in dorsiflexion.

Usually the hemiparetic extends there knee rigidly throughout stance phase, another manifestation of the gross extensor pattern. The normal flexion-extension-flexion sequence to absorb shock and to smooth progression is absent. Quadriceps tension should decrease rapidly as the limb transfers from swing to stance instead; the knee extensors are active from late in swing until toe off. The moment of force at the knee is strongly positive, indicating extension throughout the cycle. The hemiparetic patient strikes with the knee fully extended, with little or no subsequent flexion. Some patients ultimately flex the knee, but the onset of flexion is delayed because the calf muscles do not reflex. The spastic calf muscles prevent the leg from

rotating forward at the ankle. Later in stance phase, the abnormal combination of triceps surae and quadriceps interferes with initiation of the swing. The patient begins swing phase with his knee straight. Knee flexion may occur eventually, as part of the primitive flexion synergy, facilitated by hip flexion.

In hemiparetic patient, the knee may flex excessively, collapsing under the load of the body weight. Circumduction evidences the extension synergy which prevents the limb from shortening. The hip and knee fail to flex, or flex insufficiently, and the ankle remains plantarflexed. Hemiparetic patients thrust trunk backward, toward the non-paretic side, to raise pelvis on the paretic side and thereby swing the leg. If the knee straightened actively or reflexly, supination can be prevent, otherwise lateral foot contact would occur with knee flexion as part of the flexion synergy.

Posterior trunk bending helps to advance and circumduct patient paretic limb. Occasionally, patient completes the swing of paretic leg by anterior trunk bending to drag the paretic limb forward. Anterior trunk bending persists into stance phase, to insure that the paretic knee will be stabilized by the forward position of the body's center of gravity and to minimize the paretic limb's opposition to propulsion of the normal leg. Trunk flexion early in stance on the paretic side allows the patient to lose their balance forward without overcoming the hypertonicity of the knee.

Lateral trunk bending may accompany the abnormal anterior-posterior movements during swing. Leaning toward the non-paretic side initiates swing of the paretic limb. The non-paretic gluteus medius increases its activity. The paretic leg advances with the aid of lateral tilting of the trunk, so that the abdominals, quadratus lumborum, and gluteus medius help the limb to clear the walking surface. The abductors belong to the flexion pattern and do not respond when the patient bear weight on his paretic leg. The pelvis then lowers toward the non-paretic side. The hemiparetic tends to lean toward their paretic side although.

Hip hiking assists the swing of the paretic limb, when patient cannot activate their hip flexors. The patient may rotate the paretic limb externally during

swing, so the hip adductors are more nearly in the sagittal plane. The paretic foot is directed sideways. Rotation releases the patient from the need to overcome the tension in the spastic calf and gives better anterior-posterior balance.

Rhythmic abnormalities are perhaps the most conspicuous feature of the hemiparetic gait. Stance on paretic side is considered shorter, step lengths are unequal, and patient cannot advance the normal foot in front the paretic foot. Another deviation common among hemiparetic was the flexed and adducted posture of the arm. Its inability to swing reciprocally compounds the ambulatory disability.

Decreased strength refers to several measurable factors, and may be expressed as decreased ability to generate a moment of force about a joint, a deficiency in muscle power (27), or, when taken over time, as diminished work accomplished by the muscles, the electromyography (EMG) of the paretic side is generally lower than that of the non-paretic side, and this factor is compounded by an unusually low force to EMG ratio (84), which results in overall decreased force or moment output. There may be several causes of the loss of strength (85) including: decreased capacity to activate the motor units, a reduced number of functioning motor units (86), and reduced motor unit firing rates (84). Other factors that could explain the apparent decrease in strength involve the performance of the antagonist muscle groups. Although a study of the interrelationships between static measures of strength and measures of spasticity have not supported a major role for spasticity of an opposing muscle in limiting voluntary activation (87), the role of spasticity during dynamic activities has not been adequately assessed. Another factor that can explain the apparent decrease in strength of a muscle group is increased stiffness of the opposing group. In one study, structural changes in spastic ankle plantarflexor muscles accounted for the appearance of weak dorsiflexors, as excessive activation of the dorsiflexors was required to overcome the excessive passive resistance of the plantarflexors (83). Active restraint by opposing muscles may also be implicated, and inappropriate co-contractions have been reported in the gait of subjects with hemiparesis (8,88). Winter et al (89) showed that the gait pattern of hemiparetic patients could be divided into four groups.

Group 1 subjects essentially suffer from a single problem – a foot drop on the paretic side. This causes initial contact to be by primary toestrike, and also produces a functional increase in leg length during the swing phases. The plantarflexed attitude of the foot at initial contact leads to an increased flexion of both the knee and hip. The lumbar lordosis is also increased throughout the gait cycle. The most common surgical treatment for this problem is to lengthen the Achilles tendon. However, this is unlikely to improve the gait, since the ankle is already able to dorsiflex adequately. The significant problem in these individuals is the foot drop during the swing phase, which is caused by a relative weakness of the anterior tibial muscles, and which can be treated by adequately by a simple orthosis.

Group 2 subjects, as well as having a foot drop, having a static or dynamic contracture of the calf muscles, which holds the ankle in plantarflexion throughout the whole of the gait cycle. The difference in gait between group 1 and group 2 is seen after mid stance, when the persistent plantarflexion produces an external moment which forces the knee into hyperextension. Advancement of the trunk is curtailed, and the length of the opposite step is decreased. As with the group 1 subjects, there is increased hip flexion with an associated increased lumbar lordosis. These patients generally benefit from an operation to lengthen the Achilles tendon, as well as an orthosis to control the foot drop.

Group 3 subjects have a foot drop, contracted calf muscles, and overactivity of both quadriceps and hamstrings. This causes a reduction in the total range of motion at the knee, with a marked reduction in swing phase flexion. The gait is described as stiff with short steps. The other features of the group 2 pattern are also present: hyperextension of the knee in late stance, hip flexion and increased lumbar lordosis. Surgical correction of these patients will extend as high as the knee. Waters et al (90) showed that the stiff-leg gait is caused by inappropriate contraction of one or more heads of the quadriceps at the end of the stance phase and the beginning of the swing phase. They described the surgical treatment of the condition by quadriceps tenotomy.

Group 4 subjects. In addition to the characteristics of the group 3 subjects, they also have a reduced range of hip motion, due to overactivity of iliopsoas and the adductors. The hip is unable to extend fully, so anterior pelvic tilt and increased lumbar lordosis at the end of the stance phase are used to preserve the stride length. Full treatment of these patients will include surgery to the muscle acting about the hip, knee, and ankle joints.

2.3.1 Spatio-temporal characteristics

The average walking speed reported for subjects with stroke is lower than values for normal subjects, with the values reflecting the severity of the stroke, the time since its occurrence and the age of the subjects. The averaged speed ranges from 0.23(0.11) m/s to 0.73(0.38) m/s (91,92). Consistent with speed decreases, both stride length and cadence are lower than values for normal subjects. Nakamura et al (93) reported the relationship between cadence and speed to be linear up to a speed of about 0.33 m/s and a cadence of about 90 steps/min, with further gains primarily attributable to increases in stride length.

Three differences in the proportions of stance and swing have been reported. First, the stance phase of both the paretic and non-paretic sides is no longer in duration and occupies a greater proportion of the full gait cycle in hemiparetic than in the normal walking at normal speeds. Second, the stance phase is both longer and occupies a greater proportion of the gait cycle on the non-paretic side than on the paretic side. However, if hemiparetic data are compared to that of the normal walking at similar speeds, the proportions of stance on the non-paretic side varies little from the normal (94), and significantly shorter on the paretic side. The third difference is that a greater proportion of the gait cycle is spent in double support than that of normal walking at normal speeds. However, when compared with normal subjects walking at comparable speeds, the total double support of hemiparetic subjects is significantly lower (49% compared to 53%) (94) than normal. The total double support can be viewed in two parts. The portion of double support occurring during late stance of the paretic foot (when the non-paretic foot is spatially ahead of the paretic limb) has been reported to be about 5% greater than the alternate, which at first seems counterintuitive

as the stance phase is significantly longer on the non-paretic side. However, foot contact of the non-paretic limb occurs, on average, at 45% of the gait cycle of the paretic limb, whereas the paretic limb does not make contact until about 55% of the cycle of the non-paretic side.

2.3.2 Kinematic characteristics

The kinematic pattern of the paretic side of hemiparetic patients during walking has been described. The common gait patterns in hemiparetic are decreased hip flexion, knee flexion, ankle plantarflexion, and circumduction during swing. Whereas there are knee hyperextension and lacked of roll-off at toe off during midstance.

Burdett et al in 1988 (91) assessed kinematic characteristics of 19 hemiparetic subjects during walking with and without orthoses. Their subjects' natural speed was low. The authors concluded that subjects' major kinematic differences from the noamal were: [1] decreased hip flexion at initial contact, increased hip flexion at toe off, and decreased hip flexion during midswing; [2] more knee flexion at initial contact and less knee flexion at toe off and midswing; [3] more ankle plantarflexion at initial contact and midswing and less ankle plantarflexion at toe off.

De Quervain et al in 1996 (28) reported hemiparetic gait patterns. There was a delay in the initiation of flexion of the hip during the preswing phase. During stance phase there was decreased extension of the hip. This information was found in hemiparetic patients when evaluated within one week after walking.

Several investigators (95-97) reported that the trajectory of hip flexion and extension patterns were not smooth. The lack of hip extension during terminal stance may result in a shorter stride length and decreased gait velocity of hemiparetic patients. However, hemiparetic patients exhibited a variety of patterns, which were caused by a different pathology, abnormal muscle tone, poor motor control and compensating movement.

2.3.3 Electromyographic characteristics

One of the earliest reports of EMG in stroke was by Hirschberg and Nathanson (98) in 1952, using skin electrodes over gluteus medius, adductor longus, semitendinosus, vastus lateralis, medial gastrocnemius, and tibialis anterior. They reported the EMG levels to be decreased on the paretic side in all 11 subjects, with the patterns of activation of the hip muscles showing only small deviations from normal. The quadriceps and hamstrings showed the highest levels of activity which began at the usual time but were prolonged to the end of stance phase.

Peat and colleagues (99) provided the following summary of EMG activity. The average EMGs across subjects showed that the level of all muscle groups increased simultaneously as initial contact was made, and peaked together in mid stance phase. The average profiles of tibialis anterior, quadriceps and calf muscles showed a relatively sharp peak of activity in mid stance, but the period of high level activity of the medial hamstring extended from early to late stance. After toe off, the activity of all groups decreased as the limb prepared to leave the supporting surface. Pinzur and colleagues (100) noted consistent out-of-phase activity in rectus femoris. Hirschberg and Nathanson (98) also reported activation of gastrocnemius with initial contact but described the amplitude as low and uniform, not showing the increase that characterizes push off, while Pinzur and colleagues (100) reported consistent out-of-phase activity in both tibialis anterior and gastrocnemius-soleus.

There is unanimous agreement by researchers reporting on EMG that there is a great deal of inter-individual variation (8,99) and the descriptions given above should be regarded as itemizations of variations seen in the group of subjects rather than features generally seen in all subjects. This claim has been substantiated by Knutsson and Richards (8) who attempted to reveal inter-individual differences in disturbed motor control by classifying the subjects. This approach revealed inter-individual differences in the disturbed motor control that was not revealed when the EMG of compiled groups was examined for common features. In a group of 26 subjects with hemiparetic, all in the chronic recovery stage except one, Knutsson and Richards (8) described three types of disturbed motor control during gait. Subjects

with a Type I disorder demonstrated hyperactive stretch reflexes leading to premature activation of the calf muscles in early and mid stance as the muscles were stretched following initial contact and the lower leg began pivoting over the fixed foot for weight acceptance and transfer. Early contraction of the calf muscles precluded the accomplishment of an effective push off by the plantarflexors. The activation patterns of subjects with a Type II disorder were characterized by a lack of activation during both shortening and lengthening contractions. Characterized by low levels of muscle activity in two or more major muscle groups, sometimes activity was present only during loading. In most cases, the distal groups were more disturbed than the proximal, but otherwise the disturbances were irregular. The ability to generate sufficient muscle force to stabilize and to generate power is diminished in this group. Gait ability varied among these subjects, ranging from those who could walk only a few meters to others who could walk several kilometers, reflecting the variety and extent of deficiencies. There was no attempt to categorize further, though there appeared to be several sub-groups. Subjects with Type III disorder had neither responses to muscle stretch nor lack of muscle activation. Instead, they showed excessive and stereotyped co-activations of several muscle groups. Normal sequencing was disrupted in some cases several muscle groups became co-activated during late swing and sustained into stance, but in others, co-activation occurred only during stance phase. Shiavi and colleagues (23) classified those with activation predominantly during stance as Type III-S and during transitions as Type III-T. Type IV pattern, showing combined components of the other three, was also reported.

Shiavi and colleagues (101) applied the Knutsson and Richards classification of both paretic and non-paretic extremities to the evaluation of change in locomotors control with recovery, evaluating 12 subjects early (1 – 10 weeks) and late (6-24 months) post-stroke. In the early phase, all disturbed types were observed on the paretic side but two had normal profiles. Interestingly, however, when reevaluated later, the classification changed for all subjects with disturbed patterns. Abnormal patterns were also seen on the non-paretic side, but it was not possible to determine whether they were due to neurological insult or to biomechanical compensation.

Few authors have reported on EMG of the non-paretic limb. Wortis and colleagues (102) in 1952, reporting on the results of 19 subjects, expressed surprise that in many instances the electromyograms looked more atypical on the non-paretic side than the paretic, attributing this appearance to the fact that the amplitudes were higher in the extensor muscles while many of the abnormalities present in the paretic extremity were also present. They noted particularly that the activity of the quadriceps muscle was prolonged into the second half of the stance phase, and that, frequently, there were bursts of activity of the semitendinosus that was uncommon in the paretic leg. In addition, although the patterns of the gastrocnemius muscles were higher than those of the paretic limb, they remained abnormal, showing even contraction throughout the stance phase.

2.3.4 Kinetic characteristics

Because kinetic variables are the cause of the kinematic and spatio-temporal outcomes of the walking subject, kinetic information is particularly satisfying and useful in understanding and interpreting the characteristics of hemiparetic gait. Unfortunately, there have been very few studies offering full link segment kinetic analyses.

Moment

The earliest large study of joint moments identified was reported in 1951 by Wortis and colleagues (102). Using stroboscopic methods with force plate data, they reported the internal knee moment to be flexor throughout stance phase in 19 hemiparetic subjects. The results of Lehmann and colleagues (94) did not show a continuous internal flexor moment. However, comparisons of the average knee moments during the mid stance interval showed the mean internal extensor moment to be significantly smaller in the seven subjects with stroke than in normal subjects walking at similar speeds (5.3 N.m compared to 8.6 N.m). Further, the mean internal knee flexor moment appeared to be much larger (17.3 N.m compared to 9.4 N.m), though values did not reach a level of statistical significance. The knee flexor moment may, in fact, have been attributable to hamstrings activity, made necessary by the need for a large extensor moment at the hip to balance the forward lean noted by the

authors. Although not pertinent in these cases, an exaggerated knee flexor moment in stance may be associated with a position of full extension, or hypertension, and can be attributed to the resistance offered by the knee ligaments.

Ground reaction force

A few authors have reported on ground reaction forces. Wortis and colleagues (102) described the vertical force curve in 19 subjects. In contrast to the double peaked curve that is typical of the normal, the vertical force curve was more variable and had an initial low peak. Carlsoo and colleagues (103) reported three patterns: one with a first peak during heel contact phase and the second during push off; a second pattern showed a continuous plateau, but no discernible peaks; and a third with a single peak in mid stance. More recently, researchers have used the vertical force curve as an indicator of progress in treatment using therapeutic electrical stimulation (104,105), and have suggested that trends towards the re-institution of higher forces and a double peaked vertical force pattern be regarded as positive.

Rogers and associates have reported on the laterally-directed ground reaction forces in the frontal plane, in order to characterize the initiation of single limb stance (106). During double support the normal showed a simultaneous increase in medially-directed force on the lifting limb and decrease in medially-directed force on the stance limb in a proportion of about 4:1. In subjects with stroke, however, when the subjects transferred weight to the paretic side, 86% of the resultant laterally-directed impulse was contributed from beneath the non-paretic side. This resulted in a 6:l lifting-to-stance contribution from beneath the non-paretic side. In contrast, when subjects transferred towards the non-paretic side, 70% of the contribution was from under the stance limb, resulting in a 1:2.3 lifting-to-stance proportion. In some instances, there was a lack of synchrony and reversals in the usual directions of the lateral forces, suggesting disturbances in inter-limb coordination.

Mechanical energy

The mechanical energy of walking has been calculated using a two dimensional, seven segment mechanical energy analysis. The instantaneous energy of one segment is calculated as:

$$E = mgh = ½ mv^2 + ½ Iw^2$$

where m is the mass of the segment, assuming it to be a certain proportion of body mass; g is the gravitational constant; h is the height of its centre of mass above the reference point of the floor surface; v is the linear velocity of the centre of mass; I is the rotational moment of inertia about the centre of mass; and w is the angular velocity of the segment.

When an increase in potential energy is simultaneous with a decrease in kinetic energy, or vice versa, an energy-conserving exchange is assumed to have occurred, and is known as a within-segment exchange. There are two mechanisms of changing the mechanical energy level of a segment. The first mechanism is passive, calculated as the product of the joint reaction force and the linear velocity of the joint. The second mechanism acts via the tendo-muscular interface as a result of muscle action. Calculated as the product of the moment at the joint and the angular velocity of the segment, energy is generated to the segment if the polarities are the same (concentric contraction) and absorbed if they are different (eccentric contraction). In addition, when the segments are moving in the same direction, passive transfer occurs to the segment whose moment and angular velocity are in the same direction, and the rate of transfer is the lesser of the two power components at the joint. The total energy of the body at any instant in time is the algebraic sum of the energy of each of its segments.

The total energy cost of a single stride is the absolute sum of the differences between energy levels at each instant in time over the full cycle; that is, the total cost is the sum of the rises and falls. In the normal, the total body cost has been reported to be 1.1 J/kg.m, with energy conservation to be an average of about 70%. There are three reports of energy studies of stroke subjects (12,107,108), a total of only 15 subjects. These studies reported energy conservation from 22 to 62%. In the

largest study, the costs per unit distance ranged from 0.8 J/kg.m to 3.9 J/kg.m, though most were over the normal 1.1 J/kg.m.

Three disturbances causing these reductions in conservation were identified (12). The first disturbance was characterized by the absence of the mirroring pattern of potential and kinetic energy of the large head, arms and trunk segment, indicating that little exchange between kinetic and potential energy was occurring. In the second pattern, there was some mirroring of the energy patterns of the head, arms and trunk segment, but the magnitude of the kinetic energy was so low that little exchange could occur. The third pattern was termed the hip-hiking pattern because it was characterized by a single large rise and fall of the potential energy pattern that corresponded to the time of the swinging of the paretic leg, which was accomplished with excessive rise of the trunk. This was caused by the fact that at initial contact the potential energy of the body is at its lowest point while the kinetic energy, a reflection of the velocity, is at its highest. During the period until mid stance, the body loses speed while gaining height, exchanging kinetic energy for potential energy.

Another approach to using the centre of gravity information to assess gait was reported by Iida and Yamamuro (109). The authors compared the potential and kinetic energies during gait with the total energy (derived from the algebraic sum of potential and kinetic curve). In normal subjects, the potential energy costs were greatest, the kinetic energy costs about 15 - 20% less, and total costs at least 30% less than the potential energy costs. In contrast, in four persons with stroke, the kinetic energy costs were reduced by well over 50%, but total costs were rarely more than 20% less than potential costs. Moreover, the mean work per unit distance ranged from about 0.58 J/kg.m to 1.26 J/kg.m in contrast to 0.54 J/kg.m to 0.62 J/kg.m, the range expressed for the normal. These values are predictably lower due to the error in their technique.

Joint power

In walking, the energy level of the body returns to approximately the same level at the same point in the gait cycle for each stride, and successive bursts of

positive work and negative work occur in known patterns (89). Positive work is performed by concentric (shortening) contractions while work done against gravity or other external forces is called negative work and it is performed by eccentric (lengthening) contractions. Both forms of work require metabolic energy.

The work that is performed by a muscle group that crosses a particular joint during one stride is calculated by integration of the power curve over time. The power P (watts) of a muscle group at joint j at a given instant in time is:

$$P = Mw$$

where M is the net moment of force at joint (Nm) and w is the joint angular velocity (radians/s) (32).

The speed of walking has been shown to be related to work and power (27). The peak ankle and hip power was less for both paretic and non-paretic sides with slower speeds, but the trend for knee power was less clear in this regard. Work values for the paretic side tended to decrease with declining speed, the exception being positive knee and hip work which were about the same at slow and medium speeds. On the non-paretic side, the effects of speed were less consistent, though the positive work of the ankle decreased with declining speed. Overall, the non-paretic side performed a greater proportion of the work at all speeds, roughly in 60:40 ratio. This resulted from large discrepancies between the work of the paretic and non-paretic ankle and moderate differences at the hip. There were no differences between contributions of the knee muscles.

The relationships have been reported between speed of walking and single gait variables (9). Variables that correlated significantly with self-selected speed included the maximum hip extension angle and the maximum hip flexor moment on the paretic side, and the maximum ankle and hip powers on both sides. For the paretic side, the faster walker would show a large hip flexor moment at the end of stance phase, a large range between maximum ankle dorsiflexion and ankle plantarflexion moments, and a small range between maximum knee extensor and knee flexor moments. Based on data from the non-paretic side, a fast walker should have a short

period of weight bearing, substantial ankle plantarflexor power at push off and a strong pull off by the hip flexors. This approach explores factors that are important in attaining high levels of gait performance. Although the specific results require further empirical studies before serious application in gait training, some insight into the specific factors that may be important in attaining high level of gait performance is offered.

2.3.5 Asymmetry

The achievement of symmetry in gait has been assumed to be an important objective in the rehabilitation of persons with hemiparesis (13,110,111) and, indeed, has often been used to measure success of treatment.

In an effort to determine whether symmetry in gait was related to higher walking speeds in rehabilitated subjects, the symmetry properties of 34 gait variables in 31 hemiparetic subjects were examined (112). A variable was defined as symmetric if subjects with the highest speeds had equal values on both sides of the body. If the highest speeds were achieved when the values of the paretic side significantly exceeded the non-paretic, the variable was called asymmetric. Correlations of the differences and absolute differences between the variables from the two sides with speed were used to detect symmetric and asymmetric variables, as well as variables exhibiting other forms of symmetry. Only seven variables were detected that were not indifferent to symmetry, and only one of these showed symmetry itself. Four asymmetric variables and three variables with other symmetry properties were identified. In summary, there was only weak evidence that symmetry played any role at all in promoting speed in the study group; in fact, asymmetric variables seemed more important. At present the only clear argument for making temporal, spatial or kinematic symmetry during walking a goal of treatment in chronic stroke is an aesthetic one, and there is no defensible argument for targeting kinetic symmetry as a goal of treatment for this group. This directive is probably not appropriate for persons in the early stages of treatment when one hopes to attain a high level of performance of the paretic side as recovery occurs, without the introduction of compensations.

Also, encouragement of high levels of performance by the paretic side is important at all stages of treatment.

2.3.6 Muscle work in Hemiparetic Gait

The muscle activity in hemiparetic patients varied in synergistic pattern and compensation movements. There are differences in muscle work among subjects. Some general characteristics are consistently reported such as muscle activity in the paretic sides, prolonged muscle activity duration and uncoordinated muscles timing during walking. Perry et al (113) investigated 40 hemiparetic patients and found that the soleus muscle exhibited excessive activity in 83% of the cases, the gastrocnemius in 78%, flexor digitorum longus in 85%, flexor hallucis longus in 90%, peroneus longus in 68%, and tibial posterior in only 43% of the cases.

Olney et al in 1991 (27) studied the work and patterns of both limbs in 30 hemiparetic patients (aged 47-79 years) during self-selected speed of walking using two-dimensional cinematography and force-plate. They found that about 40% of the positive work (concentric contraction) required for walking was performed by muscle of the paretic side. Major contributors were the ankle plantarflexors and hip extensors in stance and hip flexors in swing phase.

Olney and Richards in 1996 (114) reported that the electromyographic activity (EMG) decreased on the paretic limb of hemiparetic subjects. The patterns of EMG activity of the hip muscles showed small deviation from normal. The quadriceps and hamstrings showed the highest levels of activity which began at the usual time, but were prolonged to the end of stance phase.

Clinically, spasticity or abnormal muscle tone of paretic limb demonstrated different patterns for individual patient (13,115,116). Spasticity is impairment to limit movement, specially walking. Many patients often used over effort for moving some parts of the body. So that, hemiparetic patients learned the wrong muscle work and contraction, and sequence of muscle timing for walking or movements.

2.3.7 Impairment and adaptations

If a gait variable is substantially different from a value for the normal, it is usually referred to as a gait deviation. Although this term has traditionally included only those variations that can be seen by the trained observer, namely spatio-temporal and kinematic deviations, kinetic deviations should also be included. The term deviation should not be interpreted negatively, as it is, in fact, advantageous that the neuromuscular system has many different ways of fulfilling the kinematic and kinetic requirements for forward progression, balance control and maintenance of support during walking. A gait deviation may be the direct effect of an impairment, in which case it appears below with the designation of impairment. On the other hand, the gait deviation may occur to mitigate the effects of an impairment, in which case the deviation appears below under the designation of adaptation. Certain adaptations provide compensations for impairments and are of the same type as the deficit.

Spatio-temporal deviations

-Slow speed of walking; long stance phase duration and proportions, both sides; long double support period. Impairment: diminished strength; low levels of power generation by many muscle groups. Adaptation: to impaired balance; reduced push off power to reduce unbalancing effect of piston-like drive of push-off at the ankle on the non-paretic side.

-Early foot contact by non-paretic side. Impairment: diminished strength; inability to generate sufficient hip flexor moment on paretic side to reverse the thigh extension and balance the trunk while non-paretic limb approaches initial contact, inability to generate sufficient push-off power by paretic side to produce longer duration of swing phase of non-paretic side. Adaptation: to impaired balance; fear of lateral instability, need for two-foot contact to control medial-lateral stability.

Kinematic deviation

-Limited ankle dorsiflexion at initial contact and during stance**.** Impairment: diminished strength; inadequate activation of ankle dorsiflexors, co-activation of ankle plantarflexors. Changes in mechanical properties (increased

stiffness (83)) of ankle plantarflexors, making ankle dorsiflexion activation inadequate to clear the floor in swing. Adaptation: to impaired balance; provision of stability of a flat foot landing at initial contact.

-Knee hyperextension in stance phase of paretic side Impairment: spasticity of ankle plantarflexors. Changes in mechanical properties (increased stiffness (83)) of ankle plantarflexors. Adaptation: to diminish strength, seeking of position of stability with knee in a position of extension, due to diminished moment-generating capabilities by combination of hip extensors, knee extensors, and ankle plantarflexors.

-Diminished knee flexion in swing phase of paretic side Impairment: diminished strength; diminished power generation by paretic ankle plantarflexors and/or hip flexors. Spasticity of knee extensors. Adaptation: to impaired balance; desire to keep foot close to the ground during entire swing phase in preparation for next initial contact.

-Hip hiking or circumduction in swing phase of paretic side Adaptation: same as for deviation of diminished knee flexion, swing of paretic side. Diminished strength: inadequate strength of ankle dorsiflexors, with subsequent difficulty with floor clearance, co-activation of ankle plantarflexors. Changes in mechanical properties (increased stiffness (83)) of ankle plantarflexors, making ankle dorsiflexion activation inadequate to clear the floor.

-Excessive knee flexion in stance of paretic side Impairments: diminished strength; diminished support moment generation at appropriate joint angles by combination of knee extensors, ankle plantarflexors, hip extensors, knee may flex excessively to point where muscle length effect makes it more effective.

EMG and kinetic deviations

-Co-activation of knee muscles during stance, continued activation of hamstrings in stance phase of the paretic side Impairment: inappropriately timed/graded muscle contraction; an impairment of motor control. Adaptations: to diminished strength of paretic side; attempt to generate extensor power in stance to compensate for a generally low power generation of paretic side. To diminished ankle plantarflexor strength, paretic side; necessity of providing adequate extensor support moment in compensation for diminished ankle plantarflexor moment in late stance. To postural forward lean; necessity to generate sufficient hip extensor moment to balance forward-leaning body.

-Large bursts of hip flexors activity at pull off either side of body Adaptation: to diminished strength ankle plantarflexors; attempt to provide additional power for work of walking, compensation for low ankle plantarflexor power.

-Large bursts of ankle plantarflexor activity of non-paretic side Adaptation: to diminished strength of paretic side; attempt to provide additional power for work of walking, compensation for low power generation of paretic side.

-Excessive energy costs per unit distance walked Impairment: diminished strength; insufficient to cause velocity adequate for good exchange of kinetic-potential energy. Inappropriately timed/graded muscle contraction, providing irregular bursts of energy not timed for optimal energy exchange.

2.4 Strength Training

Reduced muscle strength is a common impairment after stroke (117,118). As muscle strength is closely related to gait performance, and gait performance is related to perceived participation, after stroke (119,120), one aim of stroke rehabilitation is to increase muscle strength and thereby improve walking ability and facilitate participation in everyday activities (121,122). Muscle strength can be

increased in several ways after stroke, but the evidence is insufficient to infer that one treatment is more effective than another (123).

An efficient way to increase muscle strength in general is progressive resistance strength training, whereby loads of 70% or more of the maximum strength are used (124). Progressive resistance strength training induces higher levels of neuromuscular activation than functional exercises, and so is an effective method for improving muscle strength (125).For many years, patients with stroke were advised to avoid resistive exercise training, due to the hypothesized risk of increased muscle tone (57). The evidence that strengthening exercises increases muscle tone is weak (126,127) and stroke rehabilitation recommendations now include strength training (128,129).

The principles of progressive resistance strength training for increasing force production in muscles have remained virtually unchanged since they were described by DeLorme and Watkins (130) almost 60 years ago. These principles are [1] to perform a small number of repetitions until fatigue, [2] to allow sufficient rest between exercises for recovery, and [3] to increase the resistance as the ability to generate force increases. These principles are detailed in the guidelines of the American College of Sports Medicine (131), where it is recommended that loads corresponding to an 8 to 12 repetition maximum (RM) be lifted in 1 to 3 sets, training 2 or 3 days each week. An 8RM to 12RM load is the amount of weight that can be lifted through the available range of motion 8 to 12 times before needing a rest.

Flansbjer et al (132) reported that muscle strength increased significantly after 10 weeks of progressive resistance training with no increase in muscle tone and improvements were maintained at 5 months follow-up.

Quellette et al (133) studies the effects of high-intensity progressive resistance training on lower extremity strength, function, and disability in older, long-term stroke survivors. They found that high-intensity progressive resistance training

improves both paretic and non-paretic lower extremity strength after stroke, and results in reductions in functional limitations and disability.

Weiss et al (134) investigated the effects of progressive resistance training program on changes in muscle strength, gait, and balance in older individuals 1 year after stroke. They found that lower limb strength improved 68% on the paretic side and 48% on the non-paretic side during training. Motor performance assessed by the Motor Assessment Scale improved 9% and static and dynamic balance improved 12%.

2.5 Physiological cost index of stroke

Hemiparetic gait disturbances, with associated weakness, spasticity, and abnormal central neural patterning of muscle activation, can markedly reduce the gross motor efficiency of ambulation. Reduced cardiovascular fitness, along with the increased energy demands associated with hemiparetic gait, may contribute to low ambulatory activity levels and to poor rehabilitation outcomes. Likewise, low daily ambulatory activity may produce cardiovascular deconditioning.

Michael et al (135) reported that Ambulatory activity levels and cardiovascular fitness in patients with chronic stroke are extremely low. Mobility deficits, particularly in balance, are associated with low ambulatory activity. Platts et al (136) also reported that there is increased energy expenditure when walking with a hemiparetic gait. Platts et al suggested that, even for younger stroke patients, early rehabilitation should also consider aerobic evaluation and training, with the ultimate aim of optimizing functional independence in the community.

Oxygen cost has been used successfully to evaluate gait training methods and to assess response to treatment. For example, Potempa et al (6) showed greater improvements in maximal oxygen consumption and oxygen cost in chronic stroke survivors who participated in aerobic bicycle exercise versus controls. Macko et al (137) monitored 19 chronic stroke subjects after 6 months of submaximal aerobic treadmill training. They demonstrated a statistically significant increase in aerobic

fitness (10% increase in maximal rate of oxygen consumption) and improved oxygen consumption (16% decrease in oxygen consumption during treadmill walking at a constant speed). These studies demonstrated the usefulness of the oxygen cost measure in both the clinical and research environments.

However, the measurement of oxygen consumption requires expensive equipment and specialized knowledge in data reduction and analysis, as well as time-consuming procedures in a metabolic testing laboratory. Therefore, it is important to identify a proxy measure of oxygen cost that can be realistically performed in the clinical environment.

An alternative measure, the Physiological Cost Index (PCI), has been used as a simple, indirect measure of oxygen cost during exercise (39). The PCI is defined as follows: (heart rate during steady-state exercise minus heart rate at rest) divided by walking speed.

The PCI has the potential to serve as an index of oxygen cost for a given individual because of the relationship of both oxygen consumption and heart rate to cardiac output (40). During exercise, greater oxygen demand is created in the muscle. Increased oxygen demand induces sympathetic stimulation of the heart, which increases cardiac output (heart rate × stroke volume). Cardiac output and heart rate are linearly related (except for a plateau effect of cardiac output at maximal heart rates). Greater oxygen demand in the muscle and greater cardiac output (heart rate × stroke volume) result in higher oxygen consumption. These relationships among oxygen consumption, cardiac output, and heart rate (41) support an investigation regarding whether PCI (based on heart rate) can be useful as an index of oxygen cost in a clinical setting for stroke survivors.

Fredrickson et al (138) reported that the PCI can serve as a proxy index for the oxygen cost of gait in ambulatory patients after stroke because it is correlated with oxygen cost and comparable to oxygen cost in its capability to discriminate between

healthy controls and subjects with stroke. The PCI can be performed inexpensively on a routine basis in a clinical environment.

2.6 Standing Balance

Balance control is a complex sensory and motor skill. It requires spatial and temporal integration of sensory input enabling the planning and execution of movement patterns that are necessary for controlling the center of body mass within the base of support (139-141). The complex nature of balance control implies that deficiencies in each of the related sensory or motor components, as well as deficiencies in the integration of these components, may adversely affect it (140,141).

It has been repeatedly demonstrated that following a stroke, patients often suffer from impaired balance control (139,142-145). Some patients never achieve the ability to stand (146), whereas the balance of those who are able to resume standing is typically characterized by increased sway during quiet stance and by an asymmetrical weight distribution between lower limbs with a shift in the average position of the center of pressure toward the unaffected lower extremity (145,147-149). These deficits affect the ability of subjects following a stroke to perform activities of daily living with confidence and are related to a high risk of falling (147,149,150).

Falling is a major concern among persons with stroke. Forster and Young (151) reported the incidence of falls to be as high as 73% of persons with stroke falling within 6 months following hospital discharge. More recently, Hyndman et al (152) reported 50% of persons with stroke living in the community experienced at least 1 fall over the course of a year, with approximately 50% of those individuals falling repeatedly.

Falls occur as a result of the inability to recover from a loss of balance, and postural stability plays a major role. Postural stability is defined as the ability to maintain the position of the body’s center of mass within specific boundaries of space

or stability limits (153) and can be quantified by the amount of body sway (i.e., postural sway).

Muscle weakness can be severe on the paretic side following stroke (154), with slight weakness with the non-paretic muscles in comparison to healthy older adults (155). The effects on the muscles ipsilateral to the lesion (i.e., non-paretic side) likely result from the small percentage of cortical tracts that descend ipsilaterally (156). Given that muscle strength (i.e., knee extensors) has been shown to be an independent predictor of postural sway in healthy older adults during standing on an unstable surface (157), one might expect to find similar results with persons with stroke.

However, given that standing balance does not require maximal muscle activation, it is possible that the effects from impairments of the sensory systems following a stroke have a larger role than motor impairment during quiet standing. Three major sensory systems, the visual, vestibular, and somatosensory, contribute to postural stability. Each system cannot provide the central nervous system (CNS) a complete picture of body position and movement for the control of posture, but rather it is the integration of all 3 systems that enables us to maintain balance and prevent a fall. Injury to the CNS from stroke may disrupt normal neural processing centers responsible for the processing and integration of afferent input from these systems and may contribute to the large number of falls in these individuals.

In stroke, postural stability is compromised (i.e., increased postural sway) when vision is removed (eyes closed) during standing compared to when vision is available (158). In addition, postural sway is correlated to ankle proprioception in stroke (158), suggesting an important role of the somatosensory system in these individuals. Furthermore, vestibular processing may be impaired in stroke and could potentially affect postural sway (159).

Although impairments in all 3 sensory systems may exist in persons with stroke, little is known about the ability to integrate afferent information in this

population and its relationship to postural stability and falls. Recently, Bonan et al (160) demonstrated greater postural sway during altered visual and ankle proprioception standing conditions in persons with stroke compared to healthy older adults.

Marigold et al (161) reported that muscle weakness appear to contribute to postural instability and falls in stroke patients. Laufer et al (162) indicated that the side of brain lesion seems to affect recovery of independent stance with an advantage to patients with right hemiparesis. However, there is no difference between balance controls of individuals with left versus right hemiparesis in patients who reach independent stance by the end of the first month following their stroke.

CHAPTER III
MATERIALS AND METHODS

3.1 Instrumentation

The following equipment were used in this study

1. Cycle ergometer (Cateye, USA)
2. Isokinetics Machine (Lido multi joint II, Loredan Biomedical, Inc.,CA)
3. Isokinetics programs (Lido multi joint II, Loredan Biomedical, Inc.,CA)
4. Force platform (KISTLER 9286AA)
5. Stop watch (Casio, Japan)
6. Treadmill (GE T2100)
7. Heart rate monitor (GE CASE)
8. Body Solid pulley system

3.2 Subject

Sixteen hemiparetic patients with a single cerebrovascular accident were recruited from Charoenkrungpracharak Hospital. All subjects were similar in age, weight, and height. The age ranges of subjects were between 40 and 70 years. All subjects had no history of injury or trauma to their lower extremity or arthritic conditions that restrict walking. All sixteen hemiparetic subjects were divided into two groups, (a): eight hemiparetic subjects with progressive resistance strength training (Hp), and (b): eight hemiparetic subjects with conventional strength training (Hc). All subjects were screened by the physician before included into this study.

The hemiparetic subjects had the following inclusion criteria:

-Having first onset of stroke caused by cerebrovascular accident and not from brain tumor, trauma or surgery.

-Post onset time more than 6 months.

-Hemiparetic stroke patients were in stable medical conditions.

-No other medical conditions that interfering exercise or testing program such as hypertension (BP>165/95 mmHg), cardiopulmonary complications, osteoarthritis of hip, knee, and ankle.

-Ability to understand and follow simple verbal instruction.

-Ability to walk independently without a walking aids for 10 meters.

-Ability to walk on a treadmill at constancy speed without human assistance or walking aids.

-Mild or moderate spasticity (modified Ashworth scale 1 - 3).

-Provided written or verbal informed consent.

The exclusion criteria were:

-Patients who had a cerebella or brain stem stroke.

-Patients who had other peripheral or central nervous system dysfunction.

-Patients who had active inflammatory or pathologic changes in the joint of the lower limbs, or foot deformities.

-Patients who had severe visual spatial dysfunction.

-Having significant musculoskeletal problems other than stroke.

-Medical instability (uncontrolled hypertension, arrhythmia, unstable cardiovascular status).

All subjects signed an inform consent prior to participation in the study.

3.3 Assessment

Subjects who met the inclusion criteria were asked to participate in the study. All subjects were explained about the objectives, process of testing and training of this study. All patients were asked to signed an inform consent prior to participation

in the study. In addition, the subjects who had recurrent stroke were excluded from the study.

3.3.1 Physical examination

All subjects were recorded for demographic characteristics including age, weight, height, time since stroke onset, gender, and paretic side. Data on the date of admission, medication, type of lesion, side of affected, and physical therapy treatment were collected from medical records. All evaluation were conducted when the subject are medically stable. Each patient were interviewed and examined by a physiotherapist about the muscle tone (Modified Ashworth scale), range of motion of lower extremity, proprioception of lower extremity, functional activities (Barthel index of activities of daily living), postural assessment (PASS), and functional ambulation (FAC).

3.3.2 Isokinetic measurement

The LIDO Multi Joint II, USA was used to measure isokinetic muscle strength for knee flexors/extensors, hip flexors/extensors, and ankle plantarflexors/dorsiflexors of both paretic and non-paretic lower extremities at angular velocity of $60°s^{-1}$. Calibration was performed immediately before the test, and the positioning and stabilization of subjects were performed in accordance with the LIDO Multi Joint II instruction manual. Subjects were allowed familiarization period of the task before data recording. After about 90 sec. rest, subjects were asked to push and pull as hard and as fast as possible throughout the full range of motion for 3 repetitions. For each subject, the average peak torque of knee flexors/extensors, hip flexors/extensor, and ankle plantarflexors/dorsiflexors of both paretic and non-paretic side were measured.

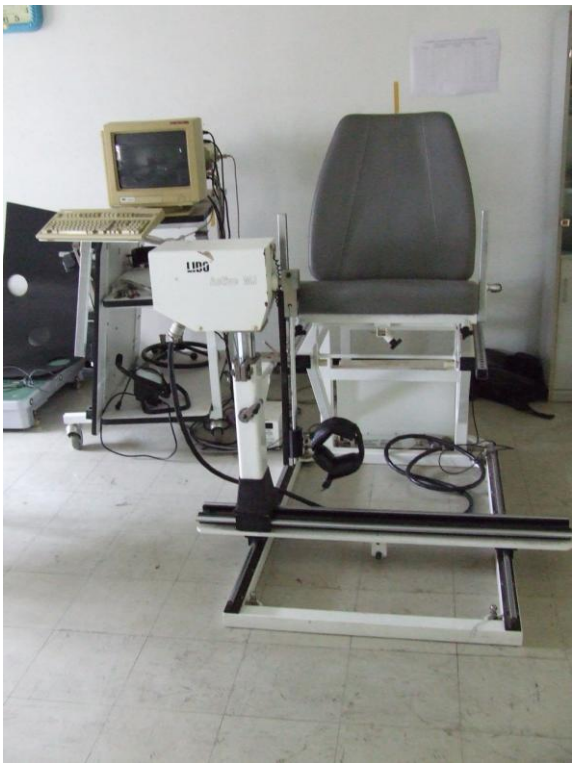


Figure 3.1 Isokinetics Machine (Lido multi joint II, Loredan Biomedical, Inc.,CA)

3.3.3 Force platform

All subjects were asked to walk barefeet at their most comfortable speeds along 6 meters walkway with force plate (KISTLER 9286AA) embedded in the midle. After each walking trial, the starting position of the subjects was adjusted for landing of the target leg on the force plate. The instruction was “walk as you normally would until you reach the line of platform”. If a subject appear to adjusted their step during the walking trial to target a force plate, clear explanations were provided to the subjects regarding the importance of walking in their usual manner during the tests and avoid targeting the force plate. Three appropriate trials were collected for each limb. The step was acceptable if the whole foot and no part of the contralateral foot land on the force plate during stride. Both of paretic and non-paretic lower limbs were measured for vertical ground reaction force, anterior-posterior ground reaction force, and medial-lateral ground reaction force. When necessary, 5-minute breaks were taken between trials to ensure that subjects were not fatigue and the trial reflects their true self-selected speed.

Figure 3.2 Force platform (KISTLER 9286AA)

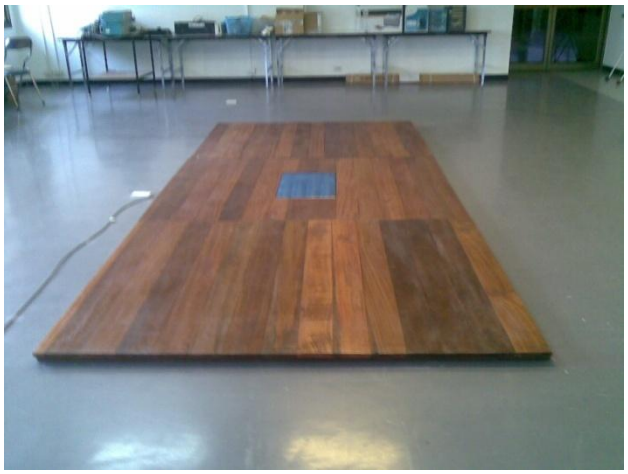


Figure 3.3 6-meter walkway with force platform

3.3.4 Maximum walking speed

Maximum walking speeds were collected when subjects were instructed to walk as fast as possible on the 6-meter walkway. Three appropriate trials were collected for further analysis. When necessary, 5-minute breaks were taken between trials to ensure that participants were not fatigue and the trial reflects their maximum walking speed.

3.3.5 Postural sway measurement

Postural sway were recorded using a force platform (KISTLER 9286AA). All subjects were asked to stand barefoot in the middle of the force platform, with

their arms across their chest. Subjects were tested under 2 conditions in a randomize order: standing with their feet together while looking at a target placed 2 meters away at their standing eye level (Eo), and standing with their feet together with their eye closed (Ec). Transducers embedded in a force platform registered the ground reaction forces and moments in the vertical, anterior-posterior, and medial-lateral directions. [The point of application of these ground reaction force vectors, which was the center of pressure, were calculated sway velocity (cm/s), sway area (cm^2), AP sway, and ML sway]. The center of pressure was measured for duration of 10 seconds. For the sway parameter test, subjects were performed 2 practice trials, follow by 3 testing trials. The average of the 3 testing trials will be collected for further analysis. When necessary, 5 minutes rest breaks were taken between trials to ensure that participants were not fatigue.

3.3.6 Measurement of gait performance

Subjects underwent an initial physician-supervised treadmill screening test to ascertain safety. All subjects were asked to walk barefoot on a treadmill for 12 min. with theirs self-selected velocity. [A treadmill was used during data acquisition (GE T2100). Gait speed and heart rate were set and recorded from the treadmill system (GE CASE)]. From these data, the Physiological Cost Index (PCI) was calculated as follows:

$$\text{PCI (beats/m)} = \frac{\text{walking heart rate (beats/min)} - \text{resting heart rate (beats/min)}}{\text{walking speed (m/min)}}$$

The time period at steady state was defined as that period of time during walking when there is a plateau of heart rate. Because there was a normal of heart rate of 3 to 4 beats per minute during rest or constant exercises, in this protocol, the plateau was defined as a period of at least 3 minutes, during which the variability of heart rate were less than 4 beats/minute.

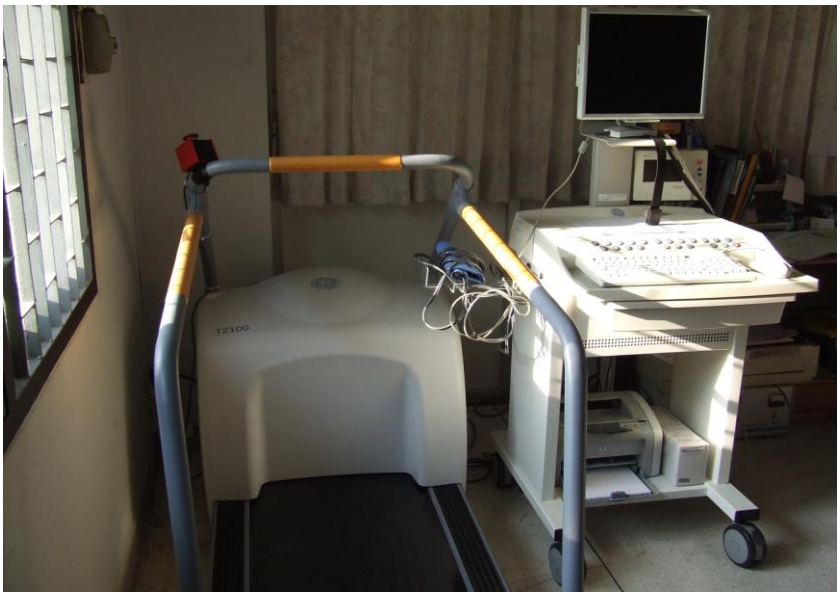


Figure 3.4 Treadmill (GE T2100) and Heart rate monitor (GE CASE)

3.4 Exercise Protocol

3.4.1 1RM muscle strength

The 1RM was used to measure muscle strength. This is defined as the maximum weight that could be lifted correctly for one repetition. In this study, the Brzycki (163) formula was used to predict 1RM of the subject. The 1 RM test was measure at baseline and every 2 weeks during the training period. The 1 RM muscle strength testing was performing all the muscle groups on both paretic and non-paretic side undergoing training and on the same equipment on which the subjects trained.

Brzycki formula for assessing 1RM:

$$1\ \text{RM} = \frac{\text{weight lifted}}{1.0278 - (0.0278 \times \text{repetitions})}$$

3.4.2 Progressive resistance strength training

All subjects in the progressive resistance strength training group (Hp) were performing exercise program for 6 weeks (3 days per week) in Charoenkrungpracharak Sport Medicine Center. The exercise program consists of the following components:

1. Warm-up on unload cycle ergometer for 3-5 minutes and stretching.

2. A circuit of the following exercises: Sitting knee extension and standing knee flexion. Standing hip flexion, and extension. Standing calf rise, using a Body Solid pulley system. Each of these exercises was performed unilaterally on both the paretic and non-paretic side. Hemiparetic subjects were able to do the exercises on the hemiparetic side through part of their range of motion. All subjects work up to 3 sets of 10 repetitions at 60%, 70%, and 80% of the 1 repetition maximum (1RM) on 1st, 3rd, and 5th week respectively. The rest between sessions are 5 minutes. 1RM was assessing every 2 weeks.

3. Cool-down on cycle ergometer for 3-5 minutes.

The entire exercise training program was supervised by physician and physiotherapist in Charoenkrungpracharak Physical Medicine and Rehabilitation unit.

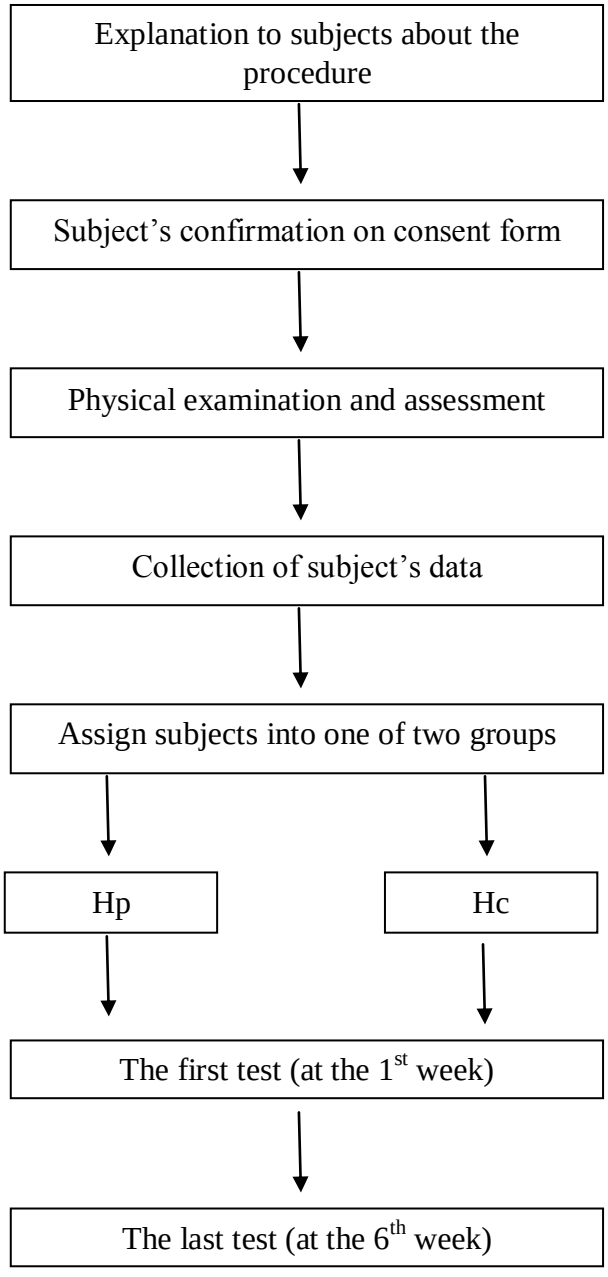
Figure 3.5 Body Solid Pulley Systems

3.4.3 Conventional strength training

Subjects in the conventional strength training group (Hc) were performed a therapeutic exercise supervised by a physiotherapist for 6 weeks (3days per week) and without increasing the resistance. The exercise program targeting on maintain lower limb muscle strength. The exercise program consists of the following components:

1. Warm-up on unload cycle ergometer for 3-5 minutes and stretching.
2. Therapeutic exercise strengthening program supervised by physiotherapist targeting on paretic and non-paretic lower limb (consists of hip flexion, hip extension, knee extension, knee flexion, ankle plantarflexion, ankle dorsiflexion) exercises.
3. Cool-down on cycle ergometer for 3-5 minutes.

3.5 Procedure of the study



3.6 Data analysis

Komogorov-Sminov Goodness of Fit test was used:

-To determined the distribution of the data.

Unpaired t-test was used:

-To compared subject's characteristics.

-To compared muscle average peak torque between Hp and Hc.

-To compared sway velocity, sway area, AP sway, and ML sway between Hp and Hc.

-To compared physiological cost index between Hp and Hc.

-To compared maximum walking speed between Hp and Hc.

Paired t-test was used:

-To compared muscle average peak torque between baseline and 6-week training in each group.

-To compared vertical, anterior-posterior and medial-lateral ground reaction force between baseline and 6-week training in each group.

-To compared sway velocity, sway area, AP sway, and ML sway in baseline and 6-week training in each group.

-To compared physiological cost index between baseline and 6-week training in each group.

-To compared maximum walking speed between baseline and 6-week training in each group.

The level of significance is set at a probability level (p-value) less than 0.05 ($p<0.05$)

CHAPTER IV
RESULTS

4.1 The characteristics of subjects

All sixteen subjects, eight were randomly allocated to the progressive resistance strength training group (Hp), and the other eight subjects were randomly allocated to the conventional strength training group (Hc). Table4.1 indicated the group mean and standard deviations for age, weight, height, stroke onset and the frequency counts for gender and hemiparetic side. There were no statistically significant differences between groups for age, weight, height, and stroke onset. All subjects successfully completed the study protocol and able to perform the exercises as planned.

Table4.1. Demographic data of subjects.

Variables	**Hp**(n=8)	**Hc**(n=8)	**p-value**
Age(years)	57.63(9.84)	57.38(7.69)	0.96
weight(kg)	71.94(15.31)	70.63(9.37)	0.84
height(cm)	162.88(7.3)	163.75(8.26)	0.83
time since stroke onset (months)	22.88(9.96)	23.63(9.07)	0.88
Gender			
male	4(50%)	4(50%)	
female	4(50%)	4(50%)	
Paretic side			
right	3(37.5%)	4(50%)	
left	5(62.5%)	4(50%)	

Values are means (standard deviation) or frequency (percentage).

Sixteen hemiparetic subjects met the inclusion criteria and participated in the study. Functional Ambulation Classification (FAC), Postural Assessment Scale for

Stroke Patients (PASS), Barthel Index of activities of daily living, and level of spasticity assessed by Modified Ashworth scale were presented in Table4.2.

Table4.2. Characteristics of hemiparetic subjects.

Characteristics	**Hp** (n=8)	**Hc** (n=8)
FAC	5	5
PASS	33	33
Barthel index	90	90
Modified Ashworth scale (spasticity)	mild	mild

Modified ashworth scale: 0 – 1 mild, 1^{+} - 2 moderate, 3 – 4 severe.

4.2 Average peak torque

The average peak torque of all muscle groups was measured by LIDO II isokinetic dynamometer at speed of $60°s^{-1}$. By measured before training (baseline), and after 6-week training, the values are means and standard deviations of average peak torque were presented in Table4.3.

Table4.3. Comparisons of average peak torque for both groups.

Variables	**Hp** (n=8)		**Hc** (n=8)		**Change scores**	
	Baseline	**6-week**	**Baseline**	**6-week**	**Hp**	**Hc**
KF (Nm)						
paretic	25(4.14)	38.63(5.29)*	25.75(9.35)	24.75(8.03)[a]	13.63(2.97)	-1(2.62)[b]
non-paretic	57.63(22.25)	74.25(24.55)*	52.63(19.7)	53.38(20.51)	16.63(3.16)	0.75(2.6)[b]
KE (Nm)						
paretic	69.38(29)	84.88(32.28)*	65.25(33.65)	64.38(34.99)	15.5(5.66)	-0.88(3.36)[b]
non-paretic	88.38(51.22)	120.38(50.57)*	92.38(38.69)	92(37.86)	32(14.71)	-0.38(2.45)[b]
HF (Nm)						
paretic	56.13(24.09)	74.75(22.35)*	55.25(24.48)	55.25(24.05)	18.63(11.02)	0(1.93)[b]
non-paretic	67.88(33.27)	90.25(34.03)*	69.75(34.85)	68.88(33.85)	22.38(12.03)	-0.88(2.03)[b]
HE (Nm)						
paretic	59(21.95)	72.38(29.1)*	55.13(25.11)	55.38(25.98)	13.38(13.44)	0.25(2.31)[b]
non-paretic	76.88(33.75)	101.5(36.44)*	71.38(32.39)	70.5(31.44)	24.63(6.35)	0.88(4.02)[b]
AD (Nm)						
paretic	11(4.93)	13.75(5.85)*	11.13(5.38)	10.88(4.45)	2.75(1.04)	-0.25(1.39)[b]
non-paretic	15.5(4.31)	19.88(4.67)*	16.38(6.28)	16.5(6.55)	4.38(0.92)	0.13(1.13)[b]
AP (Nm)						
paretic	20.38(12.88)	30.5(15.51)*	20.63(12.78)	20.5(11.45)	10.13(3.52)	-0.13(2.10)[b]
non-paretic	48.88(34.86)	59(32.72)*	43.63(16.38)	43.38(16.82)	10.13(5.62)	-0.25(1.83)[b]

Values are means (standard deviation), *within-group comparisons between baseline and 6-week training, statistical comparison show were paired t-test, significant value $p < 0.05$. [a]between group comparisons between Hp and Hc baseline and 6-week training, statistical comparison show were unpaired t-test, significant value $p < 0.05$. [b]between group comparisons between Hp and Hc change scores, statistical comparison show were unpaired t-test, significant value $p < 0.05$. KF=knee flexion, KE=knee extension, HF=hip flexion, HE=hip extension, AD=ankle dorsiflexion, AP=ankle plantarflexion.

At baseline, there were no significant differences between groups for both paretic and non-paretic knee flexors, knee extensors, hip flexors, hip extensors, ankle dorsiflexors, and ankle plantarflexors. After 6-week training, the average peak torques of all muscle groups both paretic and non-paretic side were significant improved from the baseline in the Hp. However, none were found in the Hc, as demonstrated in Figure 4.1 to 4.6.

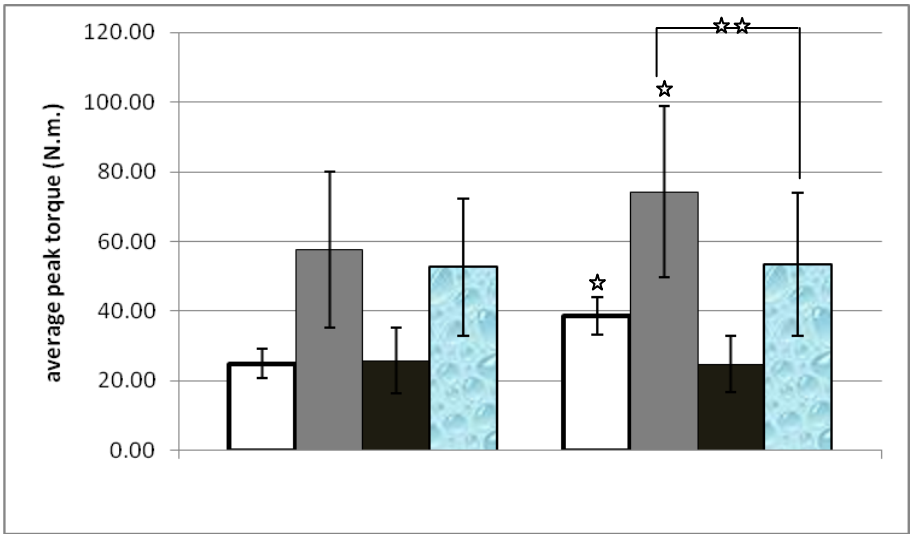


Figure 4.1 Comparison of knee flexion average peak torque between baseline and 6-week training. (☐ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc) ☆significant differences from baseline, ☆☆significant differences between group.

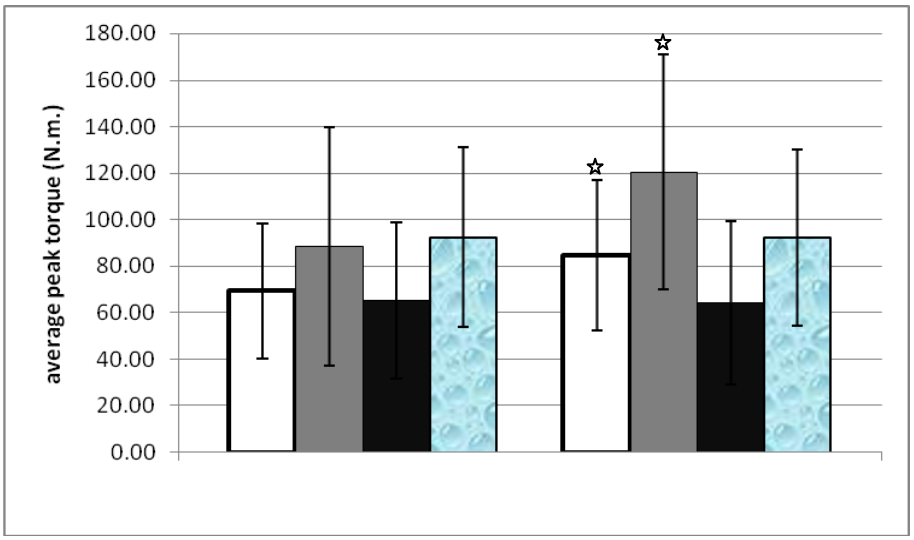


Figure 4.2 Comparison of knee extension average peak torque between baseline and 6-week training. (☐ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc) ☆significant differences from baseline.

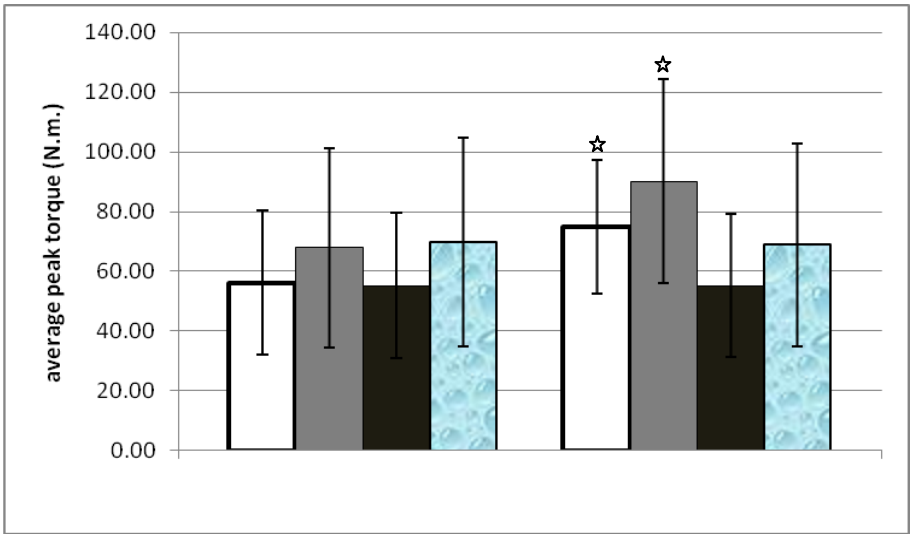


Figure 4.3 Comparison of hip flexion average peak torque between baseline and 6-week training. (paretic Hp, non paretic Hp, paretic Hc, non paretic Hc) ☆significant differences from baseline.

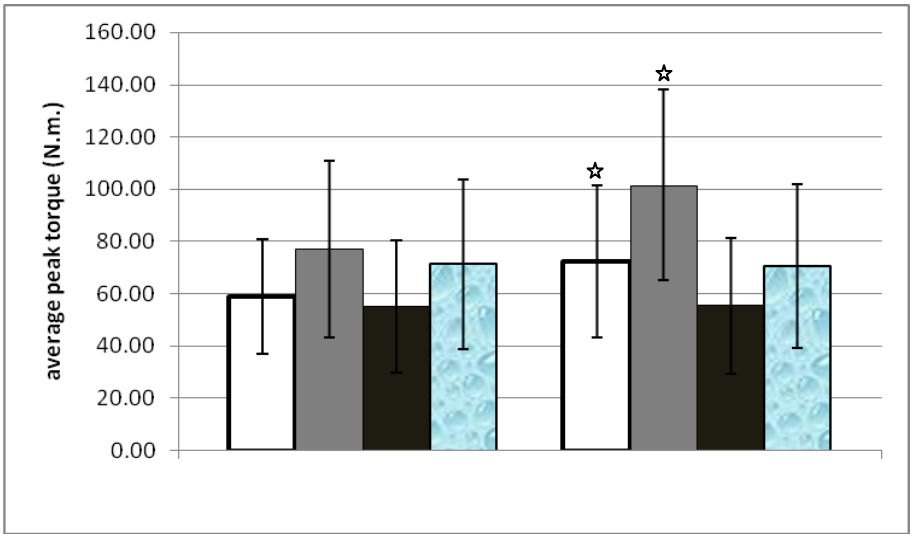


Figure 4.4 Comparison of hip extension average peak torque between baseline and 6-week training. (paretic Hp, non paretic Hp, paretic Hc, non paretic Hc) ☆significant differences from baseline.

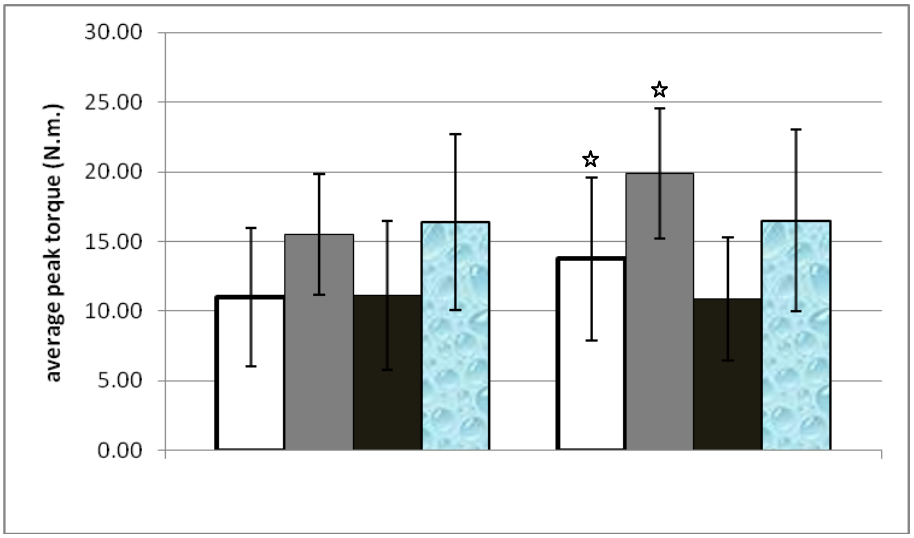


Figure 4.5 Comparison of ankle dorsiflexion average peak torque between baseline and 6-week training. (paretic Hp, non paretic Hp, paretic Hc, non paretic Hc), ☆significant differences from baseline.

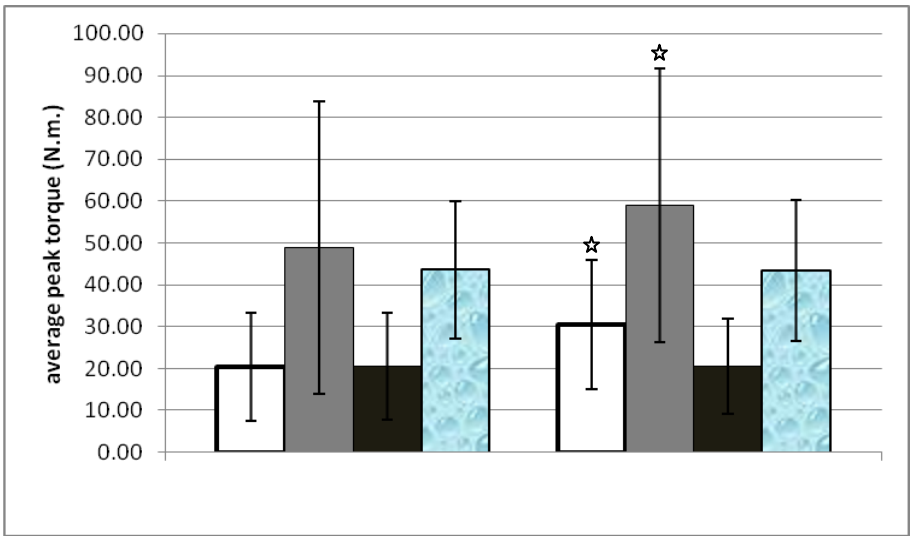


Figure 4.6 Comparison of ankle plantarflexion average peak torque between baseline and 6-week training. (paretic Hp, non paretic Hp, paretic Hc, non paretic Hc), ☆significant differences from baseline.

4.3 Ground reaction force

There are three axis of ground reaction force. The vertical ground reaction force (Fz) are composed of two peak, the first peak occurred during heel strike and the second peak occurred during the push off. The second axis was anterior-posterior ground reaction force (Fx), the first component was breaking force occurred durnig heel strike (Fx1), the second component was propulsive force occurred during push off (Fx2). The third axis was medial-lateral ground reaction force (Fy), the first component was lateral shear force (Fy1) and the second component was medial shear force (Fy2).

Table4.4. Comparisons of peak of walking ground reaction force for both groups.

Variables	**Hp (n=8)**		**Hc (n=8)**	
	Baseline	**6-week**	**Baseline**	**6-week**
Fz1 (BW)				
paretic	1.064(0.058)	1.055(0.029)	1.063(0.046)	1.061(0.047)*
non-paretic	1.013(0.075)	0.992(0.052)	1.017(0.060)	1.027(0.037)*
Fz2 (BW)				
paretic	1.024(0.059)	0.972(0.031)	1.029(0.058)	0.999(0.051)
non-paretic	1.052(0.060)	0.978(0.073)	1.056(0.041)	0.996(0.040)
Fx1 (BW)				
paretic	0.110(0.023)	0.123(0.045)*	0.104(0.027)	0.118(0.021)*
non-paretic	0.098(0.014)	0.104(0.019)*	0.099(0.022)	0.095(0.014)*
Fx2 (BW)				
paretic	0.092(0.032)	0.098(0.037)*	0.091(0.031)	0.095(0.019)*
non-paretic	0.128(0.018)	0.132(0.024)*	0.113(0.022)	0.117(0.021)*
Fy1 (BW)				
paretic	0.030(0.018)	0.028(0.021)*	0.031(0.023)	0.032(0.021)*
non-paretic	0.041(0.018)	0.041(0.020)*	0.049(0.026)	0.052(0.021)*
Fy2 (BW)				
paretic	0.081(0.036)	0.072(0.030)*	0.083(0.021)	0.090(0.014)*
non-paretic	0.073(0.027)	0.069(0.034)*	0.072(0.015)	0.075(0.017)*

Values are means (standard deviation), *within-group comparisons between baseline and 6-week training, statistical comparison show were paired t-test, significant value $p < 0.05$.

Table4.4 showed the significant differences between baseline and 6-week training both paretic and non-paretic side in Fx1, Fx2, Fy1, and Fy2 of Hp and both paretic and non-paretic side in Fz1, Fx1, Fx2, Fy1, and Fy2 of Hc. The comparisons of walking ground reaction force in both groups were demonstrated in Figure 4.7 to 4.12.

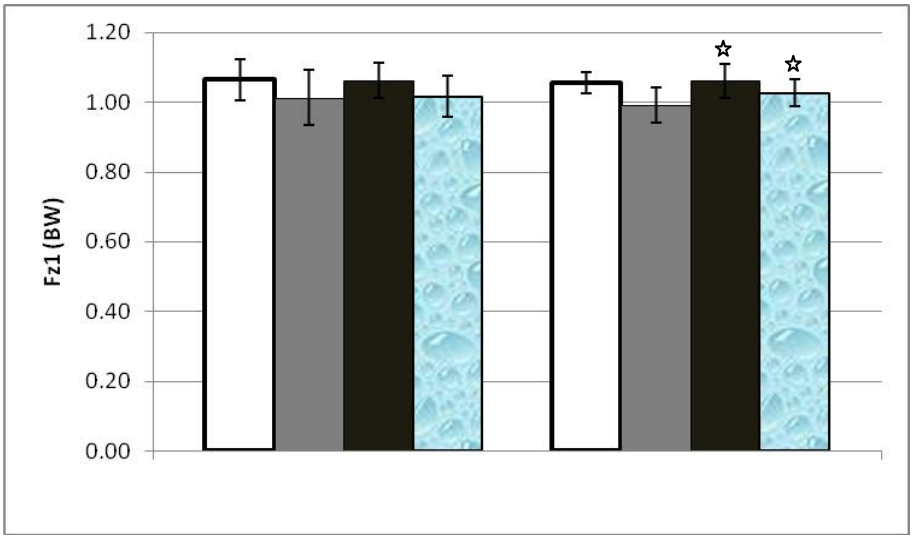


Figure 4.7 Comparison of Fz1 between baseline and 6-week training. (□ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc), ☆ significant differences from baseline.

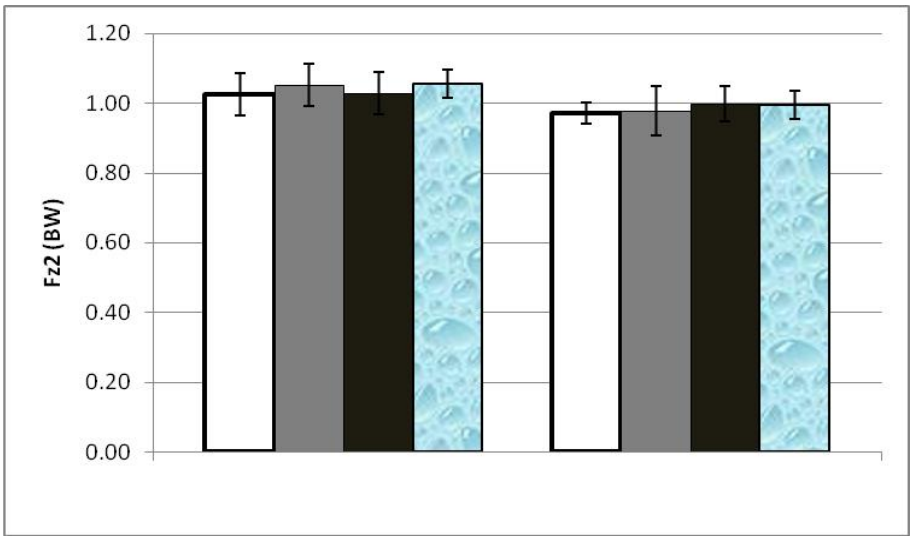


Figure 4.8 Comparison of Fz2 between baseline and 6-week training. (□ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc).

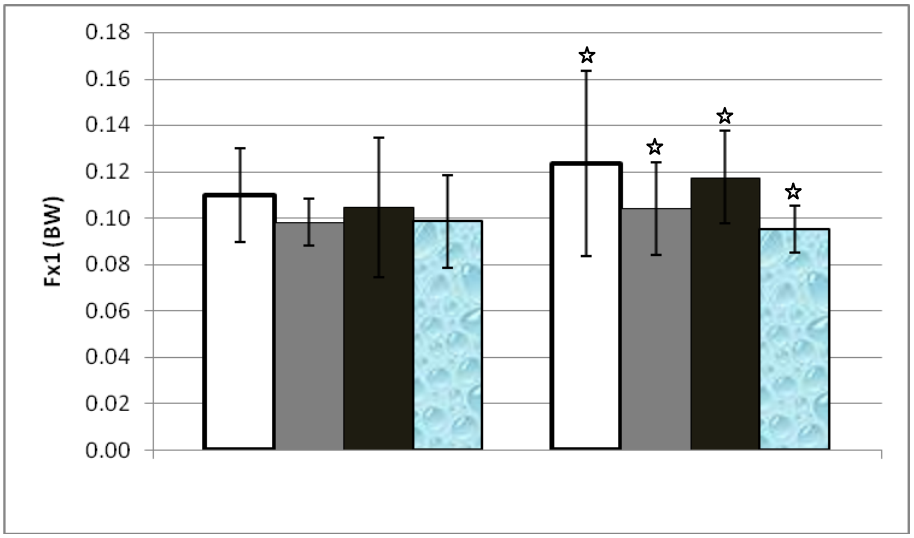


Figure 4.9 Comparison of Fx1 between baseline and 6-week training. (□ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc) , ☆ significant differences from baseline.

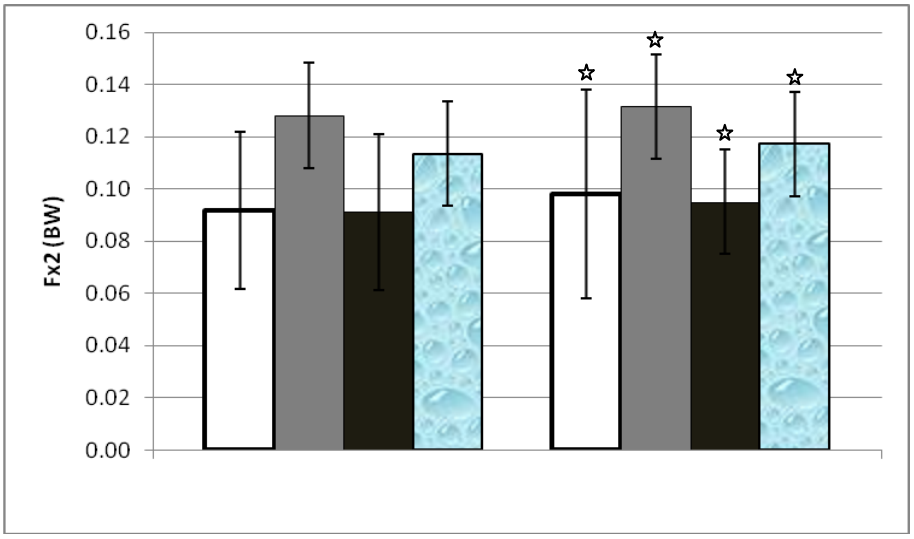


Figure 4.10 Comparison of Fx2 between baseline and 6-week training. (□ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc)) , ☆ significant differences from baseline.

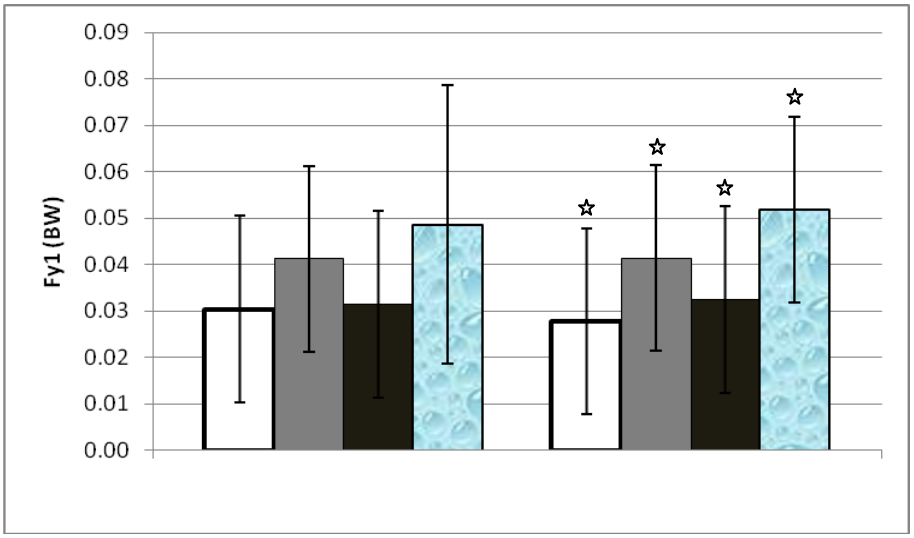


Figure 4.11 Comparison of Fy1 between baseline and 6-week training. (□ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc)) , ☆ significant differences from baseline.

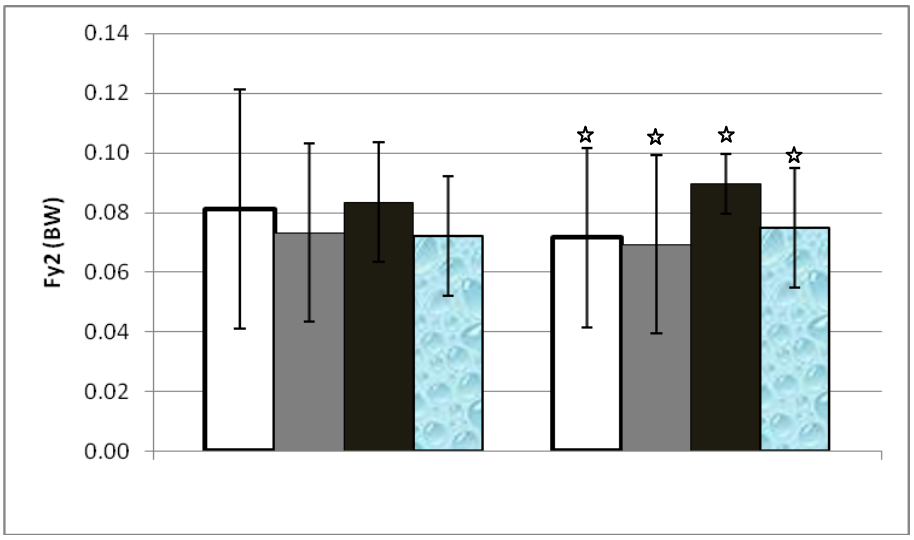


Figure 4.12 Comparison of Fy2 between baseline and 6-week training. (□ paretic Hp, ▒ non paretic Hp, ■ paretic Hc, ▒ non paretic Hc), ☆ significant differences from baseline.

Table 4.5. Comparison of paretic and non-paretic stance time.

Stance time (s)	**Hp** (n=8)		**Hc** (n=8)	
	Baseline	**6-week**	**Baseline**	**6-week**
Paretic	0.84(0.1)	0.82(0.1)	0.9(0.11)	0.87(0.09)
non-paretic	0.89(0.19)	0.88(0.07)	0.92(0.08)	0.92(0.11)

Values are means (standard deviation).

Table 4.5 showed that there were no significant differences of paretic and non-paretic stance time between baseline and 6-week training in both groups, as showed in Figure 4.13.

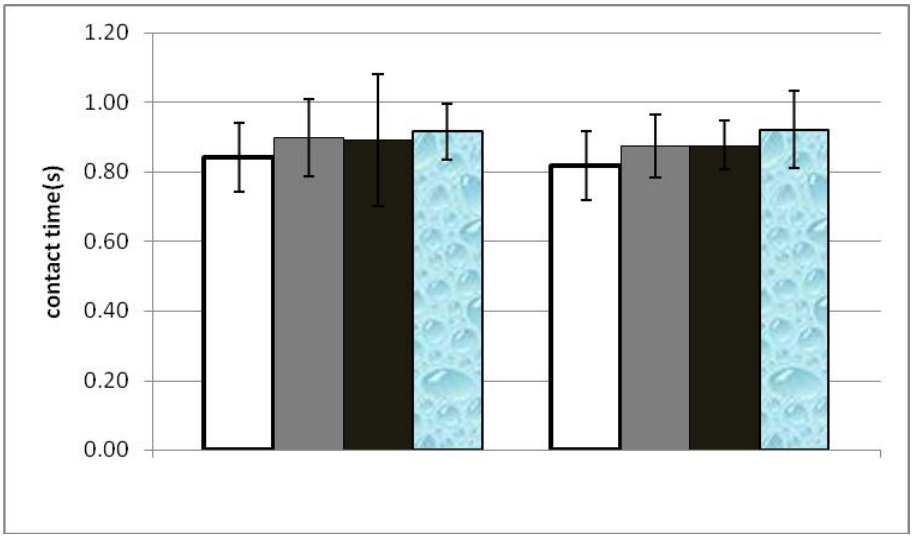


Figure 4.13 Comparison of stance time between baseline and 6-week training. (paretic Hp, non paretic Hp, paretic Hc, non paretic Hc).

Table 4.6 Comparison of paretic and non-paretic heel strike transient.

Heel strike transient (BW)	**Hp** (n=8)		**Hc** (n=8)	
	Baseline	**6-week**	**Baseline**	**6-week**
Paretic	0.54(0.13)	0.51(0.17)	0.53(0.12)	0.54(0.06)
non-paretic	0.42(0.11)	0.44(0.11)	0.45(0.15)	0.48(0.08)

Values are means (standard deviation).

From Table 4.6, there were no significant differences of paretic and non-paretic heel strike transient between baseline and 6-week training in both groups, as demonstrated in Figure 4.14.

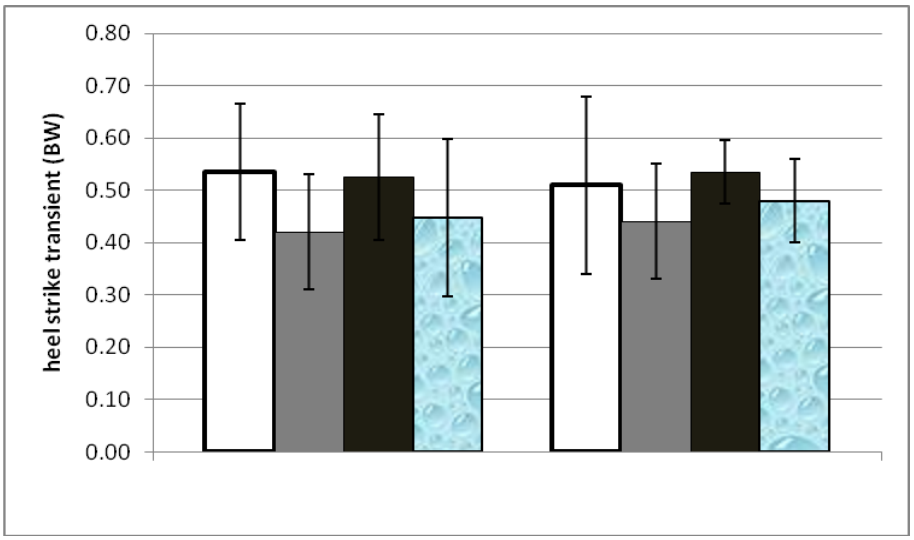


Figure 4.14 Comparison of heel strike transient between baseline and 6-week training. (paretic Hp, non paretic Hp, paretic Hc, non paretic Hc).

4.4 Postural sway

Postural sway was recorded using force platform (KISTLER 9286AA) under 2 conditions in a randomized order: double feet standing while looking at a target placed 2 meters away at their standing eye level (EO) and double feet standing with their eyes closed (EC). Sway velocity, sway area, anterior-posterior sway (APsway), and medial-lateral sway (MLsway) were recorded for duration of 10 seconds. Means and standard deviation of sway velocity, sway area, APsway, and MLsway were presented in Table4.7.

Table4.7. Comparisons of postural sway for both groups.

Variables	**Hp** (n=8)		**Hc** (n=8)		**Change scores**	
	Baseline	**6-week**	**Baseline**	**6-week**	**Hp**	**Hc**
Eye opened						
sway velocity(cm/s)	2(0.34)	1.66(0.29)*	2.09(0.54)	1.64(0.43)	- 0.35(0.38)	- 0.45(0.68)
sway area (cm^2)	0.89(1.16)	0.62(0.57)	1.81(2.16)	1.81(1.72)	- 0.27(1.33)	0(2.12)
APsway (cm)	0.37(0.29)	0.17(0.08)	0.53(0.27)	0.51(0.24)^	- 0.2(0.3)	- 0.2(0.26)
MLsway (cm)	0.23(0.19)	0.41(0.22)	0.36(0.33)	0.4(0.32)	0.18(0.29)	0.05(0.38)
Eye closed						
sway velocity(cm/s)	1.97(0.34)	1.67(0.33)	1.91(0.32)	1.75(0.49)	- 0.3(0.52)	- 0.16(0.54)
sway area (cm^2)	1.25(1.54)	1.11(0.68)	1.46(1.64)	1.82(1.03)	- 0.13(1.38)	0.36(1.39)
APsway (cm)	0.5(0.41)	0.51(0.5)	0.51(0.35)	0.58(0.28)	0.01(0.24)	0.08(0.39)
MLsway (cm)	0.3(0.29)	0.42(0.3)	0.33(0.18)	0.42(0.18)	0.11(0.37)	0.09(0.16)

Values are means (standard deviation), *within-group comparisons between baseline and 6-week training, statistical comparison show were paired t-test, significant value $p < 0.05$. ^between-group comparisons between 6-week training of Hp and 6-week training of Hc, statistical comparison show were unpaired t-test, significant value $p < 0.05$.

Table4.7 revealed that after 6-week training, there were significant differences in sway velocity in Hp in eye opened condition. However, none were found in Hc. There were no significant differences at the baseline between the 2 groups. However, after 6-week training the result showed significant difference only in anterior-posterior sway (AP sway) under the eye opened condition. The comparison between Hp and Hc in baseline postural sway parameters and 6-week training postural sway parameters under eye opened and eye closed conditions were demonstrated in Figure 4.15 to 4.18.

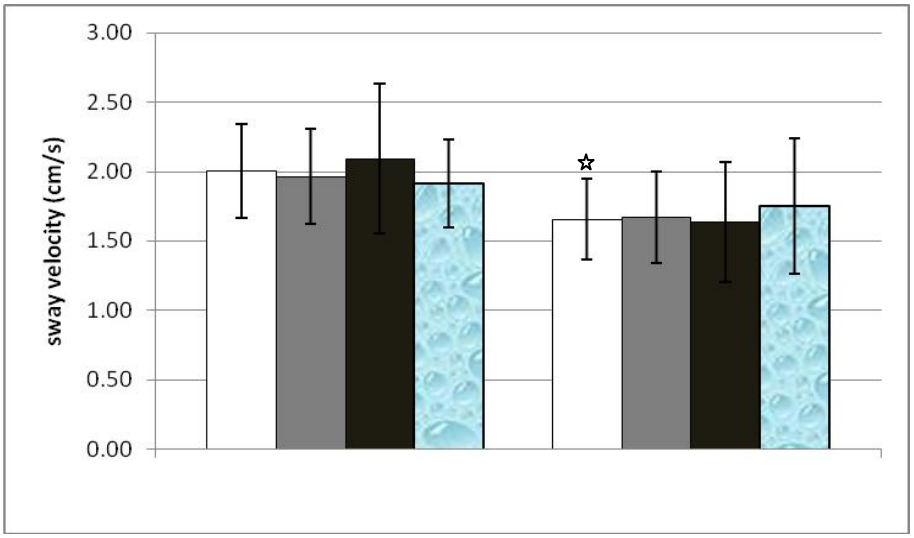


Figure 4.15 Comparison of sway velocity between baseline and 6-week training. (□ eyes opened Hp, ▒ eyes closed Hp, ■ eyes opened Hc, ▒ eyes closed Hc)
☆ significant differences from baseline.

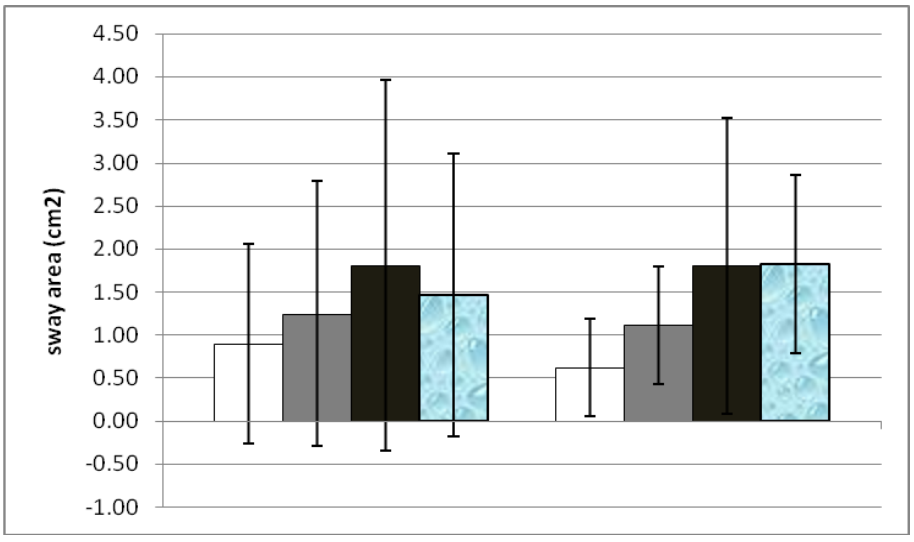


Figure 4.16 Comparison of sway area between baseline and 6-week training. (□ eyes opened Hp, ▒ eyes closed Hp, ■ eyes opened Hc, ▒ eyes closed Hc).

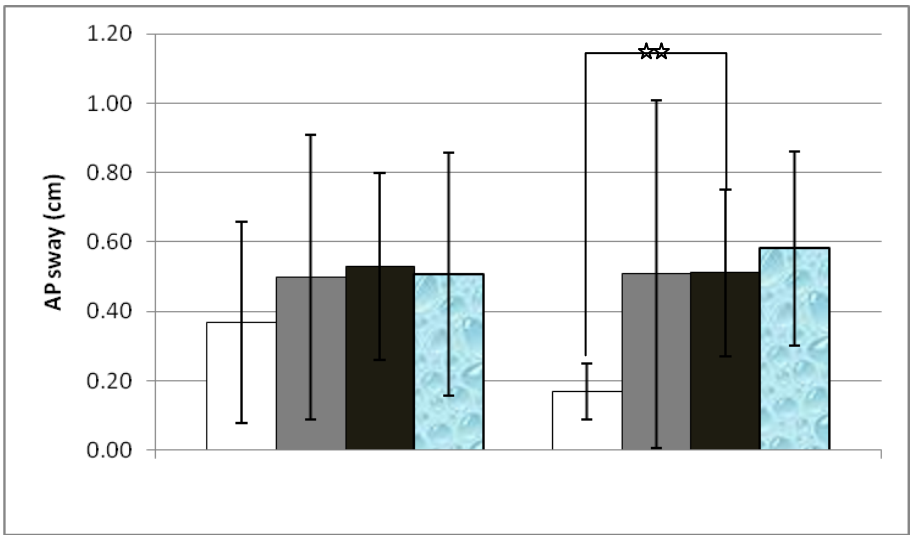


Figure 4.17 Comparison of AP sway area between baseline and 6-week training. (□eyes opened Hp, ▒ eyes closed Hp, ■ eyes opened Hc, ▒eyes closed Hc) ☆☆significant differences between group.

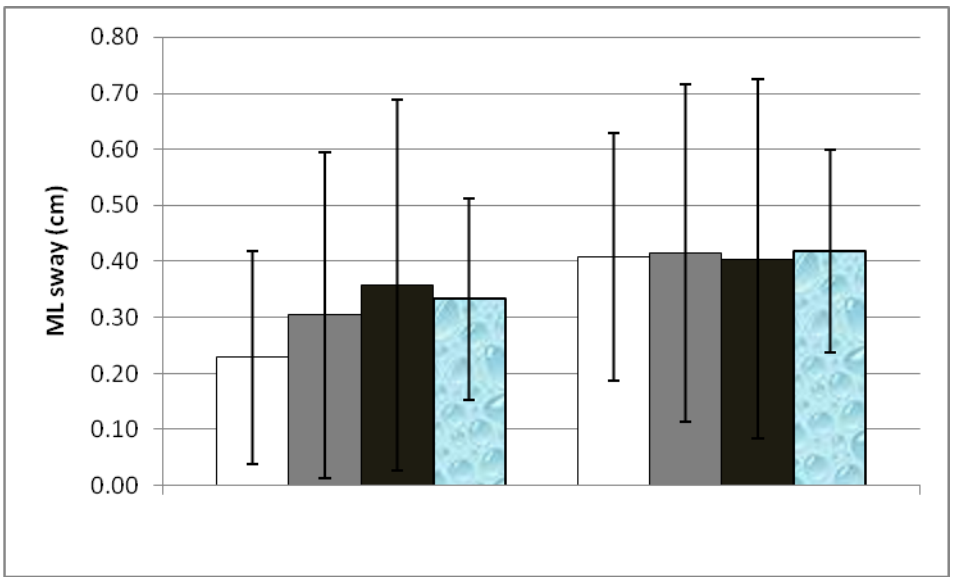


Figure 4.18 Comparison of ML sway area between baseline and 6-week training. (□eyes opened Hp, ▒eyes closed Hp, ■eyes opened Hc, ▒eyes closed Hc).

4.5 Physiological Cost Index and maximum walking speed

The physiological cost index and maximum walking speed have been used to studying the gait performance of subjects. Means and standard deviation of physiological cost index and walking speed were presented in Table4.8.

Table4.8. Comparisons of physiological cost index for both groups.

Variables	**Hp** (n=8)		**Hc** (n=8)	
	baseline	**6-week**	**baseline**	**6-week**
PCI (beats/m)	0.67 (0.18)	0.43 (0.17)*	0.69 (0.14)	0.68 (0.14)^

Values are means (standard deviation), *within-group comparisons between baseline and 6-week training, statistical comparison show were paired t-test, significant value $p < 0.05$. ^between-group comparisons between 6-week training of Hp and 6-week training of Hc, statistical comparison show were unpaired t-test, significant value $p < 0.05$.

Table4.8 revealed that after 6-week training, the physiological cost index were significant differences from baseline in Hp, while none were found in Hc. When compared between groups, there were no significant differences at the baseline between the 2 groups. However, after 6-week training the results showed significant differences in physiological cost index between groups. The comparisons of physiological cost index were demonstrated in Figure 4.19.

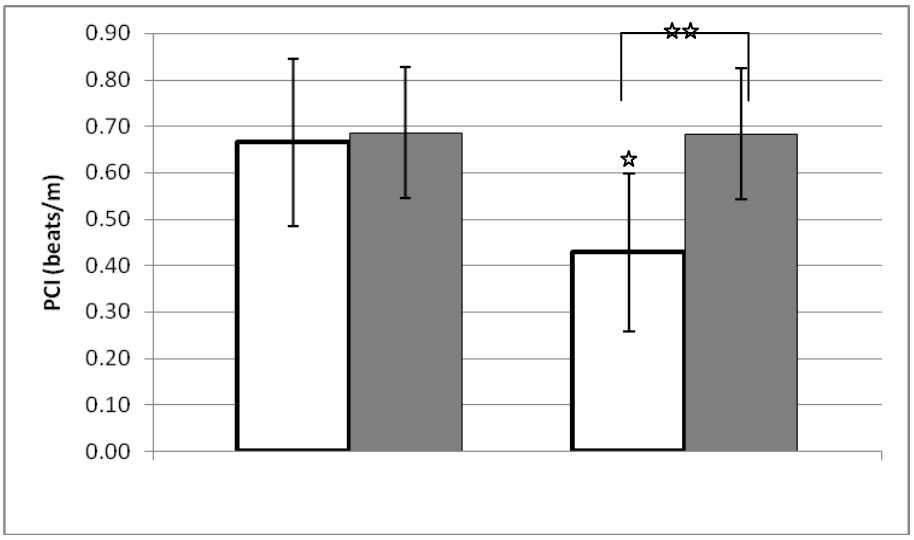


Figure 4.19 Comparison of physiological cost index between baseline and 6-week training (□ Hp, ■ Hc)☆ significant differences from baseline,☆☆ significant differences between groups

Table 4.9 Physiological cost index components of Hp.

Variables	Baseline	6-week	p-value
HRrest (beats/min)	76.88(11.69)	76.88(12.99)	0.98
HRexercise (beats/min)	101.08(6.39)	97.38(6.99)	0.22
Walking speed (m/min)	38.76(12.47)	48.76(10.53)	0.04

Values are means (standard deviation).

Table 4.9 showed the significant differences of Hp treadmill selected walking speed between baseline and 6-week training. However, there were no significant differences of HRrest and HRexercise between baseline and 6-week training.

Table 4.10 Comparisons of maximum walking speed for both groups.

Variables	**Hp** (n=8)		**Hc** (n=8)	
	baseline	**6-week**	**baseline**	**6-week**
Maximum walking speed (m/s)	0.79 (0.15)	1.05 (0.27)*	0.77 (0.14)	0.77 (0.14)^

Values are means (standard deviation), *within-group comparisons between baseline and 6-week training, statistical comparison show were paired t-test, significant value $p < 0.05$. ^between-group comparisons between 6-week training of Hp and 6-week training of Hc, statistical comparison show were unpaired t-test, significant value $p < 0.05$.

Table 4.10 revealed that after 6-week training, the maximum walking speed were significant differences from baseline in Hp, while none were found in Hc. When compared between groups, there were no significant differences at the baseline between the 2 groups. However, after 6-week training the results showed significant differences in maximum walking speed between groups. The comparisons of maximum walking speed were demonstrated in Figure 4.20.

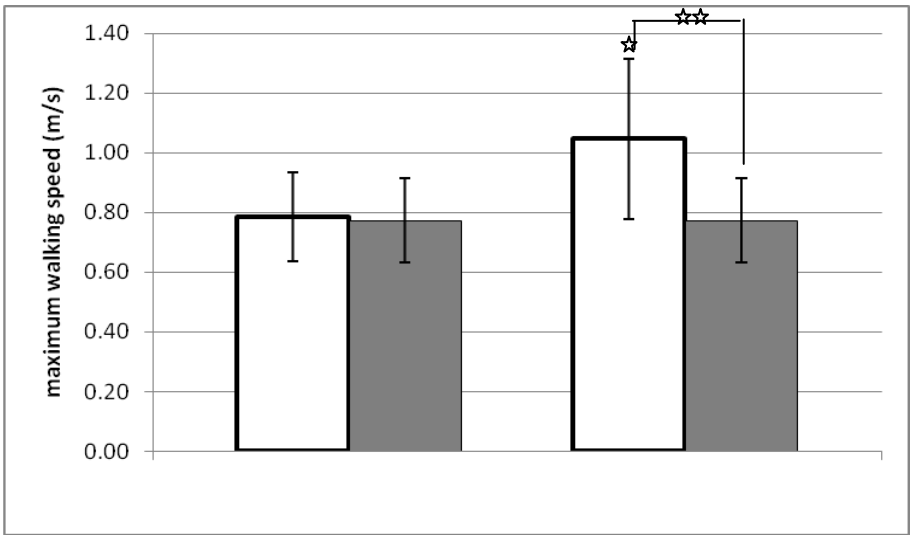


Figure 4.20 Comparison of maximum walking speed between baseline and 6-week training (□ Hp, ■ Hc) ☆ significant differences from baseline, ☆☆ significant differences between groups

CHAPTER V
DISCUSSION

As muscle weakness is a common impairment after stroke, interventions that can improve muscle strength are an important part of stroke rehabilitation. The aim of this study is to evaluate the effects of progressive resistance strength training program on lower extremity muscle strength, walking ground reaction forces, postural sway, physiological cost index, and maximum walking speed.

5.1 Characteristics of subjects

All subjects in this study were volunteered from Sports Medicine section, Department of Physical Medicine and Rehabilitation, Charoenkrungpracharak Hospital, where their exercise programs and activities could be controlled. Totally, there were 16 hemiparetic patients (8 males and 8 females). Nine subjects were left hemiplegia and seven subjects were right hemiplegia. The ages of these subjects were between 41 to 70 years.

Subjects were divided into 2 groups of different program training: 8 hemiparetic patients were in progressive resistance strength training group (Hp) and the others 8 were in conventional strength training group (Hc). Both groups were similar in age, weight, height, gender, time since stroke onset, Functional Ambulation Classification (FAC) scores, Postural Assessment Scale for Stroke Patients (PASS) scores, Barthel Index of activities of daily living scores, and level of spasticity.

This study included the analysis on conventional strength training intervention for comparing with the progressive resistance strength training intervention. If the average peak torque of lower extremity, walking ground reaction

force, standing postural sway, physiological cost index during walking and maximum walking speed did not change in the conventional strength training group, while improvement was noted following progressive resistance strength training group, then, it could be concluded that the improvement was resulted from the effects of progressive resistance strength training intervention, not from spontaneous recovery.

5.2 Average peak torque of lower extremity

The present study demonstrated that the 6-week of progressive resistance strength training intervention were significant improved lower extremity average peak torque of knee flexors (55%), knee extensors (22%), hip flexors (33%), hip extensors (23%), ankle dorsiflexors (25%), and ankle plantarflexors (50%) of the paretic side. The significantly improvement were also found in non-paretic knee flexors (29%), knee extensors (36%), hip flexors (33%), hip extensors (32%), ankle dorsiflexors (29%), and ankle plantarflexors (20%). However, there were no significant improvements in conventional strength training intervention.

The improvements in muscle strength are comparable with previous studies of chronic post-stroke subjects. Previous studies have examined the effects of strength training on the paretic lower extremity after stroke (164-166) and many have combined strength training with other interventions (165-168). For example, using a 10-week isotonic lower extremity progressive resistance strength training intervention, Teixeira-Salmela et al. (168) showed a 42.3% total mean torque improvement in the paretic lower extremity muscle groups trained, including an 18% to 46% gain for paretic knee extensors. Weiss et al. (134) reported that dynamic paretic knee extensors strength increased 67% and non-paretic knee extensors strength increased 42%. These differences may reflect the varying degree of stroke severity, the length of time since stroke onset, or the specific exercise training protocol used.

The comparison of the mean differences change scores between progressive resistance strength training group and conventional strength training group

indicated that, after 6-week training, there were significant differences in both paretic and non-paretic side change scores between progressive resistance strength training group and conventional strength training group. However, the conventional strength training group showed some decreased in strength of paretic knee flexion, paretic knee extension, non-paretic knee extension, non-paretic hip flexion, paretic ankle dorsiflexion, paretic ankle plantarflexion, and non-paretic ankle plantarflexion after 6-week training. From the results of this study, it could be concluded that the progressive resistance strength training intervention had greater benefits of average peak torque than conventional strength training intervention.

In addition, all subjects in the present study had low or no increased muscle tone at baseline and no increase was detected after progressive resistance strength training, muscle tone actually remained the same as baseline throughout the study. This finding is consistent with spasticity being rare in hemiparetic stroke patients with mild to moderate disability (169) and progressive resistance strength training intervention having no negative effect on muscle tone (164,168,170,171).

5.3 Ground reaction force

A comparison of walking ground reaction force between baseline and 6-week training revealed the significant differences of paretic and non-paretic Fx1, Fx2, Fy1, Fy2 in both groups and Fz2 in conventional strength training group. However, a comparison of stance time and heel strike transient between baseline and 6-week training showed no significant differences in both groups.

Clinically, the paretic initial contact heel strike transient that reflects the peak breaking force was decreased after 6-week training in progressive resistance strength training group, this results may due to the increase in pretibial muscle activation by eccentric contraction of anterior tibialis and eccentric contraction of quadriceps to opposed the external flexion moment and prevent the excessive heel strike. The result of the first peak of vertical ground reaction force (Fz1) during

loading response in progressive resistance strength training group were no significant differences from baseline, while there were significant changed in the conventional strength training group. For the second peak of vertical ground reaction force (Fz2) during late terminal stance found that there were no significant differences between baseline and 6-week training of both paretic and non-paretic in both groups.

The anterior-posterior ground reaction force, by the breaking force that occurred at initial contact (Fx1) after 6-week training was greater than baseline in both paretic and non-paretic side of progressive resistance strength training group. These significant changes were also found in conventional strength training group. The propulsive force that occurred at push off (Fx2) of 6-week training was greater than baseline in both paretic and non-paretic side of both groups.

The medial-lateral ground reaction force related to balance of the body during walking. The result of this study demonstrated that, after 6-week training the lateral shear force (Fy1) were significant differences from the baseline in both paretic and non-paretic of both group. Moreover, the results of medial shear force (Fy2) were also significant differences from baseline.

For the stance time, there were no significant changed after 6-week training of both paretic and non-paretic side in both groups. These indicated that, both strength training interventions were not directly related to the changes of comfortable walking speed in hemiparetic patients.

Even the strength lower limb muscles were significant increased in both paretic and non-paretic side of progressive resistance strength training group. However, the results were remained unclear for the walking ground reaction force. Because there were confounding factors that affect walking ground reaction force, for examples, walking speed and motor recovery in stroke patients (172). For safety reason, this study allowed the subjects to walk at their comfortable walking speed during data collection to prevent risk of falling and that directly affects to the walking

ground reaction force data. The others possible reason was the muscles coordination patterns (synergy pattern, slow increased and/or decreased muscular tension) that remained inadequate after 6-week of strength training. Further research is needed to control these confounding factors of the subjects during walking.

5.4 Postural sway

After 6-week training, the progressive resistance strength training group showed the significant improvements in the sway velocity. The improvements were also found by the decreased of sway area, and AP sway under the eye opened condition and the decreased of sway velocity, and sway area under the eye closed condition. For the conventional strength training group, the improvements were found in the decreased of sway velocity under both conditions, and AP sway under eye opened condition. However, most of these improvements were no significant differences from baseline but for the clinical it could be benefit from strength training. The significant differences between groups were only found in AP sway under eye opened condition.

This lack of significant change implies that postural sway parameters may not directly relate to changes in muscle strength. Because postural control is a complex sensory and motor skill. It requires spatial and temporal integration of sensory input enabling the planning and execution of movement patterns that are necessary for controlling the center of body mass within the base of support (139-141). However, standing balance may do not require maximal muscle activation, it is possible that the effects from impairments of the sensory systems following a stroke have a larger role than motor impairment during quiet standing. Three major sensory systems, the visual, vestibular, and somatosensory, contribute to postural stability. Each system cannot provide the central nervous system (CNS) a complete picture of body position and movement for the control of posture. Injury to the CNS from stroke may disrupt normal neural processing centers responsible for the processing and integration of

afferent input from these systems and may contribute to the large number of falls in these individuals.

These indicate that progressive resistance strength training alone was no benefit on the postural sway parameters. Further research is needed to identify and combine specific interventions that enhance recovery of postural sway parameters.

5.5 Physiological cost index and maximum walking speed

It is generally accepted that locomotors impairment, which results from cerebrovascular disease, increases the walking PCI more than healthy subjects at matched speed (173). It seems that hemiparetic patients decreased their walking speed to maintain their walking PCI at a level they could sustain for a long time. The same strategy, i.e. decreasing speed of motion to maintain a reasonable power in spite of increasing energy cost, is adopted by hemiparetic patients when climbing stairs (174).

The present study revealed that after 6-week training, the progressive resistance strength training group were significant decreased the walking PCI from 0.67 to 0.43 beats/m. On the contrary, there were no significant changed of the walking PCI in conventional strength training group. These changed in walking PCI in Hp was the results of an increased in selected treadmill walking speed after 6-week of progressive resistance strength training intervention not from the changes of resting heart rate or exercise heart rate.

Recent studies have shown that muscle strength of the paretic lower extremity was closely related to the walking speed in hemiparetic stroke patients (18,175,176). Previous studies (9,177,178) reported that work production at the hip and ankle were highly important resource in controlling and modulating gait velocity. Production of work at the hip and ankle has been shown to be positively related to preferred gait speed in subjects with stroke (9,179). During a rehabilitation program with a hemiparesis group, Parvatanemi et al. (180) investigated muscle group work

variation in relation to an increase in preferred gait speed. They found that increase in preferred gait speed was associated with increase in work at both the hip and the ankle.

Similarly to the walking PCI, the maximum walking speed were significant increased from 0.79 to 1.05 m/s in progressive resistance strength training group, while there were no significant changed of maximum walking speed in conventional strength training group. It can be concluded that the progressive resistance strength training intervention could improved the ability of lower limb muscles to generate force on both paretic and non-paretic sides during walking.

CHAPTER VI
CONCLUSION

This present study was to compare the effects of progressive resistance strength training intervention and conventional strength training intervention on average peak torque of paretic and non-paretic lower limb muscles, paretic and non-paretic walking ground reaction force, standing postural sway, physiological cost index during walking, and maximum walking speed in first onset of stroke patients. Sixteen patients were volunteered for the study. The patient in experimental group was received progressive resistance strength training intervention for 6 weeks, while the other group was received conventional strength training intervention for 6 weeks.

In summary, this study demonstrated that after 6-week training a long-term stroke survivors have the capacity to safely improve lower extremity musculoskeletal strength in both the paretic and non-paretic limbs with progressive resistance strength training intervention and these improvements led to a reduction of physiological cost index during walking and increased in maximum walking speed. However, there were significant changed in walking ground reaction force in both groups but the results were remained unclear because these changes were not directly related to the improvements in strength of lower limb muscles. Clinically, the improvements were found in standing postural sway after 6-week of progressive resistance strength training intervention. However in statistical, these improvements were no significant differences from the baseline.

The present results showed that progressive resistance strength training can profoundly improve strength and result in modest improvements in function late in the course of stroke recovery. However, the number of participating subjects was relatively small, which affects the statistical power and lack of some significance

between progressive resistance strength training group and conventional strength training group. There was difficulty recruiting enough eligible participants, because many of the contacted subjects who expressed an interest were either unable to walk independently. Further studies should address the video graphic technique or electromyography (EMG) during data collection.

REFERENCE

1. Viriyavejakul A. Stroke in Asia: An epidemiological consideration. Clin Neurophamacol 1990;13 suppl 3:526-33.
2. Wood-Dauphince S. The epidemiology of stroke: relevance for physical therapists. Physiother Can 1985;37:377-86.
3. O'Sullian S. Stroke. In: O'Sullivan S, Schmitz TJ, editors. Physical rehabilitation assessment and treatment. 3rd ed. Philadelphia: FA Davis Company; 1994, p. 327-60.
4. Rosenfalck A, Andreassen S. Impaired regulation of force and firing pattern of single motor units in patients with spasticity. J Neurol Neurosurg Psychiatry 1980;43:907-16.
5. Fish DK, Kosta CS. Walking impediments and gait inefficiencies in the CVA patient. J Prosthet Orthot 1999;11:33-7.
6. Potempa K, Lopez M, Braun LT, Szidon JP, Fogg L, Tincknell T. Physiological outcomes of aerobic exercise training in hemiparetic stroke patients. Stroke 1995;26:101-5.
7. Brandstater ME, de Bruin H, Gowland C, Clark BM. Hemiplegic gait: analysis of temporal variables. Arch Phys Med Rehabil 1983;64:583-7.
8. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiplegic patients. Brain 1979;102:405-30.
9. Olney SJ, Griffin MP, McBride ID. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. Phys Ther 1994;74:872-85.
10. Wade DT, Heller WA, Maggs J, Hewer RL. Walking after stroke. Scand J Rehabil Med 1987;19:25-30.
11. Murray MP, Kory RC, Clarkson SB. Walking patterns in healthy old men. J Gerontol 1969;24:164-78.

12. Olney SJ, Monga TN, Costigan PA. Mechanical energy of walking of stroke patients. Arch Phys Med Rehabil 1986;67:92-8.
13. Wall JC, Turnbull GI. Gait asymmetries in residual hemiplegia. Arch Phys Med Rehabil 1986;67:550-3.
14. Hill KD, Goldies PA, Baker PA. Retest reliability of the temporal and distance characteristics of hemiplegic gait using a foot switch system. Arch Phys Med Rehabil 1994;75:577-83.
15. Bohannon RW, Andrews AW, Smith MB. Rehabilitation goals of patients with hemiplegia. Int J Rehabil Res 1988;11:181-2.
16. Bohannon RW, Horton MG, Wikholm JB. Importance of 4 variables of gait to patients with stroke. Int J Rehabil Res 1991;14:246-50.
17. Bohannon RW. Gait performance of hemiparetic stroke patients: selected variables. Arch Phys Med Rehabil 1987;68:777-81.
18. Bohannon RW. Selected determinants of ambulatory capacity in patients with hemiplegia. Clin Rehabil 1980;3:47-53.
19. Bohannon RW. Knee extension force measurements are reliable and indicative of gait speed in stroke patients. Int J Rehabil Res 1989;12:193-4.
20. Bohannon RW, Andrews AW. Correlation of knee extensor muscle torque and spasticity with gait speed in patients with stroke. Arch Phys Med Rehabil 1990;71:330-3.
21. Bohannon RW, Walsh S. Nature, reliability, and predictive value of muscle performance in patients with hemiparesis following stroke. Arch Phys Med Rehabil 1992;73:721-5.
22. Nadeau S, Arsennault AB, Gravel D, Bourbonnais D. Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. Am J Phys Med Rehabil 1999;78:123-30.
23. Nakamura R, Hosokawa T, Tsuji I. Relationship of muscle strength for knee extension to gait capacity in patients with spastic hemiparesis. Tohoku J Exp Med 1985;145:335-40.
24. Nakamura R, Watanabe S, Handa T, Morohashi I. The relationship between walking speed and muscle strength for knee extension in hemiparetic stroke patients: a follow-up study. Tohoku J Exp Med 1988;154:111-3.

25. Suzuki K, Imada G, Iwaya T, Handa T, Kurogo H. Determinants and predictors of maximum walking speed during computer-assisted gait training in hemiparetic stroke patients. Arch Phys Med Rehabil 1999;80:179-82.
26. Suzuki K, Nakamura R, Yamada Y, Handa T. Determinants of maximum walking speed in hemiparetic stroke patients. Tohoku J Exp Med 1990;162:337-44.
27. Olney SJ, Griffin MP, Monga TN, McBride ID. Work and power in gait of stroke patients. Arch Phys Med Rehabil 1991;72:309-14.
28. De Quervain IA, Simon SR, Leurgans S, Pease WS, McAllister D. Gait pattern in the early recovery period after stroke. J Bone Joint Surg Am 1996;78:1506-14.
29. Keenan MA, Perry J, Jordan C. Factors affecting balance and ambulation following stroke. Clin Orthop 1984;Jan-Feb(182):165-71.
30. Friedman PJ. Spatial neglect in acute stroke: the line bisection test. Scand J Rehabil Med 1990;22:101-6.
31. Charteris J. Effects of velocity on upper to lower extremity muscular work and power output ratios of intercollegiate athletes. Br J Sports Med 1999;33:250-4.
32. Winter DA. Biomechanics and Motor Control of Human Movement (2nd ed.) New York: Wiley, 1990.
33. Benedetti M, Catani F, Leardini A, Pignotti E, Giannini S. Data management in gait analysis for clinical applications. Clin Biomech 1998;13:204-15.
34. Vachalathiti R. Three-dimensional kinematic and kinetic gait analysis in young thai adults, using the Motion Analysis™ system. Siriraj Hosp Gaz 1998;50:378-96.
35. Nilsson J, Thorstensson A. Ground reaction forces at different speeds of human walking and running. Acta Physiol Scand 1989;163:217-27.
36. Bowden MG, Balasubramanian CK, Neptune RR, Kautz SA. Anterior-posterior ground reaction forces as a measure of paretic leg contribution in hemiparetic walking. Stroke 2006;37:872-6.
37. Winstein CJ, Gardner ER, McNeal DR, Barto PS, Nicholson DE. Standing balance training: effect on balance and locomotion in hemiparetic adults. Arc Phys Med Rehabil 1989;70:755-62.

38. Schenkman M, Butler RB. A model for multisystem evaluation, interpretation, and treatment of individuals with neurologic dysfunction. Phys Ther 1989;69:538-47.

39. MacGregor J. The objective measurement of physical performance with long term ambulatory physiological surveillance equipment. In:Stott FD, Raftery EB, Goulding L, eds. Proceedings of the Third International Symposium on Ambulatory Monitoring. London: Academic Press; 1979:29-39.

40. Fedrickson E, Robert L, et al. Physiological cost index as a proxy measure for the oxygen cost of gait in stroke patients. [online]. Neurorehabil Neural Repair 2007. Available from: http://nnr.sagepub.com/cgi/content/abstract/21/5/429 [Access 2008 Nov 30].

41. Guyton AC, Hall JE. Textbook of Medical Physiology. Philadelphia: Elsevier and Saunders; 2006.

42. Bailey MJ, Ratcliffe CM. Reliability of Physiological Cost Index measurements in walking normal subjects using steady-state, non-steady state, and post exercise heart rate recording. Physiotherapy. 1995;81:618-23.

43. Hood VL, Granat MH, Maxwell DJ, Hasler JP. A new method of using heart rate to represent energy expenditure: the Total Heart Beat Index. Arch Phys Med Rehabil. 2002;83:1266-73.

44. Winchester PK, Carollo JJ, Parekh RN, Lutz LM, Aston JW Jr. A comparison of paraplegia gait performance using two types of reciprocating gait orthoses. Prosther Orthot Int. 1993;17:101-6.

45. Winchester PK, Carollo JJ, Habasevich R. Physiologic cost of reciprocal gait in FES assisted walking. Paraplegia. 1994;32:680-6.

46. Nene AV. Physiological cost index of walking in able-bodied adolescents and adults. Clin Rehabil. 1993;7:319-26.

47. Mossberg KA. Reliability of a timed walk test in persons with acquired brain injury. Am Phys Med Rehabil. 2003;82:385-90.

48. Bohannon RW. Recovery and correlates of trunk muscle strength after stroke. Int J Rehabil Res 1995;18:162-67.

49. Giuliani C. Strength training for patients with neurological disorders. Neurol Report 1995;19:29-34.

50. Andrews AW, Bohannon RW. Distribution of muscle strength impairments following stroke. Clin Rehab 2000;14:79-87.
51. Adams RW, Gandevis SC, Skuse NF. The distribution of muscle weakness in upper motorneuron lesions affecting the lower limb. Brain 1990;113:1459-76.
52. Bohannon RW. Strength of lower limb related to gait velocity and cadence in stroke patients. Physiother Can 1986;38:204-6.
53. Bohannon RW. Determinants of transfer capacity in patients with hemiparesis. Physiother Can 1988;40:236-9.
54. Kim CM, Eng JJ. Symmetry in vertical ground reaction force is accompanied by symmetry in temporal but not distance variables of gait in persons with stroke. Gait Posture 2003;18:23-8.
55. Hsu AL, Tang PF, Jan MH. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. Arch Phys Med Rehabil 2003;84:1185-93.
56. Kraemer W, Adams K, Cafarelli E, et al. Progression models in resistance training for healthy adults. Med Sci Sports Exerc 2002;34:364-80.
57. Bobath B. Adult hemiplegia: evaluation and treatment, third edition. Toronto: Butterworth-Heinemann , 1990.
58. Carr J, Shepherd R, Ada L. Spasticity: research findings and implications for intervention. Physiotherapy 1995;81:421-9.
59. Boyd R, Ada L. Physiotherapy management of spasticity. Cambridge: Cambridge University Press, 2001.
60. Ada L, O'Dwyer N, Green J, Yeo W, Neilson P. The nature of the lost strength and dexterity in the upper limb following stroke. Hum Mov Sci 1996;15:671-87.
61. Roth EJ, Harvey RL. Rehabilitation of stroke syndromes. Physical Medicine and Rehabilitation, second edition 2000;50:1117-63.
62. Marshall J. Types of hemiplegia. Physiotherapy 1974;60:334-6.
63. McGill HC. The pathogenesis of atherosclerosis. Clin Chem 1988;34:B33-B39.
64. Pessin MS, Duncan GW, Mohr JP, et al. Clinical and angiographic features of carotid transient ischemic attacks. N Emgl J Med 1977;296:358-62.

65. Bogousslavsky J, Regli F. Borderzone infarctions distal to internal carotid artery. Ann Neurol 1986;20:346-50.
66. Cerebral Embolism Task Force. Cardiogenic brain embolism: The second report of the Cerebral Embolism Task Force. Arch Neurol 1989;46:727-43.
67. Caplan LR. Brain embolism, revisited. Neurology 1993;43:1281-7.
68. Jones HR, Caplan LR, Come PC, et al. Cerebral emboli of paradoxical origin. Ann Neurol 1983;13:314-9.
69. Mohr JP. Lacunes. Stroke 1982;13:3-11.
70. Furlan AJ, Whisnant JP, Elveback LR. The decreasing incidence of primary intracerebral hemorrhage: A population study. Ann Neurol 1979;5:367-73.
71. Fisher CM. Pathological observations in hypertensive cerebral hemorrhage. J Neuropathol Exp Neurol 1971;30:536-50.
72. Feldmann E, Tornabene J. Diagnosis and treatment of cerebral amyloid angiopathy. Clin Geriatr Med 1991;7:617-30.
73. Sundt TM, Whisnant P. Subarachnoid hemorrhage from intracranial aneurysms. N Engl Med 1987;299:116-22.
74. Winn HR, Richardson AE, Jane JA. The long-term prognosis in untreated cerebral aneurysms: I. The incidence of late hemorrhage in cerebral aneurysm: A 10-year evolution of 364 patients. Ann Neurol 1977;1:358-70.
75. Perret G, Nishioka H. Report on the Cooperative Study of Intra cranial Aneurysms and Subarachnoid Hemorrhage: VI. Arteriovenous malformations: An analysis of 545 cases of craniocerebral arteriovenous malformations and fistulae reported to the cooperative study. J Neurosurg 1966;25:467-90.
76. Mohr JP, Hilal SK, Stein BM. Arteriovenous malformations and other vascular anomalies. In Barnett HJM, Mohr JP, Stein BM, et al (eds): Stroke: Pathophysiology, Diagnosis, and Management, ed 2. New York, Churchill Livingstone, 1992.
77. Ondra SL, Troupp H, George ED, et al. The natural history of symptomatic arteriovenous malformations of the brain: A 24-year follow-up assessment. J Neurosurg 1990;73:387-91.

78. Huckman MS. Normal pressure hydrocephalus: Evaluation of diagnostic and prognostic tests. Am J Neurol Res 1981;2:385-95.
79. Collins JJ, Whittle MW. Impulsive forces during walking and their clinical implication. Clinical Biomechanics 1989;4:179-87.
80. Murray MP. Gait as a total pattern of movement. American Journal of Physical Medcine. 1967;46:290-333.
81. Perry J. Kinesiology of lower extremity bracing. Clinical Orthopaedics and Related Research 1974;102:18-31.
82. Inman VT, Ralston HJ, Todd F. Human walking. Baltimore: Williams and Wilkins. 1981.
83. Dietz V, Berger W. Interlimb coordination of posture in patients with spastic hemiparesis. Brain 1984;107:965-78.
84. Tang A, Rymer WZ. Abnormal force-EMG relations in paretic of hemiparetic human subjects. J Neurol Neurosurg Psychiatry 1981;44:690-8.
85. Bourbonnais D, Vander Noven S. Weakness in patients with hemiparesis. Am J Occup Ther 1989;43:313-9.
86. McComas AJ, Sica REP, Upton ARM, Aguilera N. Functional changes in motorneurons of hemiparetic patients. J Neurol Neurosurg Psychiatry 1973;36:183-93.
87. Bohannon RW, Larkin PA, Smith MB. Relationship between static muscle strength deficits and spasticity in stroke patients with hemiparesis. Phys Ther 1987;67:1068-71.
88. Conrad B, Benecke R, Meick HM. Gait disturbances in paraspastic patients. In: Delwaide PJ and Young RR, eds. Clinical Neurophysiology in Spasticity. Amsterdam: Elsevier. 1985:155-74.
89. Winter DA. The Biomechanics and Motor Control of Human Gait. Waterloo, Ontario: University of Waterloo Press. 1987.
90. Waters RL, Garland DE, Perry J, Habig T, Slabaugh P. Stiff-legged gait in hemiplegia: surgical correction. Journal of Bone and Joint Surgery 1979;61A: 927-33.

91. Burdett RG, Borello-France D, Blatchly C. Gait comparison of subjects with hemiplegia walking unbrace, with ankle-foot orthosis, and with air-stirrup brace. Phys Ther 1988;68:1197-1203.
92. Von Schroeder HP, Coutts RD, Lyden PD, et al. Gait parameters following stroke: practical assessment. J Rehabil Res Dev 1995;32:25-31.
93. Nakamura R, Handa T, Watanabe S, Morohashi T, Walking cycle after stroke. Tohoku J Exp Med 1988;154:241-4.
94. Lehmann JF, Condon SM, Price R, deLateur BJ. Gait abnormalities in hemiplegia: their correction by ankle-foot orthoses. Arch Phys Med Rehabil 1987;68:763-71.
95. Hu MS. Pelvic drop in hemiplegic locomotion: a kinematic and EMG study. Master's thesis University of North Carolina, Chapel Hill, 1988.
96. Hesse S, Luecke D, Jahnke MT, Mauritz KH. Gait function in spastic hemiparetic patients walking barefoot, with firm shoes, and with ankle-foot orthoses. Int J Rehabil Res 1996;19:133-41.
97. Hannah RE, Morrison JB, Chapman AE. Kinematic symmetry of lower limbs. Arch Phys Med Rehabil 1984;65:155-8.
98. Hirschberg GG, Nathanson M. Electromyographic recording of muscular activity in normal and spastic gaits. Arch Phys Med Rehabil 1952;33:217-25.
99. Peat M, Dubo HIC, Winter DA, Quanbury AO, Steinke T, Grahame R. Electromyographic temporal analysis of gait: hemiplegic locomotion. Arch Phys Med Rehabil 1976;57;421-5.
100. Pinzur MS, Sherman R, Dimonte-Levine P, Trimble J. Gait changes in adult onset hemiplegia. Am J Phys Med 1987;66:228-37.
101. Shiavi R, Bugle HJ, Limbird T, Electromyographic gait assessment, part2: Preliminary assessment of hemiparetic synergy patterns. J Rehabil Res Dev 1987;24:24-30.
102. Wortis SB, Marks M, Hirchberg GG, Nathanson M. Gait analysis in hemiplegia. Trans Am Neurol Assoc 1951;76:181-3.
103. Carlsoo S, Dahllof AG, Holm J. Kinetic analysis of gait in patients with hemiparesis and in patients with intermittent claudication. Scan J Rehabil Med 1974;6:166.

104. Bogataj U, Gros N, Malezic M, Kelih B, Kljajic M, Acimovic R. Restoration of gait during two or three weeks of therapy with multichannel electrical stimulation. Phys Ther 1989;69:319-27.

105. Malezic M, Kljajic M, Acimovic-Janezic R, Gros N, Krajnik J, Stanic U. Therapeutic effects of multisite electric stimulation of gait in motor-disabled patients. Arch Phys Med Rehabil. 1987;68:553-60.

106. Rogers M, Hedman LD, Pai YC. Kinetic analysis of dynamic transitions in stance support accompanying voluntary leg flexion movements in hemiparetic adults. Arch Phys Med Rehabil 1993;74:19-25.

107. Lowery LL. Mechanical energy analysis of hemiplegic gait: monitoring and diagnosis. Proc Can Soc Biomech 1980;56-57.

108. Winter DA. Energy assessments in pathological gait. Physiother Can 1978;30:183-91.

109. Iida H, Yamamuro T. Kinetic analysis of the center of gravity of the human body in normal and pathological gaits. J Biomech 1987;20:987-995.

110. Hesse SA, Jahnke MT, Schreiner C, Mauritz KH. Gait symmetry and functional walking performance in hemiparetic patients prior to and after a 4-week rehabilitation program. Gait Posture 1993;1:166-71

111. Dewar ME, Judge G. Temporal asymmetry as a gait quality indicator. Med Biol Eng Comput 1980;18:689-93.

112. Griffin MP, Olney SJ, McBride ID. The role of symmetry in gait performance of stroke subjects with hemiplegia. Gait Posture 1995;3:132-42.

113. Perry J. the mechanics of walking in hemiplegia. Clin Orthop 1969;63:23.

114. Olney SJ, Richard C. Hemiparetic gait following stroke. Part I: characteristics. Gait and Posture 1996:136-48.

115. Takebe K, Basmajian JV. Gait analysis in stroke patients to asses treatments of foot-drop. Arch Phys Med Rehabil 1976;57:305-10.

116. Peat M, Dubo HIC, Winter DA, Quanbury AO, Steinke T, Grahame R. Electromyographic temporal analysis of gait: hemiplegic locomotion. Arch Phys Med Rehabil 1976;57;421-5.

117. Ng SM, Shepherd RB. Weakness in patients with stroke: Implications for strength training in neurorehabilitation. Phys Ther Rev2000;5:227–38.

118. Patten C, Lexell J, Brown HE. Weakness and strength training in persons with post-stroke hemiplegia: rationale, method and efficacy. J Rehabil Res Dev 2004;41:293–312.

119. Von Schroeder HP, Coutts RD, Lyden PD, et al. Gait parameters following stroke: practical assessment. J Rehabil Res Dev 1995;32:25-31.

120. Flansbjer UB, Downham D, Lexell J. Knee muscle strength, gait performance, and perceived participation after stroke. Arch PhysMed Rehabil 2006;87:974–80.

121. Richards CL, Malouin F, Dean C. Gait in stroke: assessment and rehabilitation. Clin Geriatr Med 1999;15:833–55.

122. Parker CJ, Gladman JR, Drummond AE. The role of leisure in stroke rehabilitation. Disabil Rehabil 1997;19:1–5.

123. Pollock A, Baer G, Pomeroy VM, Langhorne P. Physiotherapy treatment approaches for the recovery of postural control and lower limb function following stroke. The Cochrane Database of Systematic Reviews Library 1999: Art. No.: CD003316.

124. Kraemer WJ, Ratamess NA. Fundamentals of resistance training: progression and exercise prescription. Med Sci Sports Exerc 2004;36:674–88.

125. Andersen LL , Magnusson SP, Nielsen M, Haleem J, Poulsen K, Aagaard P. Neuromuscular activation in conventional therapeutic exercises and heavy resistance exercises: implications for rehabilitation. Phys Ther 2006;86: 683–97.

126. Morris SL, Dodd KJ, Morris ME. Outcomes of progressive resistance strength training following stroke: a systematic review. Clin Rehabil 2004;18:27–39.

127. Patten C, Lexell J, Brown HE. Weakness and strength training in persons with post-stroke hemiplegia: rationale, method and efficacy. J Rehabil Res Dev 2004;41:293–312.

128. Gordon NF, Gulanick M, Costa F, Fletcher G, Franklin BA, Roth EJ, et al. Physical activity and exercise recommendations for stroke survivors: an American Heart Association scientific statement from the Council on Clinical Cardiology, Subcommittee on Exercise, Cardiac Rehabilitation,

and Prevention; the Council on Cardiovascular Nursing; the Council on Nutrition, Physical Activity, and Metabolism; and the Stroke Council. Circulation 2004;109:2031–41.

129. National guidelines for stroke, second edition. London: Royal Colleges of Physicians of London;2004.

130. DeLorme TL, Watkins AL. Techniques of progressive resistance exercise. Arch Phys Med. 1948;29:263–73.

131. American College of Sports Medicine. Progression models in resistance training for healthy adults. Med Sci Sports Exerc. 2002;34:364–80.

132. Flansbjer UB, Miller M, Downham D, Lexell J. Progressive resistance training after stroke: Effects on muscle strength, muscle tone, gait performance, and perceived participation. J Rehabil Med 2008;40:42–8.

133. Quellette MM, LeBrasseur NK, Bean JF, Phillips E, Stein J, Frontera WR, Fielding RA. High-Intensity Resistance Training Improves Muscle Strength, Self-Reported Function, and Disability in Long-Term Stroke Survivors. Stroke 2004;35:1404-9.

134. Weiss A, Suzuki T, Bean J, Fielding RA. High intensity strength training improves strength and functional performance after stroke. Am J Phys Med Rehabil. 2000;79(4):369-76.

135. Michael KM, Allen JK, Macko RF. Reduced ambulatory activity after stroke: the role of balance, gait, and cardiovascular fitness. Arch Phys Med Rehabil 2005;86:1552-6.

136. Platts MM, Rafferty D, Paul L. Metabolic cost of overground gait in younger stroke patients and healthy controls. Med Sci Sports Exerc. 2006;38:1041–6.

137. Macko RF, Smith GV, Dobrovolny CL, Sorkin JD, Goldberg AP, Silver KH. Treadmill training improves fitness reserve in chronic stroke patients. Arch Phys Med Rehabil 2001;82:879-84.

138. Fredrickson E, Ruff RL, Daly JJ. Physiological cost index as a proxy measure for the oxygen cost of gait in stroke patients. Neurorehabil Neural Repair. 2007;21:429-34.

139.Collen FM. The measurement of standing balance after stroke. Physiotherapy Theory & Practice 1995;11:109-18.

140. Horak FB. Clinical assessment of balance disorders. Gait & Posture 1997;6:76-84.

141. Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: new insights for treatment of balance disorders. Phys Ther 1997;77:517-33.

142.Juneja G, Czyrny JJ, Linn RT. Admission balance and outcomes of patients admitted for acute inpatient rehabilitation. Am J Phys Med Rehabil 1998;77:388-93.

143. Hill K, Ellis P, Bernhardt J, Maggs P, Hull S. Balance and mobility outcomes for stroke patients: a comprehensive audit. Aust J Physiother 1997;43:173-80.

144. Laufer Y. Effects of one-point and four-point canes on balance and weight distribution in patients with hemiparesis. Clin Rehabil 2002;16:141-8.

145. Bohannon R, Walsh S, Joseph M. Ordinal and timed balance measurements: reliability and validity in patients with stroke. Clin Rehabil 1993;7:9-13.

146.Sackley CM. Falls, sway, and symmetry of weight-bearing after stroke. Int Disabil Stud 1991;13:1-4.

147.Shumway-Cook A, Anson D, Haller S. Postural sway biofeedback: its effect on reestablishing stance stability in hemiplegic patients. Arch Phys Med Rehabil 1988;69:395-400.

148. Rode G, Tiliket C, Boisson D. Predominance of postural imbalance in left hemiparetic patients. Scand J Rehabil Med 1997;29:11-6.

149. Sackley CM. The relationship between weight-bearing asymmetry after stroke, motor function and activities of daily living. Physiotherapy Theory & Practice 1990;6:179-85.

150.Ring C, Nayak US, Isaacs B. Balance function in elderly people who have and who have not fallen. Arch Phys Med Rehabil 1988;69:261-4.

151.Forster A, Young J. Incidence and consequences of falls due to stroke: a systematic inquiry. BMJ 1995;311:83-6.

152. Hyndman D, Ashburn A, Stack E. Fall events among people with stroke living in the community: circumstances of falls and characteristics of fallers. Arch Phys Med Rehabil 2002;83:165-70.

153. Shumway-Cook A, Woollacott MH. Motor control: theory and practical applications. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2001.

154. Eng JJ, Kim CM, MacIntyre DL. Reliability of lower extremity strength measures in persons with chronic stroke. Arch Phys Med Rehabil 2002;83:322-8.

155. Adams RW, Gandevia SC, Skuse NF. The distribution of muscle weakness in upper motoneuron lesions affecting the lower limb. Brain 1990;113:1459-76.

156. Davidoff RA. The pyramidal tract. Neurology 1990;40:332-9.

157. Lord SR, Menz HB. Visual contributions to postural stability in older adults. Gerontology 2000;46:306-10.

158. Niam S, Cheung W, Sullivan PE, et al. Balance and physical impairments after stroke. Arch Phys Med Rehabil 1999;80:1227-33.

159. Catz A, Ron S, Solzi P, Korczyn AD. The vestibulo-ocular reflex and dysequilibrium after hemispheric stroke. Am J Phys Med Rehabil 1994;73:36-9.

160. Bonan IV, Colle FM, Guichard JP, et al. Reliance on visual information after stroke. Part I: balance on dynamic posturography. Arch Phys Med Rehabil 2004;85:268-73.

161. Marigold DS, Eng JJ, Tokuno CD, Donnelly CA. Contribution of muscle strength and integration of afferent input to postural instability in persons with stroke. Neurorehabil Neural Repair 2004;18:222–9.

162. Laufer Y, Sivan D, Schwarzmann R, Sprecher E. Standing balance and functional recovery of patients with right an left hemiparesis in the early stages of rehabilitation. Neurorehabil Neural Repair 2003;17:207-13.

163. Brzycki, Matt. A Practical Approach to Strength Training. McGraw-Hill. 1998.

164. Sharp SA, Brouwer BJ. Isokinetic strength training at the hemiparetic knee: effects on function and spasticity. Arch Phys Med Rehabil. 1997;78:1231–36.

165. Nugent J, Schurr K, Adams R. A dose response relationship between amount of weight-bearing exercise and walking outcome following cerebrovascular accident. Arch Phys Med Rehabil. 1994;75:399–402.

166. Inaba M, Edberg E, Montgomery J, Gillis M. Effectiveness of functional training, active exercise, and resistance exercise for patients with hemiplegia. Phys Ther. 1970;53:28–35.

167. Duncan P, Richards L, Wallace D, Stoker-Yates J, Pohl P, Luchies C, Ogle A, Studenski S. A randomized, controlled pilot study of a home-based exercise program for individuals with mild and moderate stroke. Stroke. 1998;29:2055–60.

168. Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B. Muscle strengthening and physical conditioning to reduce impairment in chronic stroke survivors. Arch Phys Med Rehabil. 1999;80:1211–18.

169. Sommerfeld DK , Eek EU , Svensson AK, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. Stroke 2004; 35:134–39.

170. Badics E, Wittmann A, Rupp M, Stabauer B, Zifko UA. Systematic muscle building exercises in the rehabilitation of stroke patients. NeuroRehabilitation 2002; 17: 211–14.

171. Bourbonnais D, Bilodeau S, Lepage Y, Beaudoin N, Gravel D, Forget R. Effect of force-feedback treatments in patients with chronic motor deficits after a stroke. Am J Phys Med Rehabil 2002; 81: 890–97.

172. Masani K, Motoki K, and Tetsuo F. Variability of ground reaction forces during treadmill walking. J Appl Physiol 2002 92: 1885–90.

173. Detrembleur C, Dierick F, Stoquart G, Chantraine F, Lejeune T. Energy cost, mechanical work, and efficiency of hemiparetic walking. Gait and Posture 2003; 18: 47-55.

174. Bernardi M, Macaluso A, Sproviero E, Coratella D, Felici F, Rodio A, et al. Cost of walking and locomotor impairment. J Electromyogr Kinesiol 1999;9:149 -57.

175. Dettmann MA, Linder MT, and Sepic S.B. Relationships among walking performance, postural stability, and functional assessments of the hemiplegic patients. Am. J. Phys. Med 1987; 66:77-90.

176. Hamrin E, Elkund G, Hillgren AK, Borges 0, Hall J, and Hellstrom 0. Muscle strength and balance in poststroke patients. Upsala J. Med. Sci 1982;87:11-26.

177. Jonsdottir J, Recalcati M, Rabuffetti M, Casiraghi A, Boccardi S, and Ferrarin M. Functional resources to increase gait speed in people with stroke: Strategies adopted compared to healthy controls. Gait and Posture 2009;29: 355–59.

178. Teixeira-Salmela LF, Nadeau S, Mcbride I, Olney SJ. Effects of muscle strengthening and physical conditioning training on temporal, kinematic and kinetic variables during gait in chronic stroke survivors. J Rehabil Med 2001;33:53–60.

179. Kim M, Eng JJ. Magnitude and pattern of 3D kinematic and kinetic gait profiles in persons with stroke: relationship to walking speed. Gait and Posture 2004;20: 140–6.

180. Parvatanemi K, Olney SJ, Brouwer B. Changes in muscle group work associated with changes in gait speed of persons with stroke. Clin Biomech 2007;22:813–20.

APPENDICES

APPENDIX A

Human subject approval documents from Mahidol University

COA. No. MU-IRB 2010/246.2308

Documentary Proof of Mahidol University Institutional Review Board

Title of Project: Effects of Progressive Resistance Strength Training on Hemiparetic Gait (Thesis for Master Degree)

Principal Investigator: Mr. Ratakorn Aiimkosa

Name of Institution: College of Sports Science and Technology

Approval includes: Annual Report version received date 20 August 2010

Mahidol University Institutional Review Board is in full compliance with International Guidelines for Human Research Protection such as Declaration of Helsinki, The Belmont Report, CIOMS Guidelines and the International Conference on Harmonization in Good Clinical Practice (ICH-GCP)

Date of Renewal (1st): 23 August 2010

Date of Expiration: 22 August 2011

Signature of Chairman: .....
(Professor Shusee Visalyaputra)

Signature of Head of the Institute: .....
(Associate Professor Sansanee Chaiyaroj)
Vice President for Research and Academic Affairs

Office of the President, Mahidol University, 999 Phuttamonthon 4 Rd., Salaya, Phuttamonthon District, Nakhon Pathom 73170. Tel. (662) 8496223-5 Fax. (662) 8496223

APPENDIX B
Consent Form

วันที่..... เดือน..... พ.ศ.....

ข้าพเจ้า.....อายุ.....ปี อาศัยอยู่บ้านเลขที่..... .....ถนน .....ตำบล.....อำเภอ..... ..... จังหวัด .....รหัสไปรษณีย์.....โทรศัพท์ .....

ขอแสดงเจตนายินยอมเข้าร่วมโครงการวิจัย เรื่องผลของการฝึกความแข็งแรงโดยการเพิ่มแรงต้านแบบก้าวหน้าต่อลักษณะท่าทางการเดินในผู้ป่วยอัมพฤกษ์ครึ่งซีก

โดยข้าพเจ้าได้รับทราบรายละเอียดเกี่ยวกับที่มาและจุดมุ่งหมายในการทำวิจัยรายละเอียดขั้นตอนต่างๆ ที่จะต้องปฏิบัติหรือได้รับการปฏิบัติ ประโยชน์ที่คาดว่าจะได้รับของการวิจัยและความเสี่ยงที่อาจจะเกิดขึ้นจากการเข้าร่วมการวิจัย รวมทั้งแนวทางป้องกันและแก้ไขหากเกิดอันตรายขึ้น ค่าตอบแทนที่จะได้รับค่าใช้จ่ายที่ข้าพเจ้าจะต้องรับผิดชอบจ่ายเอง โดยได้อ่านข้อความที่มีรายละเอียดอยู่ในเอกสารชี้แจงผู้เข้าร่วมการวิจัยโดยตลอด อีกทั้งยังได้รับคำอธิบายและตอบข้อสงสัยจากหัวหน้าโครงการวิจัยเป็นที่เรียบร้อยแล้ว

ข้าพเจ้าจึงสมัครใจเข้าร่วมในโครงการวิจัยนี้

หากข้าพเจ้ามีข้อข้องใจเกี่ยวกับขั้นตอนของการวิจัย หรือหากเกิดเหตุการณ์ที่ไม่พึงประสงค์จากการวิจัยขึ้นกับข้าพเจ้า ข้าพเจ้าจะสามารถติดต่อกับนายรฐกร เอมโกษา โทรศัพท์ 08-9776-4515 ได้ตลอด 24 ชั่วโมง

หากข้าพเจ้าได้รับการปฏิบัติไม่ตรงตามที่ระบุไว้ในเอกสารชี้แจงผู้เข้าร่วมการวิจัย ข้าพเจ้าสามารถติดต่อกับประธานคณะกรรมการจริยธรรมการวิจัยในคนหรือผู้แทน ได้ที่สำนักงานคณะกรรมการจริยธรรมการวิจัยในคน สำนักงานอธิการบดี มหาวิทยาลัยมหิดล ถนนพุทธมณฑล สาย 4 ตำบลศาลายา อำเภอพุทธมณฑล จังหวัดนครปฐม 73170 หมายเลขโทรศัพท์ 02-849-6241-6, 02-849-6066 โทรสาร 02-849-6247

ข้าพเจ้าได้ทราบถึงสิทธิ์ที่ข้าพเจ้าจะได้รับข้อมูลเพิ่มเติมทั้งทางด้านประโยชน์และโทษจากการเข้าร่วมการวิจัย และสามารถถอนตัวหรือองดเข้าร่วมการวิจัยได้ทุกเมื่อ โดยจะไม่มีผลกระทบต่อการบริการและการรักษาพยาบาลที่ข้าพเจ้าจะได้รับต่อไปในอนาคต และยินยอมให้ผู้วิจัยใช้ข้อมูลส่วนตัวของข้าพเจ้าที่ได้รับจากการวิจัย แต่จะไม่เผยแพร่ต่อสาธารณะเป็นรายบุคคล โดยจะนำเสนอเป็นข้อมูลโดยรวมจากการวิจัยเท่านั้น

ข้าพเจ้าเข้าใจข้อความในเอกสารชี้แจงผู้เข้าร่วมการวิจัย และหนังสือแสดงเจตนายินยอมนี้โดยตลอดแล้ว จึงลงลายมือชื่อไว้

ลงชื่อ.....ผู้เข้าร่วมการวิจัย/ผู้แทนโดยชอบธรรม/ วันที่.....

(.....)

ลงชื่อ.....ผู้ให้ข้อมูลและขอความยินยอม/หัวหน้าโครงการวิจัย/ วันที่.....

(.....)

ในกรณีผู้เข้าร่วมการวิจัยไม่สามารถอ่านหนังสือได้ผู้ที่อ่านข้อความทั้งหมดแทนผู้เข้าร่วมการวิจัยคือ ..... จึงได้ลงลายมือชื่อไว้เป็นพยาน

ลงชื่อ..... พยาน/ วันที่.....

(.....)

APPENDIX C

Physical examination form

รหัสผู้เข้าร่วมวิจัย_____

วัน/เดือน/ปี _____ ครั้งที่ 1

วัน/เดือน/ปี _____ ครั้งที่ 2

น้ำหนัก _____/_____กิโลกรัม ส่วนสูง _____/_____ เซนติเมตร

ชีพจรขณะพัก _____/_____ครั้ง/นาที ความดันโลหิต _____/_____ มิลลิเมตรปรอท

ข้างที่ถนัด _____

Date of admission _____ Date of discharge _____

Diagnosis_____

_____

☐ Infarction _____

☐ Haemorrhage _____

ข้างที่อ่อนแรง ☐ ซ้าย ☐ ขวา

ยาที่ใช้ _____

เข้าร่วมโครงการวิจัยในกลุ่ม _____

Physical examination

1. Muscle tone (test in supine)

		Pre-training		**Post-training**	
		Affected	Non-affected	Affected	Non-affected
Hip	Flexor				
	Extensor				
	Abductor				
	Adductor				
	External rotate				
	Internal rotate				
Knee	Flexor				
	Extensor				
Ankle	Dorsiflexor				
	Plantarflexor				
	Invertor				
	Evertor				

2. Passive range of motion (degrees)

		Pre-training		**Post-training**	
		Affected	Non-affected	Affected	Non-affected
Hip	Flexion/extension				
	Abduction/adduction				
	External/internal rotate				
Knee	Flexion/extension				
Ankle	Dorsiflexion/plantarflexion				

3. Proprioceptive sense

	Pre-training			**Post-training**		
	Normal	**Impair**	**Loss**	**Normal**	**Impair**	**Loss**
Hip						
Knee						
Ankle						

4. Barthel index of activities of daily living

กิจกรรม/คะแนน	**ก่อนฝึก**	**หลังฝึก**
1. Feeding (การรับประทานอาหารเมื่อเตรียมสำรับไว้ให้เรียบร้อยต่อหน้า)		
0 = ไม่สามารถตักอาหารเข้าปากได้ ต้องมีคนป้อนให้		
5 = ช่วยใช้ช้อนตักอาหารไว้ให้หรือตัดให้เป็นชิ้นเล็กๆ ไว้ล่วงหน้า		
10 = ตักอาหารและช่วยตัวเองได้ปกติ		
2. Transfer (ลุกนั่งจากที่นอน หรือจากเตียงไปยังเก้าอี้)		
0 = ไม่สามารถนั่งได้ (นั่งแล้วจะล้มเสมอ) หรือต้องใช้คนสองคนช่วยกันยกขึ้น		
5 = ต้องการความช่วยเหลืออย่างมากจึงจะนั่งได้ เช่น ต้องใช้คนที่แข็งแรงหรือมีทักษะ 1 คน หรือใช้คนทั่วไป 2 คนพยุง หรือดันขึ้นมาจึงจะนั่งอยู่ได้		
10 = ต้องการความช่วยเหลือบ้าง เช่น บอกให้ทำตาม หรือช่วยพยุงเล็กน้อย หรือต้องมีคนดูแลความปลอดภัย		
3. Grooming (ล้างหน้า หวีผม แปรงฟันโกนหนวด ในระยะ 24-48 ชั่วโมงที่ผ่านมา)		
0 = ต้องการความช่วยเหลือ		
5 = ทำได้เอง (รวมทั้งที่ทำได้เองถ้าเตรียมอุปกรณ์ไว้ให้)		

กิจกรรม/คะแนน	ก่อนฝึก	หลังฝึก
4. Toilet Use (การเข้าห้องน้ำ)		
0 = ช่วยเหลือตัวเองไม่ได้		
5 = ทำเองได้บ้าง (อย่างน้อยทำความสะอาดตัวเองได้หลังเสร็จธุระ) แต่ต้องการความช่วยเหลือในบางสิ่ง		
10 = ช่วยเหลือตัวเองได้ดี (ขึ้นนั่งและลงจากโถส้วมได้เอง ทำความสะอาดได้เรียบร้อยหลังเสร็จธุระแล้ว ถอดใส่เสื้อผ้าได้เรียบร้อย)		
(การอาบน้ำ)		
0 = ต้องมีคนช่วย หรือทำให้		
5 = อาบน้ำได้เอง		
6. Mobility (การเคลื่อนที่ภายในห้องหรือบ้าน)		
0 = เคลื่อนที่ไปไหนไม่ได้		
5 = ต้องใช้รถเข็นช่วยตัวเองให้เคลื่อนที่ได้เอง (ไม่ต้องมีคนเข็นให้) และจะต้องเข้าออกมุมหรือประตูได้		
10 = เดินหรือเคลื่อนที่โดยมีคนช่วย เช่น พยุง หรือบอกให้ทำตาม หรือต้องให้ความสนใจดูแลเพื่อความปลอดภัย		
7. Stairs (การขึ้นลงบันได 1 ขั้น)		
0 = ไม่สามารถทำได้		
5 = ต้องการคนช่วยเหลือ		
10 = ขึ้นลงได้เอง (ถ้าต้องใช้อุปกรณ์ช่วยเดิน เช่น walker จะต้องเอาขึ้น-ลงได้ด้วย)		
8. Dressing (การสวมใส่เสื้อผ้า)		
0 = ต้องมีคนสวมใส่ให้ ช่วยตัวเองไม่ได้เลยหรือได้น้อย		
5 = ช่วยตัวเองได้ราวร้อยละ 50 ที่เหลือต้องมีคนช่วย		
10 = ช่วยตัวเองได้ดี (รวมทั้งการติดกระดุม รูดซิบ หรือสวมใส่เสื้อผ้าที่ดัดแปลงที่เหมาะสมก็ได้)		
9. Bowels (การกลั้นอุจจาระในระยะ 1 สัปดาห์ที่ผ่านมา)		
0 = กลั้นไม่ได้ หรืออาจต้องการสวนอุจจาระอยู่เสมอ		
5 = กลั้นไม่ได้เป็นบางครั้ง (ไม่เกิน 1 ครั้งต่อสัปดาห์)		
10 = กลั้นได้ปกติ		
10. Bladder (การกลั้นปัสสาวะในระยะ 1 สัปดาห์ที่ผ่านมา)		
0 = กลั้นไม่ได้ หรือใส่สายสวนปัสสาวะและไม่สามารถดูแลเองได้		
5 = กลั้นไม่ได้เป็นบางครั้ง (ไม่เกินวันละ 1 ครั้ง)		
10 = กลั้นได้ปกติ		

5. Postural Assessment Scale for Stroke patients (PASS)

PASS	Assessment	
	ก่อนฝึก	หลังฝึก
Maintaining a Posture		
1. Sitting without support		
2. Standing with support		
3. Standing without support		
4. Standing on nonparetic leg		
5. Standing on paretic leg		
Changing Posture		
6. Supine to affected side lateral		
7. Supine to non-affected side lateral		
8. Supine to sitting up on the edge of table		
9. Sitting to the edge of table to supine		
10. Sitting to standing up		
11. Standing up to sitting down		
12. Standing picking up a pencil from the floor		
Total Scores		

6. Functional Ambulation Classification (FAC)

FAC Scores	Assessment	
	ก่อนฝึก	หลังฝึก
0		
1		
2		
3		
4		
5		

APPENDIX D

Interpretation

Muscle tone scale (Modified Asworth Scale)

0 : no increase in tone

1 : slight increase, catch and release or minimal resistance at the end of range of motion

1+ : slight increase, catch, and minimal resistance

2 : more marked increase through most of range, parts easily moved

3 : considerable increase, passive movement difficult

4 : parts rigid in flexion or extension

Barthel index of activities of daily living scores

0-20 = very severely disabled

25-45 = severely disabled

50-70 = moderately disabled

75-90 = mildly disabled

Postural Assessment Scale for Stroke patients (PASS)

Maintaining a Posture

1. Sitting without support (sitting on the edge of an 50-cm-high examination table with the feet touching the floor)

0 = cannot sit

1 = can sit with slight support, for example, by 1 hand

2 = can sit more than 10 seconds without support

3 = can sit for 5 minutes without support

2. Standing with support (feet position free, no other constraints)

0 = cannot stand, even with support

1 = can stand with strong support of 2 people

2 = can stand with moderate support of 1 people

3 = can stand with support of only 1 hand

3. Standing without support (feet position free, no other constraints)

0 = cannot without support

1 = can stand without support for 10 seconds or leans heavily on 1 leg

2 = can stand without support for 1 minute or stand slightly asymmetrically

3 = can stand without support for more than 1 minute and at the same time perform arm movement above the shoulder level

4. Standing on nonparetic leg (no other constraints)

0 = cannot stand on nonparetic leg

1 = can stand on nonparetic leg for a few seconds

2 = can stand on nonparetic leg for more than 5 seconds

3 = can stand on nonparetic leg for more than 10 seconds

5. Standing on paretic leg (no other constraints)

0 = cannot stand on paretic leg

1 = can stand on paretic leg for a few seconds

2 = can stand on paretic leg for more than 5 seconds

3 = can stand on paretic leg for more than 10 seconds

Changing Posture

Scoring of items 6 to 12 is as follow (items 6 to 11 are to be performed with a 50-cm-high examination table, items 10 to 12 are to be performed without any support; no other constraints):

0 = cannot perform the activity

1 = can perform the activity with much help

2 = can perform the activity with little help

3 = can perform the activity without help

6. Supine to affected side lateral

7. Supine to non-affected side lateral

8. Supine to sitting up on the edge of table

9. Sitting to the edge of table to supine

10. Sitting to standing up
11. Standing up to sitting down
12. Standing picking up a pencil from the floor

Functional Ambulation Classification (FAC)

Category	Definition
0 = Nonfunctional Ambulation	Patient cannot ambulate, ambulates in parallel bars only, or requires supervision or physical assistance from more than one person to ambulate safely outside of parallel bars.
1 = Ambulator-Dependent for Physical Assistance level 2	Patient requires manual contacts of no more than one person during ambulation on level surfaces to prevent falling. Manual contacts are continuous and necessary to support body weight as well as maintain balance and/or assist coordination.
2 = Ambulator-Dependent for Physical Assistance level 1	Patient requires manual contacts of no more than one person during ambulation on level surfaces to prevent falling. Manual contacts are consists of continuous or intermittent light touch to assist balance or coordination.
3 = Ambulator-Dependent for Supervision	Patient can physically ambulate on level surfaces without manual contacts of another person but for safety requires standby guarding of no more than one person because of poor judgment, question able cardiac status, or need for verbal cueing to complete the task.
4 = Ambulator-Dependent Level Surfaces only	Patient can ambulate independently on the level surfaces but requires supervision or physical assistance to negotiate any of the following: stairs, inclines, or non-level surfaces.
5 = Ambulator-Independent	Patient can ambulate independently on non-level and level surfaces, stairs, and inclines.

APPENDIX E

Data collection form

รหัสผู้เข้าร่วมวิจัย _____

วัน/เดือน/ปี _____ ครั้งที่ 1

วัน/เดือน/ปี _____ ครั้งที่ 2

ชีพจรขณะพัก

การทดสอบครั้งที่ 1 _____ครั้ง/นาที

การทดสอบครั้งที่ 2 _____ครั้ง/นาที

ความดันโลหิต

การทดสอบครั้งที่ 1 _____มิลลิเมตรปรอท

การทดสอบครั้งที่ 2 _____ มิลลิเมตรปรอท

มีความรู้สึกไม่สบายก่อนการทดสอบหรือไม่

การทดสอบครั้งที่ 1 ☐ ไม่ ☐ ใช่ ระบุ _____

การทดสอบครั้งที่ 2 ☐ ไม่ ☐ ใช่ ระบุ _____

1. Isokinetic peak torque

Average peak torque (Nm)	Baseline		6-week	
	Paretic	Non-paretic	Paretic	Non-paretic
Knee extensors				
Knee flexors				
Hip extensors				
Hip flexors				
Hip abductors				
Ankle plantarflexors				
Ankle dorsiflexors				

2. Ground reaction force

Variables		Side	Baseline				6-week			
			Trial 1	Trial 2	Trial 3	Mean	Trial 1	Trial 2	Trial 3	Mean
V GRF	Fz1 (N)	Paretic								
		Non-paretic								
	Fz2 (N)	Paretic								
		Non-paretic								
A-P GRF	Fx1 (N)	Paretic								
		Non-paretic								
	Fx2 (N)	Paretic								
		Non-paretic								
M-L GRF	Fy1 (N)	Paretic								
		Non-paretic								
	Fy2 (N)	Paretic								
		Non-paretic								

3. Standing postural sway

Condition	Variables	Baseline				6-week			
		Trial 1	Trial 2	Trial 3	Mean	Trial 1	Trial 2	Trial 3	Mean
Eye opened	Sway velocity (cm/s)								
	Sway area (cm^2)								
	M-L sway (cm)								
	A-P sway (cm)								
Eye closed	Sway velocity (cm/s)								
	Sway area (cm^2)								
	M-L sway (cm)								
	A-P sway (cm)								

4. Physiological cost index

Variables	Baseline	6-week
PCI (b/m)		

5. Maximum walking speed

Trial	1	2	3	Mean
Walking speed (m/s)				

BIOGRAPHY

NAME	Mr. Ratakorn Aimkosa
DATE OF BIRTH	10 May 1982
PLACE OF BIRTH	Nakonrathchasima, Thailand
INSTITUTIONS	Mahidol University, 2001- 2004 Bachelor of Science (Sports Science) Mahidol University, 2005- 2010 Master of Science (Sports Science)
HOME ADDRESS	41/382 Therddumri Road, Thanonnakornchaisri, Dusit, Bangkok Tel. 02-668-4881 E-mail. Ratakorn_Aimkosa @ Hotmail.com
EMPLOYMENT ADDRESS	Charoenkrungpracharuk Hospital 8 Charoenkrung, 111 Road, Bangkholam, Bangkok Tel. 02-289-7872-3