RELATIONSHIP BETWEEN PARETIC LOWER EXTREMITY LOADING DURING SIT-TO-STAND AND GAIT SPEED FROM 1 TO 6 MONTHS POST STROKE

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ABSTRACT

SUKWIDA MANORANGSAN: Relationship Between Paretic Lower Extremity Loading During Sit-To-Stand And Gait Speed From 1 to 6 Months Post Stroke
(Under the direction of Vicki S. Mercer, PT, Ph.D.)

The purpose of this study was to determine the relationship between paretic lower extremity (LE) loading during sit-to-stand (STS) and gait speed in persons recovering from stroke. Subjects (n=17; mean age = 56.8 ± 14.2 years) were tested at monthly intervals from 1 to 6 months (M1 – M6) post stroke using force platform measures to assess lower extremity loading during STS and the 10-meter walk test to assess self-selected and fast gait speeds. Paretic LE loading during STS tended to increase over the first three months and then plateau, with significant differences between M1 and M3-M6 and between M2 and M5-M6 (Tukey's $HSD_{.05} = 3.22$; $p<.05$). Gait speeds also improved, with the largest gains between M1 and M2. Measures of magnitude of paretic LE loading were positively correlated with gait speeds, while measures of the time required to load the LEs were negatively correlated with gait speeds at every month.

DEDICATION

To my parents, parent-in-law, and to my husband,

Rangsarit Vanijjirattikhan, for their endless love, support, and encouragement

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LIST OF ABBREVIATIONS

AP = antero-posterior direction

% BW = percent body weight

COM = center of mass

COP = center of pressure

CPT = conventional physical therapy

EMG = electromyography

GRF = ground reaction force

LE = lower extremity

ML = medio-lateral direction

NP = non-paretic

OG = over ground

P = paretic

STS = sit-to-stand

TM = treadmill

vGRF = vertical ground reaction force

LIST OF DEFINITIONS

- BW distribution ratio = weight load ratio between paretic and non-paretic lower limb
- COM = the point where the entire mass of the body is concentrated
- COP = the point where the resultant of all ground reaction forces acts
- GRF = the reaction to the force the body exerts on the ground; equal in magnitude and opposite in direction to the force that the body exerts on the supporting surface through the foot
- Flexion-momentum phase of STS = trunk forward flexion generates upper-body momentum during rising from a chair
- Momentum-Transfer phase of STS = forward momentum of the upper body is transferred to forward and upward momentum of the total body
- Extension phase of STS = hips and knees extend and the body rises to its full upright position
- Stabilization phase of STS = begins when hip and knee achieve full extension and continues until all motion associated with stabilization from rising is completed
- Seat-off = the instant at which the buttocks first leave the chair during sit-to-stand

INTRODUCTION

Stroke is the third leading cause of death in the United States after heart disease and cancer. The prevalence of stroke is higher among older people; approximately 88 percent of stroke deaths occur in people age 65 and older. In 2009, approximately 795,000 strokes were reported; 610,000 were first stroke, and 185,000 were recurrent.[1] A stroke may cause serious, long-term disabilities, at great cost to the health care system and to individuals.

Impairments after Stroke

The impairments resulting from stroke may involve motor, sensory, visual, perceptual, language, and cognitive systems. Kelly-Hayes et al[2] reported that residual disability in individuals who were at least 6 months post stroke included hemiparesis (50%), walking disability (30%), and dependence in activities of daily of living (ADL; 26%). Hemiparesis may underlie deficits in walking ability and performance of functional activities.[3-7] Hemiparesis may be caused by disruption in descending neural pathways leading to inadequate recruitment of motorneuron pools[8], disuse atrophy[9], and/or co-contraction of antagonist muscles.[10-12] Strength deficits after stroke may become long term. In one study, individuals who were 6 to 24 months post stroke continued to show deficits in lower extremity (LE) strength and power on the paretic compared to the nonparetic side.[13] In addition, there was a strong relationship between these deficits and performance of functional activities such as walking and stair climbing.

Factors Leading to Asymmetrical Weight Bearing after Stroke

Along with impairments in balance, sensation and/or perception,[14-16] hemiparesis is an important factor contributing to asymmetrical weight bearing during different functional activities.[13, 17-20] People with hemiparesis tend to bear more weight through the non-paretic LE and also have difficulty shifting their weight onto the paretic LE.[17-19, 21, 22] In addition, some evidence indicates that people with stroke may have difficulty shifting their weight onto the non-paretic leg.[23] Rogers et al[23] suggested that people with hemiparesis may adopt an asymmetrical static weight bearing posture to pre-compensate for a reduced capacity to produce dynamic weight transfer in the direction of the non-paretic limb.

If the pattern of avoiding weight bearing through the paretic leg continues, the individual may develop a learned non-use syndrome.[24] He or she may learn that use of the non-paretic limb permits easier and quicker performance of functional tasks. Continued disuse of the paretic limb may result in greater impairments in strength and range of motion. These impairments, in turn, may further limit functional performance.

How does Asymmetrical Weight Bearing Affect Functional Performance after Stroke?

A number of studies indicate that people with asymmetrical limb loading after stroke have difficulty performing tasks such as sit to stand, standing, and walking.[19, 20, 25-30]

Sit-To-Stand

The sit-to-stand (STS) movement is a complex functional movement that is influenced by lower limb strength and joint range of motion. Researchers have shown that people with hemiparesis after stroke have asymmetrical limb loading during STS.[9, 17-20, 27] Reported average paretic limb loading during STS range from 24% to 37.5% of body weight

(BW).[17, 19] The rate of rise in vertical ground reaction force (vGRF) during STS in people with stroke is lower than in their peers without stroke.[17] Cheng et al[17] found that the rate of rise in vGRF during STS was significantly lower in fallers with stroke (23.78 %BW/sec) as compared to non-fallers with stroke (55.23 %BW/sec) and comparison group subjects (85.96 %BW/sec).

People with asymmetrical limb loading after stroke may have differences not only in timing of vGRFs, but also in timing of muscle activation during STS. Cheng et al [25] reported that people with stroke show delayed onset of the tibialis anterior muscle and earlier onset of soleus muscle activation on the paretic side during STS.

Evidence suggests that individuals with stroke can increase limb loading when instructed to do so. Engardt and Olsson[19] reported that BW distribution was less asymmetrical when people with stroke (mean time post stroke = 38±22 days) were instructed to rise from a chair with "even" BW distribution compared to using a habitual pattern. The average limb loading on the paretic leg increased from 37.5%BW for rising habitually to 44.4%BW for rising evenly. In addition, the BW distribution ratio (paretic/non-paretic leg) increased from 0.60 to 0.80. Although deficits often remain, individuals typically show less weight bearing asymmetry during STS in the later stages of stroke recovery than in the early stages. Lomaglio et al[27] reported that the BW distribution ratio during STS in people with chronic stroke was 0.84. They also found significant relationships between the BW distribution ratio and STS duration at both self-paced and fast speeds ($r = -0.565$ and $r = -0.564$, respectively).

People with asymmetrical limb loading after stroke may take longer to perform STS compared to people with no known pathology. In the literature, mean STS completion times

range from 2.73 to 4.32 sec for people with stroke, considerably longer than for their peers without stroke (1.56 sec to 2.3 sec).[17, 19, 28, 29] Cheng et al[17] found that STS duration was significantly longer in fallers with stroke (4.32±2.22 sec) than in non-fallers with stroke (2.73±1.19 sec) and in non-stroke subjects (1.88±0.48 sec). In addition, people with stroke can perform STS more quickly when they are instructed to get up from a chair as fast as they can.[27, 28] Mazza et al[27, 28] reported that STS duration in individuals with stroke was significantly faster at maximal speed (1.58±0.68 sec) as compared to normal speed (2.80±1.15 sec).

Asymmetrical BW distribution during STS may be related to other functional abilities in people with stroke. Chou et al[26] reported that STS control was related to ambulation ability. They found that people with chronic stroke who could stand up within 4.5 sec or who had a vGRF difference of less than 30%BW between paretic and non-paretic legs during STS had better gait performance (velocity, cadence, stride time, single support).[26] Degree of paresis has been correlated with weight bearing difference during STS transfer.[20] Stroke patients who scored lower on the locomotion and mobility sub-tests of the Functional Independence Measure (FIM) had greater weight bearing difference during STS.[20]

One weakness in STS research in people post stroke is that most previous studies have used a cross-sectional design. Longitudinal relationships between STS performance and other functional abilities, including walking speed, in individuals recovering from stroke remain largely unknown. Therefore, the purpose of this study was to examine, for the first 6 months of stroke recovery, how measures of paretic lower extremity loading during STS and gait speed change over time, as well as how these measures relate to each other.

Research Questions and Hypotheses

1. How do measures of lower extremity loading during STS and gait speed change over time in the first 6 months of recovery after stroke?

-Hypotheses

1.1. Peak vertical ground reaction force (vGRF) beneath the paretic lower extremity and BW distribution ratio between paretic and non-paretic lower extremities during STS will increase from each month to the next over the first 6 months post stroke.

1.2. Gait speeds will increase from each month to the next over the first 6 months post stroke.

2. How do measures of lower extremity loading during STS relate to gait speeds in the first 6 months of recovery after stroke?

-Hypotheses

2.1 Peak vGRF beneath the paretic lower extremity and BW distribution ratio between paretic and non-paretic lower extremities during STS will be positively correlated with gait speeds at every month during the first 6 months post stroke.

2.2 Time to peak (TTP) vGRF beneath the paretic and non-paretic lower extremities, and absolute difference in TTP vGRF between paretic and non-paretic lower extremities during STS will be negatively correlated with gait speeds at every month during the first 6 months post stroke.

METHODS

This study was a secondary analysis of a dataset from a larger multiyear longitudinal study. The study included both laboratory and clinical tests of paretic lower limb loading and weight transfer conducted at monthly intervals during the first 6 months of stroke recovery. All subjects in the larger study for whom complete STS data were available (i.e., who attended all test sessions and were able to perform the STS task at each session) were included in the study.

Subjects

Participants were men and women over the age of 21 years who had sustained a unilateral hemispheric stroke less than 1 month before enrollment in the study. Participants were recruited from UNC Hospitals in Chapel Hill, North Carolina and WakeMed Rehab in Raleigh, North Carolina. Inclusion criteria were: 1) a primary diagnosis of stroke affecting one cerebral hemisphere (not cerebellum), as indicated by review of the medical record, 2) medically stable and free of major cardiovascular or musculoskeletal problems, as indicated by physician's approval for participation in the study, 3) able to understand and read English, 4) able to follow 3-step commands, 5) significant lower extremity motor impairment as indicated by a score of less than 28 on the lower extremity motor scale of the Fugl-Meyer Assessment[31] 6) adequate vision and hearing for completing the study protocol, as indicated by the ability to follow written and oral instructions during screening, and 7) residence within

a 50-mile radius of UNC and willingness to return to UNC for testing at monthly intervals over a 6 month period.

Exclusion criteria were: 1) a history of previous strokes or other neurologic diseases or disorders, such as Parkinson's Disease or Alzheimer's Disease, 2) inability to live independently or to ambulate independently in the community prior to the stroke, 3) terminal illness, or 4) pain, limited motion, or weakness in the non-paretic lower extremity that affected performance of daily activities (by self-report).

Participants were recruited during their initial hospitalization after stroke. Patients who appeared eligible for the study were contacted by a member of their health care team (e.g., nurse, physical therapist), who provided a brief explanation of the study and a permission slip for the patient to sign if he/she was willing to be contacted by the principal investigator (PI). The PI described the study in detail and completed a screening interview to obtain information about demographics, medical history, functional status prior to the stroke, and other eligibility criteria. Informed consent was obtained from patients deemed eligible for the study after this screening interview. Medical information such as lesion location then was determined from the medical record, and the presence of lower extremity motor impairment was determined by use of the lower extremity motor scale of the Fugl-Meyer Assessment. At this point, eligible patients were enrolled in the study, and the attending physician was asked to give medical approval (in writing) for the patient's participation. The study was approved by the Biomedical Institutional Review Board (IRB) at UNC and the IRB at WakeMed Rehab.

Data Collection Procedures

Subjects were tested one time per month from 1 to 6 months post stroke at the Center for Human Movement Science at UNC at Chapel Hill by the PI, a physical therapist who had 15 years of clinical experience at the time of the study. At the first test session, height and right foot length were measured for each subject. The foot length measurement was used to standardize subject foot placement during STS testing. Each subject's body weight was measured at every test session, and these measures were used to normalize the force plate data for that session. Tests of walking speed and lower extremity loading during STS were administered by the same investigator at each test session and are described below.

Walking Speed

For each participant, walking speed was assessed using a digital stopwatch to record the time needed to walk 10 meters. Each subject started five meters back from a 10-meter walkway and continued five meters beyond it to avoid including acceleration and deceleration in the determination of walking speed. Each participant performed this test under two conditions: self-selected speed and fast speed. For self-selected walking speed, subjects were instructed to walk at their usual, comfortable pace, and to begin walking whenever they were ready. For fast walking speed, subjects were instructed to walk as fast as they could without feeling unsafe, and to begin walking whenever they were ready. Two trials were performed for each speed. Walking speed measurement in individuals with neurological dysfunction has high test-retest reliability.[32, 33]

Lower Extremity Loading during STS

Two Bertec* (#N60501, Type 4060A, 40-cm x 60-cm) force plates mounted in the floor were used to measure ground reaction forces during STS. Subjects sat on a standard armless wooden chair (seat height 44.4 cm) with their bare feet positioned on a sheet of paper overlying two force platforms. The feet were positioned so that one foot was on each force platform and the distance between the midpoints of the heels was equal to the right foot length.[23] Foot tracings were used to facilitate consistency of foot position during testing. Two pressure switches were used to facilitate identification of STS movement phases. One was placed on the seat of the chair under the subject's buttocks to signal seat-off. The second pressure switch was depressed by one of the researchers when the subject achieved upright standing to help with identification of completion of the STS movement.

Each subject was asked to perform two practice and four test trials of the STS task. Although assistive devices and physical assistance were not allowed, subjects were guarded closely by one of the researchers for safety during task performance. Subjects were instructed to come to standing as rapidly as possible without feeling unsafe, and to try not to use their upper extremities to push up from the chair. At each test session, subjects first attempted to stand without UE support, and then were allowed to use UE support only if they were unable to stand without it. During each trial, the force platform and pressure switch data were collected at a sampling frequency of 500 Hz. The STS trials were videotaped to assist the researchers with interpretation of kinetic (force platform) data.

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* Bertec Corporation, Columbus, OH 43229

Data Reduction

Force plate data were exported from Peak Motus[†] to customized software programs[‡] for processing and reduction. The ground reaction force (GRF) signals were calibrated and converted into Newtons. Onset of the STS movement was defined as the point at which the vGRF exceeded 150% of baseline (determined for a 500 ms period with the feet resting on the force plates prior to start of the task). Dependent measures obtained from the force platform data are shown in Figure 1 and included: 1) **peak vGRF** for the paretic (P) and the non-paretic (NP) limbs, defined as the maximum value of the vertical GRF beneath the respective limb during the STS transfer; expressed as a percentage of body weight (%BW), 2) **BW distribution ratio,** defined as the ratio of peak vGRF of the P limb to that of the NP limb, 3) **TTP vGRF** for each lower limb, defined as the time interval between onset of the STS movement and peak vGRF beneath the limb, and 4) **absolute difference in TTP vGRF,** defined as the absolute value of the difference in TTP between P limb and NP limb. We chose to examine the absolute value of the difference in TTP vGRF between the LEs in order to quantify the degree of asymmetry in the temporal aspects of loading. Some people post stroke tended to load the P limb before the NP, whereas others do the reverse. Either situation represents a deviation from the synchronized increase in loading of the two LEs that typically occurs during STS.

[†] Performance Technologies Inc, Centennial, CO 80112

[‡] MotionSoft 3D v. 6.5 and MotionSoft Discrete Data Reader v. 6.0, Bing Yu, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7135

Data Analysis

All statistical analyses were performed using SPSS 15.0 for Windows (SPSS Inc., Chicago, Illinois). Descriptive statistics were calculated for subject characteristics (age, gender, race, weight, height, time since stroke onset, side of hemiparesis). Mean values also were calculated for the four STS test trials and for the two walking trials at each speed for each subject, and these means were entered into subsequent analyses. To address the first research question, a repeated measures one-way analysis of variance (ANOVA) and Tukey's HSD test were used to examine changes in STS performance and gait speed from months 1 to 6 post stroke. Separate ANOVAs were performed for each of the following dependent variables: 1) peak vGRF beneath the P limb, 2) BW distribution ratio between P and NP limbs, and 3) self-selected and fast gait speeds.

To address the second research question, Pearson product-moment correlation coefficients were calculated to determine bivariate relationships between measures of lower extremity loading during STS (i.e., peak vGRF for P limb, BW distribution ratio, TTP vGRF for each lower limb, absolute difference in TTP vGRF) and gait speed at months 1-6 post stroke. An alpha level of 0.05 was used as the level of significance for all statistical tests.

RESULTS

Seventeen participants from the larger study were able to attend all six test sessions and complete the STS task at each session, and thus were included in the present study. Descriptive statistics for subject characteristics are presented in Table 1. The sample had a wide age range, from 33 to 84 years, and was 82% male. Distribution of scores on the Fugl-Meyer LE motor scale is shown in Figure 2. All participants received usual medical care and rehabilitation during the study.

Most participants (n=12; 70.6%) had physical therapy (PT) intervention during the first 2-3 months post stroke (Table 2). At the first month post stroke, four participants reported receiving PT 2 times a day (on an inpatient basis), eight received PT 2-3 times a week (as outpatients), and five received no PT treatment. The number of participants receiving PT declined substantially after the second month post stroke, so that only 7 participants (41.2%) and 4 participants (23.5 %) were still receiving PT at the 3rd and 6th month post stroke, respectively. Seventy-one percent of participants used assistive devices, such as walkers or canes, to assist with ambulation at the first testing session, but only 5 (29%) still did so by the 3rd session In addition, 77% of participants used upper extremity (UE) support to come to standing at the first month, but only 6 participants (35.3%) and 3 participants (17.6 %) still did so at the 3rd and 6th month post stroke, respectively.

The first research question asked about changes from 1 to 6 months post stroke in lower extremity (LE) loading during STS and in fast and self-selected gait speeds. Mean values and standard deviations for these measures are presented in Table 3 and Figures 3 - 5.

Hypotheses 1.1 and 1.2, which stated that peak vGRF beneath the paretic LE, BW distribution ratio, and gait speeds would increase each month from one to six months post stroke, were not supported. Although the repeated measures ANOVAs revealed significant effects for month post stroke for all the dependent measures (Huynh-Feldt adjustment: $F_{(4.732, 75.715)} = 16.293$, $p<.001$ for peak vGRF beneath the paretic LE; $F_{(4.220, 67.522)} = 8.007$, $p<.001$ for BW distribution ratio; $F_{(3.371, 53.941)} = 26.927$, $p<.001$ for self-selected gait speed; and $F_{(2.557, 40.909)} = 16.156$, $p<.001$ for fast gait speed, respectively), there were no significant differences between any two consecutive months, except for between the first month (M1) and the second month (M2) for the gait speed measures.

Mean peak vGRF beneath the paretic LE tended to increase over the first three months and then plateau. Tukey's HSD test indicated significant differences between M1 and the third through sixth months (M3-M6) and between M2 and M5-M6 (Tukey's $HSD_{.05} = 3.22$; $p<.05$; Figure 3). Similar results were found for BW distribution ratio. The mean value for M1 was significantly lower than the means for M3 - M6, and the mean for M2 was significantly lower than that for M5 (Tukey's $HSD_{.05} = .071$; $p<.05$; Figure 4). As was the case for peak vGRF beneath the paretic LE, there were no significant between-month differences after the third month post stroke.

For gait speeds, mean values at M1 were significantly lower than those for M2 – M6 (Tukey's $HSD_{.05} = .303$ for fast gait speed; and Tukey's $HSD_{.05} = .140$ for self-selected gait speed; $p<.05$), and the mean for M2 was also significantly lower than that for M5-M6 post stroke at self-selected gait speed (Figure 5). The largest month-to-month increases occurred between the first and second months post stroke. Standard deviations were consistently smaller for self-selected than for fast gait speed.

The second research question focused on relationships between STS measures and gait speeds from 1 to 6 months post stroke. In addition to peak vGRF beneath the paretic LE and BW distribution ratio, STS measures included temporal aspects of performance, such as time-to-peak (TTP). Means and standard deviations for TTP vGRF beneath the paretic and the non-paretic LE and the absolute value of the difference in TTP vGRF between the two LEs are presented in Table 4. Mean values of these measures tended to decrease from the first to the sixth month post stroke.

Results of correlational analyses are presented in Tables 5 and 6. Hypothesis 2.1, stating that the STS measures of peak vGRF beneath the paretic LE and BW distribution ratio would be positively correlated with gait speed at every month from 1 to 6 months post stroke, was partially supported. Peak vGRF beneath the paretic LE was positively correlated with fast (Table 5) and self-selected (Table 6) gait speeds at each time point.

Correlations between BW distribution ratio and gait speeds also were positive, except for the correlation with fast gait speed at M6. These correlations tended to be stronger at M1 than at later time points, with values at M1 of $r = 0.52$ for fast gait speed and $r = 0.64$ for self-selected gait speed. By M6, these correlations were quite low.

Hypothesis 2.2, which stated that TTP vGRF beneath the paretic and the non-paretic LE and the absolute difference in TTP vGRF between the lower extremities during STS would be negatively correlated with gait speed at every month from 1 to 6 months post stroke was supported. The strongest relationships between TTP vGRF beneath the paretic limb and gait speeds occurred at M6, whereas the strongest relationships between TTP vGRF beneath the non-paretic limb and gait speeds occurred at M1 and M2.

The absolute difference in TTP vGRF between the paretic and non-paretic LEs also was negatively correlated with gait speeds from M1 to M6. These correlations tended to be stronger at M1 – M3 (r values ranging from -0.47 to -0.63), than at M4 – M6 (r values ranging from -0.28 to -0.46).

DISCUSSION

One objective of this study was to determine how characteristics of paretic LE loading during STS and gait speed change in the first 6 months of recovery after stroke. Although peak vGRF beneath the paretic LE, BW distribution ratio, and gait speeds generally increased from the first month to the sixth month post stroke, these increases were relatively small from month to month. The majority of improvement occurred in the first 2-3 months post stroke. Results of this study support the idea that neurological recovery is completed within the first three months post stroke.[34-37] As other researchers have reported, the course of motor recovery appears to reach a plateau after an early phase of progressive improvement.[37]

Jorgensen et al[34] reported that recovery of walking function occurs primarily within the first 11 weeks after stroke. In that study, recovery time for walking function increased with the severity of initial leg paresis. Best walking function was reached within 4 weeks in patients with no leg paresis, within 9 weeks in patients with mild or moderate paresis, and within 11 weeks in patients with severe paresis or paralysis. Improvements in lower-limb motor function reached a plateau between 3 and 6 months post stroke.[34] Olsen[38] reported that 95% of people with stroke in his study had achieved their highest level of walking function within 14 weeks after stroke.

Another factor that may have influenced the pattern of improvement over time in our study is the provision of PT services. Whereas 70.6% of participants received PT intervention during the first 2-3 months post stroke, only 35.3% were receiving PT by the 4th

month post stroke. Improvement in participants' performance, especially more subtle aspects of performance, such as weight bearing symmetry, may have leveled off following discontinuation of PT services.

Mean values for peak vGRF beneath the paretic LE during STS in our study ranged from a low of 46.4±8.0 %BW at the first month to a high of 54.6±7.3 %BW at the fifth month post stroke. These results are similar to those reported previously by Dean et al[39] (48.6±8.8 %BW) and Brunt et al[40] (53.0±9.0 %BW) for subjects who were at least 6 months and at least one year post stroke, respectively. Although Engardt and Olsson[19] reported a mean value of only 37.5 %BW, subjects in their study were tested during a more acute phase of stroke recovery (mean time post stroke of 38±22 days). When tested as early as one week post stroke, as in the Engardt and Olsson[19] study, subjects may have much more difficulty loading the paretic limb during STS than when they are tested after one month of recovery.

The increase in BW distribution ratio in our study paralleled the increase in paretic limb loading during STS. Mean values of BW distribution ratio in the present study ranged from 0.73±0.20 to 0.85±0.19. The BW distribution ratios we observed for participants at 4-6 months post stroke were very similar to the ratio of 0.84±0.20 reported by Lomaglio[27] for people with chronic stroke (mean time post stroke of 5.3±2.1 yr).

Mean gait speeds in this study increased significantly from M1 to M2, and also were significantly higher at M5 and M6 than at M1 and M2. Mean self-selected gait speed reached 0.97 m/s at M6, a somewhat higher value than that reported by Monger et al[41] for people with chronic stroke (0.86±0.31 m/s) and a considerably higher value than others reported in the literature for people recovering from stroke (0.2±0.16 m/s to 0.62±0.21 m/s).[42-46] These differing results may be due not only to variations in time since stroke onset, but also to other

factors, such as stroke severity and duration of rehabilitation. Mean self-selected gait speed for our participants was lower than that reported for community-dwelling older persons with no history of stroke (1.23±0.26 m/s).[47]

Improvements in paretic limb loading ability may have contributed to gait speed improvements from M1 to M6, but gains in balance and in paretic limb control during swing may have contributed as well. Measurement of the latter abilities in future studies will increase our understanding of factors influencing gait speed after stroke. Compensations involving the non-paretic lower extremity and/or the trunk may play a role in gait speed improvements. As Rogers et al[23] suggested, people with hemiparesis may adopt an asymmetrical static weight bearing posture to pre-compensate for a reduced capacity to produce dynamic weight transfer in the direction of the non-paretic limb.

Self-selected and fast gait speeds were positively correlated with peak vGRF beneath the paretic LE during STS at every month from 1 to 6 months post stroke. Increased ability to load the paretic limb may enable more effective push-off during walking. The pattern of relationship between BW distribution ratio during STS and gait speed was similar to that for peak vGRF and gait speed. These relationships tended to be moderately strong during the first few months, and then declined. This suggests that improvement in paretic limb loading during STS during the chronic phase of stroke recovery may be quite limited. Some individuals recovering from stroke may learn to compensate by transferring weight quickly onto the non-paretic limb or by pushing on an assistive device during walking. Use of assistive devices and/or orthoses can permit relatively fast gait speeds despite poor paretic limb loading. [48]

Gait speed was negatively correlated with TTP vGRF beneath the paretic and the non-paretic LE and with the absolute difference in TTP vGRF between the lower extremities during STS at every month from 1 to 6 months post stroke. The ability to load the LE quickly during STS may be related to the ability to quickly load and unload the LE for weight transfer during walking. Rapid LE loading during STS also may reflect better ability to generate force through the LE rapidly, as is needed for push-off during gait. The magnitude of the negative correlation between gait speed and TTP vGRF beneath the paretic LE during STS tended to increase over time, reaching a value of r = -0.58 at M6 for both fast and self-selected gait speed. Correlations between gait speed and the other two TTP variables, however, tended to decrease over time.

A major clinical implication of this study is that ability to load the paretic LE may be an important factor affecting walking speed in people with stroke, particularly during the first three months of recovery. In addition, the ability to load the lower limbs quickly and with similar timing between limbs may be important for improvement of walking speed. A major strength of this study is the monthly recording of identical measures of STS and gait performance from the same participants during the first 6 months post stroke.

On the other hand, this study had several limitations. First, the sample size of the study was small. With 17 subjects, we had 0.80 power to detect correlations of r = 0.70 or greater at an alpha level of 0.05. . Second, the participants in this study were selected on the basis of their ability to perform STS at each monthly test session. Consequently, they were not representative of the broader population of people post stroke, many of whom are unable to come to standing without physical assistance, especially in the first few months after stroke. Third, the researchers did not control the amount, nature, or timing of physical

therapy or other interventions that the participants received, complicating interpretation of factors which may have contributed to improvements in performance. Lastly, some subjects needed to use their hands to push up from the chair during STS. Although we believe the recorded vGRFs were an accurate reflection of each subject's ability to load the LEs, use of the NP upper extremity for support may have resulted in increased loading of the NP compared to the P lower extremity.

CONCLUSIONS

The ability to load the paretic LE during STS improved over the first 6 months of stroke recovery, with most improvement occurring in the first 2-3 months. This ability was positively correlated with self-selected and fast gait speeds at every month from 1 to 6 months post stroke. The relationship between paretic limb loading and gait speed was stronger in the first few months than in subsequent months post stroke. Clinicians may want to focus on achieving symmetrical LE weight bearing during functional activities, such as STS, as this may be a factor affecting gait speed during the first few months of recovery.

The rate of LE loading (as reflected by TTP vGRF beneath the paretic and non-paretic LEs) and the absolute difference in TTP vGRF between LEs during STS tended to decrease over the first 6 months post stroke. Time-to-peak vGRF and the absolute difference in TTP between the LEs during STS were negatively correlated with self-selected and fast gait speeds at every month from 1 to 6 months post stroke. Ability to load the paretic limb quickly and to achieve similar timing between the two lower limbs during STS is associated with faster walking speeds.

Table 1.

Descriptive statistics for subject characteristics (n=17).

Variables	Mean or Percentage	S.D.	Range
Age (years)	56.8	14.2	33-84
Gender **- Male (n=14)** **- Female (n=3)**	 82% 18%		
Body weight (kg)	95.1	31.3	47.6-189.6
Body height (cm)	176.6	8.8	162.6-193.0
Race **-White (n=10)** **-African American (n=7)**	 59% 41%		
Paretic side **-Right (n=6)** **-Left (n=11)**	 35% 65%		
Fugl Meyer Lower Extremity motor score (out of 34)	19.7	5.3	8-28

Table 2.

Percent of participants receiving physical therapy intervention, using an assistive device, and using upper extremity support to rise from a chair during STS over the first six months post stroke.

	Month					
	1	**2**	**3**	**4**	**5**	**6**
PT (%)	70.6	82.4	41.2	35.3	29.4	23.5
AD (%)	70.6	41.2	29.4	29.4	29.4	29.4
UE (%)	76.5	41.2	35.3	23.5	23.5	17.6

PT: physical therapy; AD: assistive device (during walking); UE: upper extremity support (during STS)

Table 3.

Mean (SD) for peak vertical ground reaction force beneath paretic lower extremity, body weight distribution ratio between lower extremities during sit-to-stand, and gait speeds from 1 to 6 months post stroke.

Variables	Month					
	1	2	3	4	5	6
Peak vGRF-paretic (%BW)	46.42 (8.02)	49.16 (8.12)	51.45 (7.93)	52.30 (9.57)	54.57 (7.28)	53.87 (6.01)
Body weight distribution ratio	0.73 (0.20)	0.77 (0.22)	0.80 (0.19)	0.82 (0.22)	0.85 (0.19)	0.84 (0.19)
Fast gait speed (m/s)	0.70 (0.57)	1.07 (0.54)	1.11 (0.50)	1.21 (0.54)	1.17 (0.66)	1.29 (0.55)
Self-selected gait speed (m/s)	0.58 (0.35)	0.78 (0.35)	0.84 (0.34)	0.91 (0.35)	0.93 (0.33)	0.97 (0.32)

Peak vGRF - paretic: peak value of the vertical ground reaction force (vGRF) beneath the paretic lower extremity (LE);
Body weight distribution ratio: the ratio of vGRF of paretic to that of non-paretic lower extremities.

Table 4.

Mean (SD) for time to peak vertical ground reaction force beneath paretic and non-paretic lower extremities and absolute value of the difference in time to peak vertical ground reaction force between the lower extremities during sit-to-stand from 1 to 6 months post stroke.

Variables	Months					
	1	2	3	4	5	6
Time to peak-Paretic (s)	0.704 (0.82)	0.544 (0.81)	0.377 (0.34)	0.454 (0.51)	0.330 (0.19)	0.344 (0.18)
Time to peak-nonparetic (s)	0.772 (0.69)	0.519 (0.45)	0.401 (0.35)	0.417 (0.37)	0.350 (0.29)	0.306 (0.15)
Absolute difference in time to peak (s)	0.307 (0.43)	0.225 (0.47)	0.132 (0.19)	0.101 (0.16)	0.133 (0.25)	0.079 (0.09)

Time to peak-paretic: time to peak vertical ground reaction force (vGRF) beneath paretic lower extremity (LE); Time to peak-nonparetic: time to peak vGRF beneath non-paretic LE; Absolute difference in time to peak: absolute value of the difference in time to peak vGRF between paretic and non-paretic lower extremities.

Table 5.

Pearson product-moment correlation coefficients for relationships between sit-to-stand measures and fast gait speed from 1 to 6 months post stroke.

Month / Variables	Fast gait speed					
	1	**2**	**3**	**4**	**5**	**6**
Peak vGRF-paretic	.45	.50*	.48	.38	.46	.37
Body weight distribution ratio	.52*	.43	.27	.21	.17	-.11
Time to peak-paretic	-.37	-.42	-.43	-.37	-.40	-.58*
Time to peak-nonparetic	-.67**	-.63**	-.52*	-.44	-.41	-.48*
Absolute difference in time to peak	-.62**	-.47	-.54**	-.40	-.34	-.28

*Significant at p<.05; **Significant at p<.01; Peak vGRF - paretic: peak value of the vertical ground reaction force (vGRF) beneath the paretic lower extremity (LE); Time to peak-paretic: time to peak vGRF beneath paretic LE; Time to peak-nonparetic: time to peak vGRF beneath non-paretic LE; Absolute difference in time to peak: absolute value of the difference in time to peak vGRF between paretic and non-paretic lower extremities.

Table 6.

Pearson product-moment correlation coefficients for relationships between sit-to-stand measures and self-selected gait speed from 1 to 6 months post stroke.

Month / Variables	Self-selected gait speed					
	1	**2**	**3**	**4**	**5**	**6**
Peak vGRF-paretic	.55*	.56*	.52*	.46	.68**	.44
Body weight distribution ratio	.64**	.47	.37	.33	.44	.03
Time to peak-paretic	-.32	-.45	-.36	-.30	-.46	-.58*
Time to peak-nonparetic	-.69**	-.64**	-.47	-.37	-.49*	-.42
Absolute difference in time to peak	-.63**	-.48	-.48	-.32	-.46	-.39

*Significant at p<.05; **Significant at p<.01; Peak vGRF - paretic: peak value of the vertical ground reaction force (vGRF) beneath the paretic lower extremity (LE); Time to peak-paretic: time to peak vGRF beneath paretic LE; Time to peak-nonparetic: time to peak vGRF beneath non-paretic LE; Absolute difference in time to peak: absolute value of the difference in time to peak vGRF between paretic and non-paretic lower extremities.

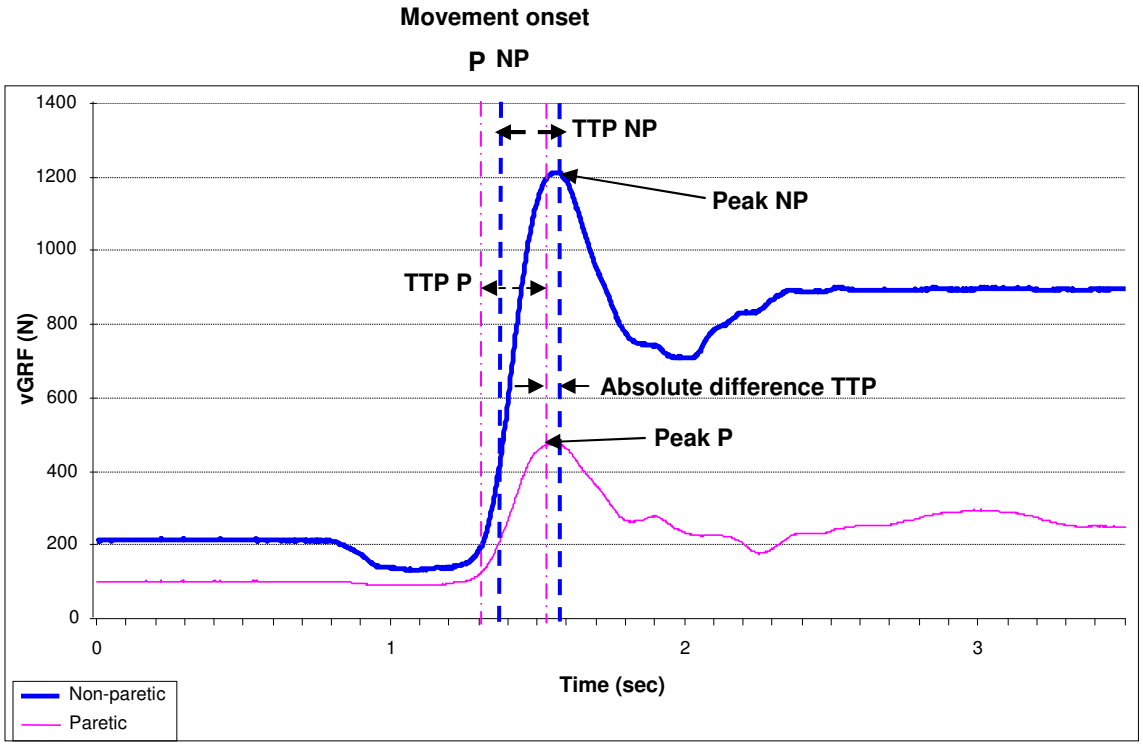


Figure 1. Vertical ground reaction force (vGRF) beneath the paretic (P) and non-paretic (NP) lower extremities (LEs) for one representative STS trial. Peak NP = peak vGRF for non-paretic LE; Peak P = peak vGRF for paretic LE; TTP NP = time to peak vGRF for non-paretic LE, TTP P = time to peak vGRF for paretic LE; Absolute difference TTP = absolute value of difference between TTP NP and TTP P.

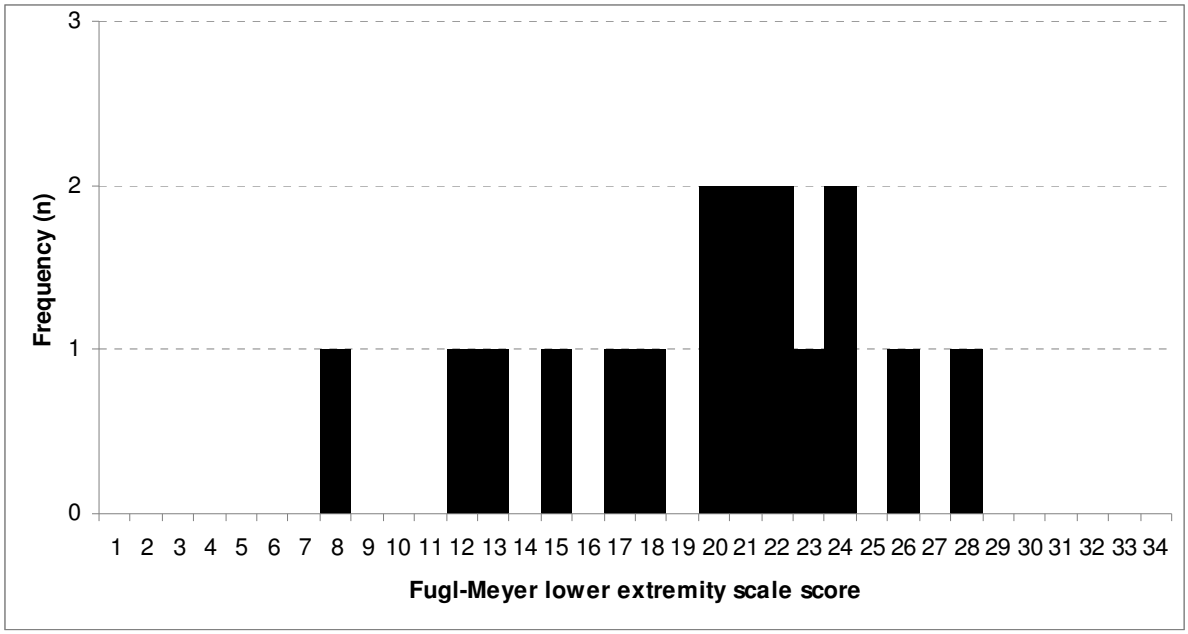
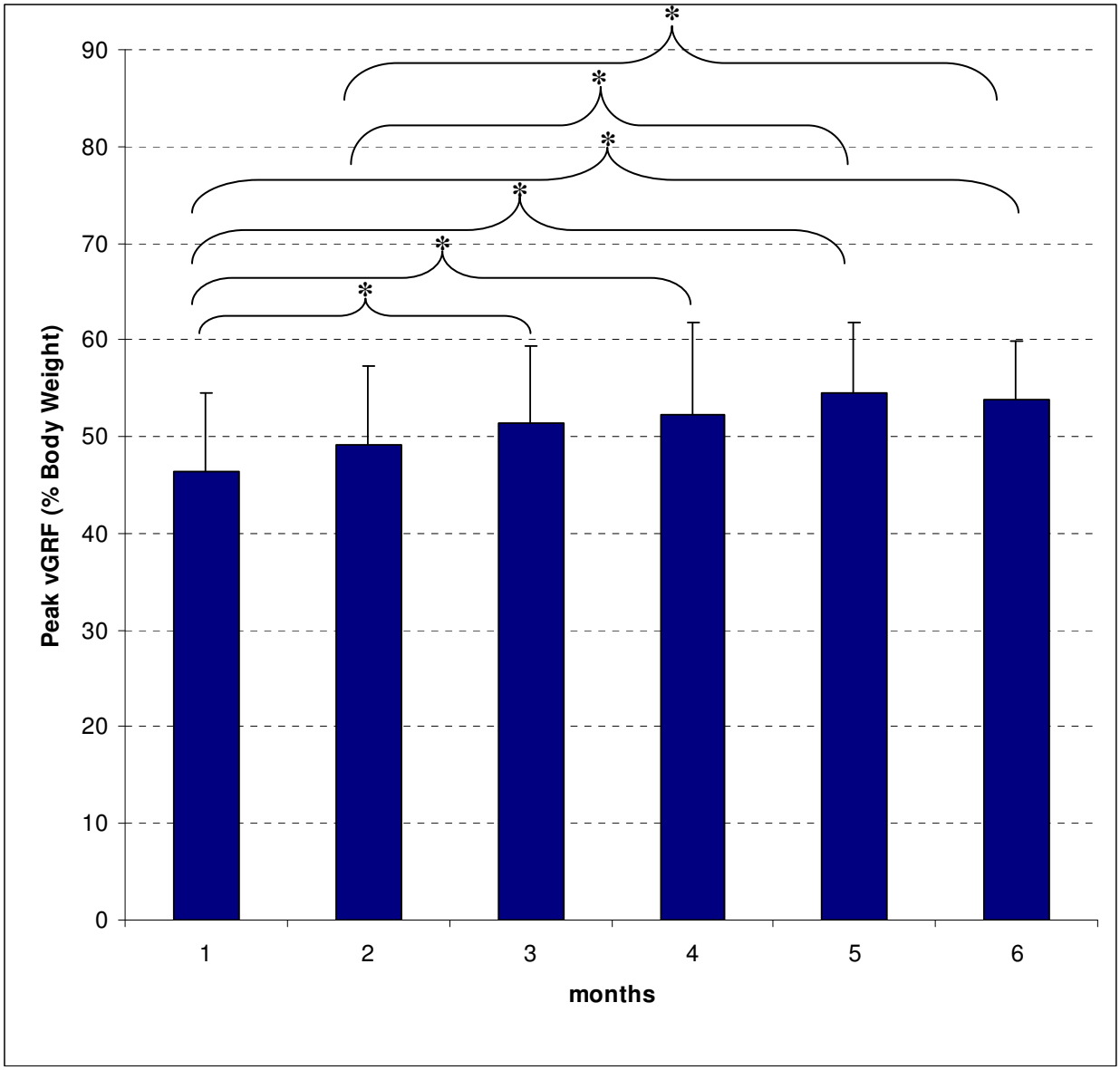
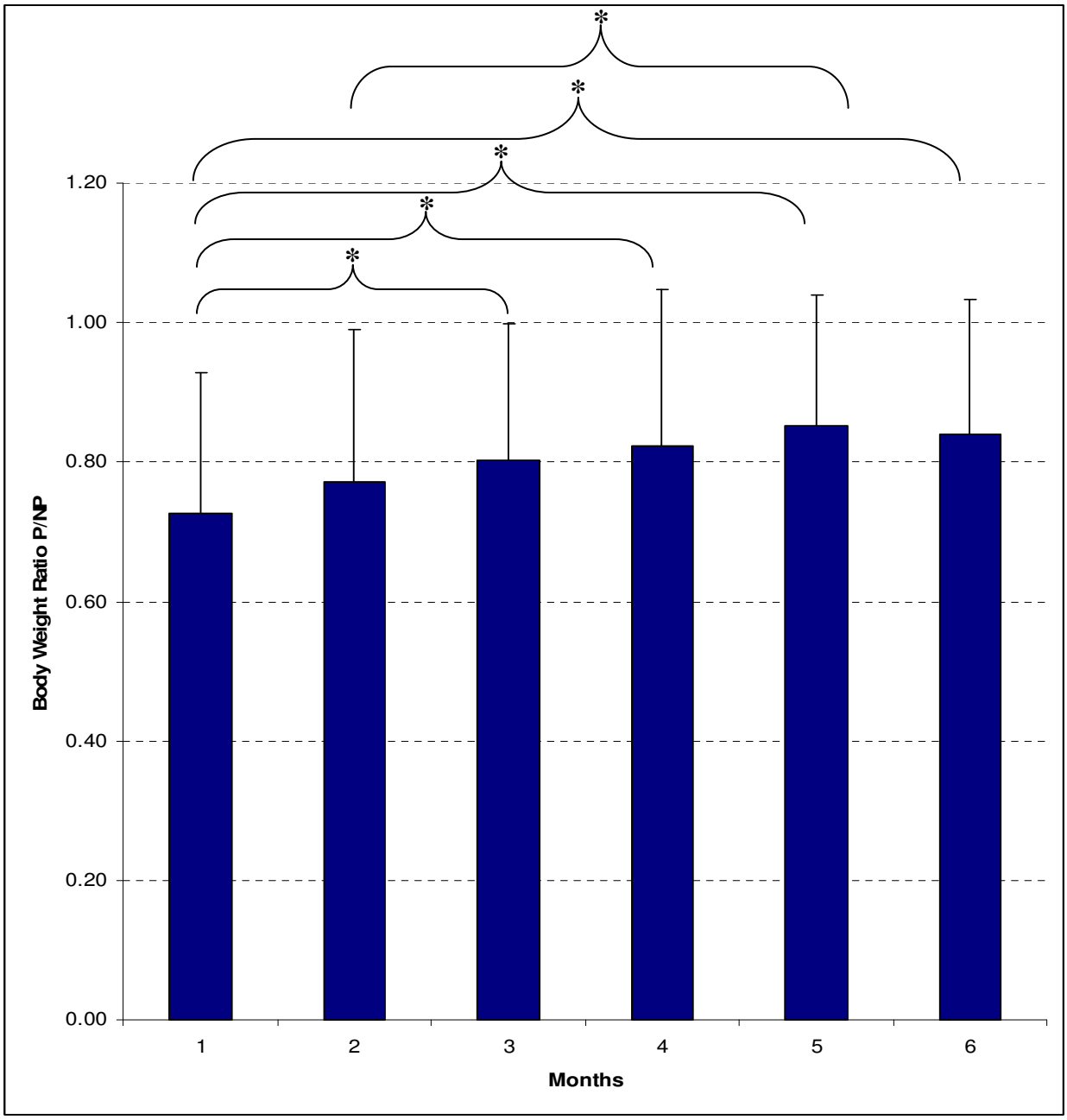


Figure 2. Histogram for the distribution of scores on the Fugl-Meyer lower extremity motor scale (maximum score = 34).



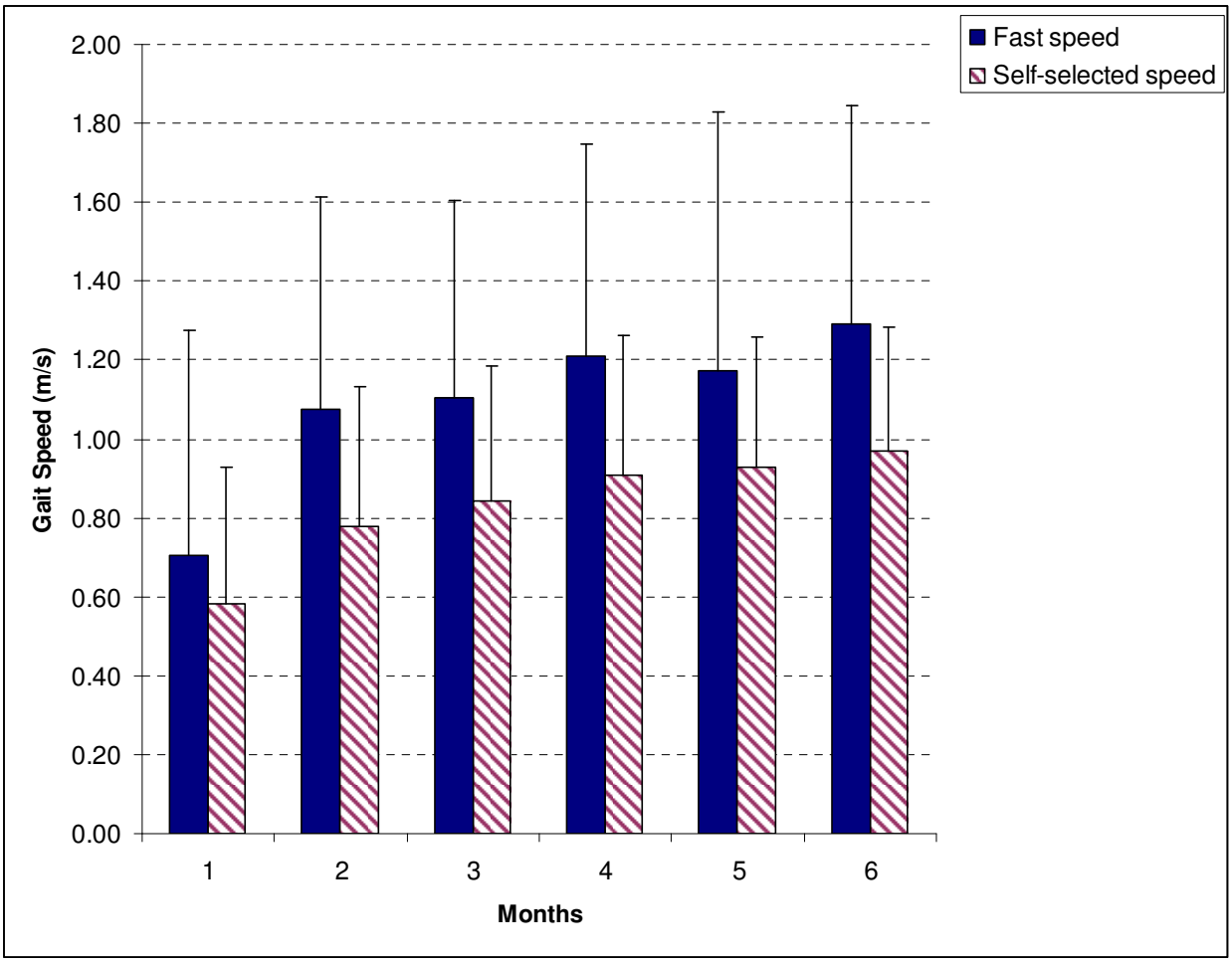
*Significant difference at p< .05; Error bars represent one standard deviation

Figure 3. Peak vertical ground reaction force beneath paretic lower extremity from 1 to 6 months post stroke.



*Significant difference at p< .05; Error bars represent one standard deviation; Body Weight Ratio P/NP: body weight distribution ratio between paretic and non-paretic lower extremities

Figure 4. Body weight distribution ratio between paretic and non-paretic lower extremities from 1 to 6 months post stroke.



Error bars represent one standard deviation

Figure 5. Fast and self-selected gait speeds from 1 to 6 months post stroke.

REFERENCES

1. *Heart Disease and Stroke Statistics: 2009 Update*: American Heart Association. 2009.

2. Kelly-Hayes M, Beiser A, Kase CS, Scaramucci A, D'Agostino RB, Wolf PA. The influence of gender and age on disability following ischemic stroke: the Framingham study. *J Stroke Cerebrovasc Dis.* May-Jun 2003;12(3):119-126.

3. 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.* Aug 2003;84(8):1185-1193.

4. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. *Clin Biomech (Bristol, Avon).* Feb 1999;14(2):125-135.

5. Neckel N, Pelliccio M, Nichols D, Hidler J. Quantification of functional weakness and abnormal synergy patterns in the lower limb of individuals with chronic stroke. *J Neuroeng Rehabil.* 2006;3:17.

6. Patterson KK, Parafianowicz I, Danells CJ, et al. Gait asymmetry in community-ambulating stroke survivors. *Arch Phys Med Rehabil.* Feb 2008;89(2):304-310.

7. Roy G, Nadeau S, Gravel D, Piotte F, Malouin F, McFadyen BJ. Side difference in the hip and knee joint moments during sit-to-stand and stand-to-sit tasks in individuals with hemiparesis. *Clin Biomech (Bristol, Avon).* 2007; 22:795-804.

8. Bourbonnais D, Vanden Noven S. Weakness in patients with hemiparesis. *Am J Occup Ther.* May 1989;43(5):313-319.

9. Scelsi R, Lotta S, Lommi G, Poggi P, Marchetti C. Hemiplegic atrophy. Morphological findings in the anterior tibial muscle of patients with cerebral vascular accidents. *Acta Neuropathol.* 1984;62(4):324-331.

10. Chae J, Yu DT. Neuromuscular electrical stimulation for motor restoration in hemiparesis. *Top Stroke Rehabil.* Winter 2002;8(4):24-39.

11. Knutsson E, Martensson A. Dynamic motor capacity in spastic paresis and its relation to prime mover dysfunction, spastic reflexes and antagonist co-activation. *Scand J Rehabil Med.* 1980;12(3):93-106.

12. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain.* Jun 1979;102(2):405-430.

13. LeBrasseur NK, Sayers SP, Ouellette MM, Fielding RA. Muscle impairments and behavioral factors mediate functional limitations and disability following stroke. *Phys Ther.* Oct 2006;86(10):1342-1350.

14. Bonan IV, Colle FM, Guichard JP, et al. Reliance on visual information after stroke. Part I: Balance on dynamic posturography. *Arch Phys Med Rehabil.* Feb 2004;85(2):268-273.

15. Marigold DS, Eng JJ. The relationship of asymmetric weight-bearing with postural sway and visual reliance in stroke. *Gait Posture.* Feb 2006;23(2):249-255.

16. Niam S, Cheung W, Sullivan PE, Kent S, Gu X. Balance and physical impairments after stroke. *Arch Phys Med Rehabil.* Oct 1999;80(10):1227-1233.

17. Cheng PT, Liaw MY, Wong MK, Tang FT, Lee MY, Lin PS. The sit-to-stand movement in stroke patients and its correlation with falling. *Arch Phys Med Rehabil.* Sep 1998;79(9):1043-1046.

18. Engardt M. Long-term effects of auditory feedback training on relearned symmetrical body weight distribution in stroke patients. A follow-up study. *Scand J Rehabil Med.* Jun 1994;26(2):65-69.

19. Engardt M, Olsson E. Body weight-bearing while rising and sitting down in patients with stroke. *Scand J Rehabil Med.* 1992;24(2):67-74.

20. Lee MY, Wong MK, Tang FT, Cheng PT, Lin PS. Comparison of balance responses and motor patterns during sit-to-stand task with functional mobility in stroke patients. *Am J Phys Med Rehabil.* Sep-Oct 1997;76(5):401-410.

21. Pai YC, Rogers MW, Hedman LD, Hanke TA . Alterations in weight transfer capabilities in adults with hemiparesis. *Phys Ther.* 1994;74(7):647-59.

22. Turnbull GI, Charteris J, Wall JC. Deficiencies in standing weight shifts by ambulant hemiplegic subjects. *Arch Phys Med Rehabil.* Apr 1996;77(4):356-362.

23. Rogers MW, 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(11):19-25.

24. Taub E, Miller NE, Novack TA, et al. Technique to improve chronic motor deficit after stroke. *Arch Phys Med Rehabil.* Apr 1993;74(4):347-354.

25. Cheng PT, Chen CL, Wang CM, Hong WH. Leg muscle activation patterns of sit-to-stand movement in stroke patients. *Am J Phys Med Rehabil.* Jan 2004;83(1):10-16.

26. Chou SW, Wong AM, Leong CP, Hong WS, Tang FT, Lin TH. Postural control during sit-to stand and gait in stroke patients. *Am J Phys Med Rehabil.* Jan 2003;82(1):42-47.

27. Lomaglio MJ, Eng JJ. Muscle strength and weight-bearing symmetry relate to sit-to-stand performance in individuals with stroke. *Gait Posture.* Oct 2005;22(2):126-131.

28. Mazza C, Stanhope SJ, Taviani A, Cappozzo A. Biomechanic modeling of sit-to-stand to upright posture for mobility assessment of persons with chronic stroke. *Arch Phys Med Rehabil.* May 2006;87(5):635-641.

29. Papa E, Cappozzo A. Sit-to-stand motor strategies investigated in able-bodied young and elderly subjects. *J Biomech.* Sep 2000;33(9):1113-1122.

30. Yoshida K, Iwakura H, Inoue F. Motion analysis in the movements of standing up from and sitting down on a chair. A comparison of normal and hemiparetic subjects and the differences of sex and age among the normals. *Scand J Rehabil Med.* 1983;15(3):133-140.

31. Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S. The post-stroke hemiplegic patient. 1. a method for evaluation of physical performance. *Scand J Rehabil Med.* 1975;7(1):13-31.

32. Collen FM, Wade DT, Bradshaw CM. Mobility after stroke: reliability of measures of impairment and disability. *Int Disabil Stud.* Jan-Mar 1990;12(1):6-9.

33. Schenkman M, Cutson TM, Kuchibhatla M, Chandler J, Pieper C. Reliability of impairment and physical performance measures for persons with Parkinson's disease. *Phys Ther.* Jan 1997;77(1):19-27.

34. Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients: the Copenhagen stroke study. *Arch Phys Med Rehabil.* 1995;76:27-32.

35. Wade DT, Wood VA, Heller A, Maggs J, Langton Hewer R. Walking after stroke: measurement and recovery over the first 3 months. *Scand J Rehabil Med*. 1987;19(1):25-30.

36. Jorgensen HS, Nakayama H, Raaschou HO, Vive-Larsen J, Steier M, Olsen TS. Outcome and time course of recovery in stroke. Part II: time course of recovery: the Copenhagen stroke study. *Arch Phys Med Rehabil.* 1995;76:406-12.

37. Bruno-Petrina A. Motor recovery in stroke. *emedicine.medscape.com/article*. Oct 2009:6.

38. Olsen TS. Arm and leg paresis as outcome predictors in stroke rehabilitation. *Stroke.* 1990; 21:247-51.

39. Dean CM, Richards CL, Malouin F. Task-related circuit training improve performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. *Arch Phys Med Rehabil.* Apr 2000;81(4):409-417.

40. Brunt D, Greenberg B, Wankadia S, Trimble MA, Shechtman O. The effect of foot placement on sit to stand in healthy young subjects and patients with hemiplegia. *Arch phys Med Rehabil.* Jul 2002;83(7):924-929.

41. Monger C, Carr JH, Fowler V. Evaluation of a home-based exercise and training programme to improve sit-to-stand in patients with chronic stroke. *Clin Rehabil.* Jun 2002;16(4):361-367.

42. Dunsky A, Dickstein R, Marcovitz E, Levy S, Deutsch JE. Home-based motor imagery training for gait rehabilitation of people with chronic poststroke hemiparesis. *Arch Phys Med Rehabil.* Aug 2008;89(8):1580-1588.

43. 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.* Aug 2003;84(8):1185-1193.

44. Laufer Y, Dickstein R, Chefez Y, Marcovitz E. The effect of treadmill training on the ambulation of stroke survivors in the early stages of rehabilitation: a randomized study. *J Rehabil Res Dev.* Jan-Feb 2001;38(1):69-78.

45. Regnaux JP, Pradon D, Roche N, Robertson J, Bussel B, Dobkin B. Effects of loading the unaffected limb for one session of locomotor training on laboratory measures of gait in stroke. *Clin Biomech (Bristol, Avon).* Jul 2008;23(6):762-768.

46. Trueblood PR. Partial body weight treadmill training in persons with chronic stroke. *NeuroRehabilitation.* 2001;16(3):141-153.

47. Deshpande N, Metter J, Bandinelli S, Guralnik J, Ferrucci L. Gait speed under varied challenges and cognitive decline in older persons: a prospective study. *Age and Ageing.* 2009; 38: 509–514.

48. Kottink A, Oostendorp L, Buurke J, Nene A, Hermens H, IJzerman M. The orthotic effect of functional electrical stimulation on the improvement of walking in stroke patients with a dropped Foot: A systematic review. Artificial Organs. 2004;28(6):577–586.

APPENDIX

LITERATURE REVIEW

1. Epidemiology and Sequelae of Stroke

Stroke is the third leading cause of death in the United States after heart disease and cancer. In 2009, approximately 795,000 strokes were reported; 610,000 were first strokes, and 185,000 were recurrent. Every 40 seconds, someone in the United States has a stroke. Eighty-seven percent of all strokes are ischemic, whereas 10 percent and 3 percent involve intracerebral and subarachnoid hemorrhage, respectively.[1] In addition, stroke is a leading cause of long-term disability. Outcomes in individuals with chronic stroke (at least 6 months post stroke) include residual hemiparesis (50%), walking disability (30%), dependence in activities of daily of living (ADL; 26%), and nursing home placement (26%).[2] Direct and indirect costs of stroke for 2009 are expected to total $68.9 billion.[1]

One month after stroke onset, 40% of patients undergoing rehabilitation are able to stand independently for one minute, 40% are not able to stand at all, and the other 20% can stand with help.[3] The median time to recover the ability to stand 10 seconds covaries with the size and site of the lesion: 0 days for a lacunar infarct, 4 days for a posterior circulation infarct, and 44 days for a total anterior circulation infarct.[4]

Impairments after stroke may involve motor, sensory, visual, perceptual, and/or cognitive systems. This literature review will emphasize impairments involving the lower extremity**. Motor impairment**: The primary contributor to lower extremity motor deficits in patients with chronic stroke is weakness.[5] Neckel et al[5] studied lower limb weakness and synergy patterns in patients with chronic stroke who were classified according to motor

deficit using a Fugl-Meyer lower extremity score. The researchers measured lower limb joint torque while subjects generated maximum voluntary isometric contractions at hip, knee and ankle joints (in the direction of flexion-extension at the ankle, knee and hip, and abduction-adduction at the hip) while standing. Primary joint torques in the paretic lower extremity of subjects with stroke were lower than those in the control (neurologically intact) subjects for ankle flexion-extension, hip extension, hip adduction-abduction, and knee flexion. Examination of secondary torques, generated at other joints, showed that both groups used similar strategies to generate maximum torques during seven of the eight joint movements tested. The only joint movement for which the two groups used a different strategy was during maximal hip abduction exertion, in which subjects in the stroke group tended to flex the hip, but those in the control group tended to extend the hip. The EMG data of the stroke group was different from that of the control group, in that the former showed a strong presence of co-contraction of antagonistic muscle groups, especially during ankle flexion and ankle and knee extension.

Patients with stroke also may have difficulty activating muscles voluntarily. [6] Newham and Hsiao[6] assessed maximal voluntary activation of quadriceps and hamstrings muscles measured by isokinetic dynamometer in subjects who were less than 6 months post stroke. They found that the paretic muscles showed greater activation failure than the non-paretic: a 40% voluntary activation deficit (voluntary activation failure during an isometric maximal voluntary contraction) of knee extensors in the paretic lower limb and a 25% deficit of knee extensors in the non-paretic lower limb. Similarly, Miller et al[7] found that mean maximum voluntary contraction of the knee extensors of the paretic limb (85.4±45.5 Nm) was significantly lower ($P< 0.001$) than that of the non-paretic limb (154.9±50.2 Nm) in

patients with chronic stroke. In addition, the mean voluntary activation ratios (calculated by dividing the maximum voluntary contraction value by the maximum torque value) of the knee extensors in the non-paretic and paretic leg were 0.97±0.04 and 0.86±0.13, respectively. The authors concluded that the strength loss of the knee extensors was partially caused by muscle atrophy. Knee extensor muscle weakness on the paretic side in subjects with chronic stroke in this study may be only partially explained by reduced voluntary activation ability, indicating that other neuromuscular structural or functional factors may contribute to post-stroke muscle weakness.

In another study, Tyson et al[8] studied weakness (measured by Motricity Index score) of lower and upper extremity musculature on the paretic side in patients at least 21 days post stroke. Mean score for the lower extremity (69.1±33.6) was significantly greater than for the upper extremity (58.5±39.6) at $p<0.01$. Thirty percent of the subjects had lower extremity strength greater than upper extremity strength, and 17% showed the opposite pattern. Approximately 50% of the subjects had no difference in strength between lower and upper limbs. In addition, no significant differences in strength were found between proximal (shoulder and hip) and distal (hand and ankle) joints in either lower extremity or upper extremity. Furthermore, muscle strength was not significantly influenced by demographic variables or stroke pathology (side and type of stroke). LeBrasseur et al[9] also reported that long term impairments from stroke include deficits in strength (>30% loss) and power (>40% loss) in the paretic lower extremity compared with the non-paretic lower extremity. Patients with stroke may depend primarily on vision to compensate for motor control deficits in the lower extremity.[10]

Sensory impairment: Connell et al[11] reported that somatosensory impairment is associated with stroke severity in subjects with a mean time post stroke of 15 days. These researchers found that proprioception and stereognosis were more frequently impaired than tactile sensations. Significant recovery of tactile sensation, proprioception, and stereognosis occurred over six months after stroke for the upper limb, but not for the lower limb. Degree of initial somatosensory impairment was the strongest influence on somatosensory recovery at 6 months post stroke, accounting for between 46% and 71% of the variance in somatosensory recovery. The main conclusion from this study is that somatosensory impairment is an important factor associated with stroke severity.

In contrast to the previous study, Tyson et al[12] reported that tactile sensation was more frequently impaired than proprioception in subjects with first stroke who were 2-4 weeks post stroke. Sensation in this study was tested by the Rivermead Assessment of Somatosensory Perception (RASP) measurement tool. The leg was significantly more impaired than the arm for all tactile sensation modalities: overall tactile sensation ($p<.016$), tactile detection and tactile discrimination ($p<.038$ and $p<.026$, respectively). However, there was no difference in proprioception between arm and leg and no difference in any sensory modality between proximal and distal joints. The degree of weakness and degree of stroke severity were factors significantly influencing sensory impairment, whereas demographics, stroke pathology, and neglect were not significant contributors.

Neglect: Sensation impairment and neglect are significantly associated with weakness ($p<0.0001$ and $p<0.004$, respectively).[8] Neglect is most commonly associated with right hemisphere damage following both cortical and subcortical injury.[13] Unilateral neglect is most often described as a perceptual disorder and attentional deficit in which the individual

fails to report or respond or orient to sensory stimuli presented on the side opposite a brain lesion.[14] Siekierka-Kleiser et al[15] found that patients with motor hemineglect had larger stroke lesions than those without motor hemineglect. It has long been known that postural recovery is worse after right hemispheric strokes than after left hemispheric strokes, especially in patients displaying spatial neglect. Several studies reported that patients with spatial neglect standing on posturographic force-platforms showed a greater amount of sway and/or a greater weight bearing asymmetry than others.[16-18]

Cognitive impairment: Cognitive impairment frequently occurs after stroke and commonly involves memory, orientation, language, and attention.[19] The percent of people with stroke who are identified as being cognitively impaired may vary from 35% to 78%, depending on how cognitive impairment is defined.[19] Cognitive impairment appears most frequently with infarcts in the left anterior and posterior cerebral artery area and least frequently with infarcts in the vertebrobasilar artery area.[19] Patel et al[20] found that factors independently associated with cognitive impairment included age 75 or older (OR = 2.5, 95% confidence interval (CI) =1.5-4.2), ethnicity [Caribbean/African (OR = 1.9, 95% CI =1.2-3.2), Asian (OR = 3.4, 95%CI =1.1-10.2], lower socioeconomic class (OR = 2.1, 95%CI =1.3-3.3), left hemispheric lesion (OR = 1.6, 95%CI =1.01-2.4), and visual field deficit (OR = 2.0, 95%CI =1.2-3.2).

Relationship between Motor Impairments and Functional Performance

Individuals after stroke who scored lower on the Functional Independence Measure (FIM) locomotion and mobility tests had greater weight bearing difference between legs during STS.[21] Lomaglio et al[21] reported that the time required to complete STS in people with chronic stroke under self-paced conditions was associated with paretic knee extension

strength (r=-0.716) and ankle dorsiflexion strength (r=-0.450). Patients with chronic stroke who had lower motor performance scores displayed greater postural instability than those who had higher motor performance scores.[10]

Relationship between Sensory Impairments and Functional Performance

Overall sensory impairments at acute stage of stroke have been found to be correlated with functional mobility ($r = 0.515$, $p<.000$) and with independence in ADL ($r = 0.541$, $p<.000$).[12] Subcomponents of overall sensory impairment were also individually correlated with functional mobility: $r = 0.416$, $p<.000$ for proprioceptive impairment and $r = 0.500$, $p<.000$ for tactile sensory impairment, as well as with independence in ADL: $r = 0.456$, $p<.000$ for proprioceptive impairment and $r = 0.518$, $p<.001$for tactile sensory impairment, respectively. These associations remain present at 3-month follow-up.

Relationship between Neglect and Functional Performance

Ring et al[22] studied scores on the Functional Independence Measure (FIM) in patients admitted for stroke rehabilitation. They found that FIM scores in patients who had neglect were significantly lower than those without neglect, both at admission ($p< .01$) and at discharge from the hospital ($p<.05$). This suggested that people after stroke who had neglect, compared to those without neglect, may be less able to perform ADL necessary for functional independence.

Relationship between Cognitive Impairment and Functional Performance

The severity of functional impairment is significantly greater in patients with cognitive impairment than in patients without cognitive impairment.[19] Tatemichi et al[19] also found that the risk of dependent living at home or nursing home after discharge from the hospital was associated with cognitive impairment (Odds Ratio, OR = 2.4), age (OR= 5.2), and physical impairment (OR = 3.7). In another study, Patel et al[20] found that cognitively impaired patients compared to cognitively intact patients at 3 months after stroke were significantly more disabled at 1, 3, and 4 years ($p<.001$, $p<.001$, $p<.001$, respectively). These patients also had significant higher mortality at 1, 3, and 4 years ($p<.001$, $p<.05$, $p<.01$, respectively). In addition, institutionalization rates were higher in patients with cognitive impairment at all time points.

2. Effects of Stroke on Gait Pattern

2.1 Gait Characteristics after Stroke

Inability to walk and slow walking speed are important functional limitations after stroke. Sixty-five to seventy-five percent of stroke survivors are not able to walk independently in the acute phase. The recovery of walking ability usually occurs within 3-6 months.[23, 24] One third of patients admitted for acute care are still non-ambulatory at three months post stroke.[24] Among patients with stroke who recover the ability to walk, a decrease in walking speed is common, with as much as a 50% reduction compared to healthy adults.[25] Slow walking speed after stroke is associated with reduction in step length or cadence, leading to compensations to correct these deficiencies.

Common gait deviations after stroke include foot drop, circumduction, and knee hyperextension.[26] Foot drop is caused by weakness of the ankle dorsiflexors and/or hypertonia of the plantarflexors.[26] An individual with foot drop might show hip hiking or excessive hip and knee flexion to clear the foot during swing phase.[27] On the other hand, weakness, stiffness, and abnormal muscle tone in the paretic leg may lead to decreased knee flexion during swing. In this circumstance, with the knee and ankle remaining stiff, the person advances the paretic leg by circumduction of the hip. The legs may also cross, leading to a scissor gait.[26] Knee hyperextension typically occurs during stance phase, and is associated with weakness of the knee extensor muscles. In addition, diminished sensation in the paretic leg may result in difficulty feeling the position of the leg during movement. Therefore, the knee is locked or hyper-extended to stabilize the paretic leg during stance.[26]

Gait in individuals following stroke is characterized by reduced speed, cadence, stride length, and step length. Several researchers have published studies to describe temporal-spatial gait characteristics after stroke.[28-32] Mean gait speed in people following stroke has been variously reported in the literature: 0.2±0.16 m/sec,[30] 0.33 m/s (95% CI, 0.24-0.43m/sec),[31] 0.38±0.17 m/sec,[28] 0.41±0.27 m/sec,[32] and 0.62±0.21 m/sec.[29] The variety of gait speeds after stroke may be due not only to variations in time since stroke onset, but also to other factors, such as stroke severity and duration of rehabilitation period. Reports of mean cadence in people after stroke range from 66.03±15.37 steps/min[32] to 84.5±14.20 steps/min[29] for comfortable speed, with a cadence of 103.3±19.40 steps/min[29] reported for subjects instructed to walk at fast speed. Mean step length on the paretic side has been reported to be as short as 0.30 m (95% CI, 0.23-0.38 m)[31] and, for walking at fast speed, as long as 0.55±0.12 m.[29]

Restoration of gait and reduction of gait deviations are major goals for both patients and therapists in stroke rehabilitation. Residual gait deficits can diminish quality of life after stroke.[33]

2.2 Intervention to Improve Gait

Residual disabilities in individuals with chronic stroke (at least six months post stroke) include walking disability (30%) and dependence in ADL (26%).[2] Poor walking ability has been found to reduce quality of life and limit participation in activities outside the home.[34] After stroke, most people cannot walk fast enough to across the road safely or far enough to do the shopping.[35] Recovery of independent walking is a critical part of the rehabilitation process to establish early quality walking.[36] Intervention to improve walking capacity can improve general well-being by promoting better health and greater community participation.[37]

Evidence suggests that gait training programs improve walking ability in individuals after stroke. Researchers have shown that home-based strengthening and flexibility exercises,[38] treadmill (TM) training combined with over ground walking training (OG),[39, 40] TM training combined with conventional physical therapy (CPT),[41, 42] and TM training with body weight support (BWS) and a variety of speeds[40, 42, 43] can significantly improve walking speed in people with chronic stroke.

Lower extremity strengthening exercises

Home-based strengthening and flexibility exercises[38] and strengthening exercise training of the paretic leg[44] can significantly improve walking speed in people with chronic

stroke.[38, 44] In a study by Monger et al[38], a home-based training program included repetitive STS training, step-up exercises, and calf stretching. Training sessions lasted 20 min a day, three days a week, for three weeks. Mean walking speed significantly increased from 0.86±.31 m/s at pretest to 1.10±.41 m/s at posttest ($p<.01$). However, the researchers did not test for retention of these effects.

Dean et al[44] used an exercise training protocol for people with chronic stroke that included walking on a treadmill, repetitive STS with body weight evenly distributed on both legs, reaching while sitting at a table, heel lifting in standing, stepping, and reciprocal leg flexion and extension to strengthen paretic leg muscles. The training sessions lasted one hour a day, three times a week, for four weeks. Mean walking speed increased from 0.58±0.51 m/s at pretest to 0.71±0.48 m/s at post test ($p<.05$) and 0.79±0.48 m/s at 2-month follow up ($p<.05$) in the training group. A control group that received only strengthening exercise for the paretic upper limb showed no improvement in gait speed. These results suggest that the combination of lower extremity strengthening exercises and treadmill training can produce gains in walking speed that are maintained over time.[39]

Treadmill training or physical therapy that focuses on gait-specific activities appears to be more effective than conventional therapy alone in promoting recovery of locomotion after stroke. In addition, TM training with BWS appears to be more effective than TM training performed without any weight support. Treadmill training using a variety of speeds can also improve walking speed in people post stroke.[40, 43]

Combination of TM and OG training

Treadmill training plus OG training can produce greater improvements in walking speed than conventional physical therapy alone. Gains in walking speed can be maintained over time with this training combination.[39] Ada et al[39] found that walking speed significantly increased immediately after training and at 3-month follow-up in people with chronic stroke who initially had a walking speed of less than 1.2 m/s. This study consisted of two groups: a TM plus OG group and a control group. The control group received general strengthening exercise at home. For the TM plus OG group, the proportion of TM walking to OG walking was decreased by 10% each week from 80% in the 1st week to 50% in the 4th week. The OG training focused on walking forward, backward, sideways, and up/down stairs and slopes. The training lasted 30-45 min, three times a week, for four weeks. Mean walking speed in the training group significantly increased from 0.62±0.24 m/s at pretest to 0.75±0.26 m/s at post test (p=.02) and 0.83±0.26 m/s at 3-month follow-up. These findings suggest that TM plus OG training can produce lasting improvements in walking speed.

Combination of TM training and CPT

Increased walking speed also has been found following TM training plus CPT in people with chronic stroke. The improvement in walking speed was independent of the order in which the TM training and CPT interventions were given.[41] In a study using a crossover design, Liston et al[41] compared the effects of TM training and CPT in people who had cerebrovascular disease with higher-level gait disorders. The training program consisted of a total of eight weeks of gait training, with four weeks of TM training and four weeks of CPT. One group of subjects received TM first, and the other group received CPT first. During TM

training, subjects were instructed to continue walking for as long as they felt comfortable. The CPT component consisted of over ground walking training, including an emphasis on turning and maintaining good postural alignment. Training sessions were 60 minutes/session, three times a week, for four weeks. Walking speed in both groups improved from pre- to immediate post-testing, and this improvement was maintained at 6-week follow-up. No significant between-group differences were found, although the researchers observed a trend for therapy to be most effective when TM retraining was the first modality used to improve walking ability.

Combination of TM training with BWS and CPT compared to TM training alone

Conventional physical therapy with over ground training may add benefits beyond TM training alone.[42] Werner et al[42] studied the effects of TM training with BWS performed with and without CPT in people with chronic non-ambulatory stroke. The first group received 30-minute sessions of TM training with BWS combined with 40-minute sessions of CPT. The second group received 30-minute sessions of TM training with BWS only. The TM speed was 0.21 m/s at the beginning of training, and was increased to 0.27 m/s during training. Body weight support at the beginning of training was 27 %BW, and was gradually reduced to 0 %BW to enable full loading of the lower limbs. Conventional physical therapy involved application of neurodevelopmental techniques (Bobath concept), including practice of sitting, standing, and gait activities. Training sessions were conducted five days a week for three weeks. Mean walking speed significantly increased from 0.20±0.06 m/s at pretest to 0.33±0.10 m/s at post test ($p<.001$) and 0.31±0.12 m/s at 4-month follow-up in the TM plus CPT group, and from 0.22±0.05 m/s at pretest to 0.30±0.09 m/s at post test ($p<.001$) and

0.31±0.09 m/s at 4-month follow-up in the TM only group. After 3-week training program, TM training plus CPT group effected a larger improvement for gait ability compared with the TM training only group. Because the TM plus CPT group received double the amount of therapy received by the TM only group, differences in outcomes may be attributable to differences in amount rather than type of training.

Treadmill training with BWS at a variety of speeds

Treadmill training with BWS appears to be more effective than TM training without any weight support.[45] Using a variety of speeds during TM training with BWS can produce significant improvements in walking speed immediately after training and at follow-up testing.[40, 43] Furthermore, greater increases in walking speed have been reported with higher speeds of TM training in people with chronic stroke.[43] In a study by Pohl et al[40], the effects of TM training with BWS were compared to those obtained by use of OG training in ambulatory patients who were more than 4 weeks post stroke. All subjects received 45-minute sessions of CPT and were divided into three groups. Two groups received 30-minute sessions of TM training with BWS, with one being a speed-dependent TM training group (STT) and the other being a limited progressive TM training group (LTT). Training sessions for the STT group included a 5-minute warm-up, 1-2 minutes of gradually increasing –TM speed, and 10 sec of TM walking at maximum speed. For the LTT group, training speed was increased by no more than 5% of maximum initial walking speed each week (maximum 20% over 4 weeks). For both the STT and LTT groups, BWS with an overhead harness was no more than 10% of the patient's body weight. Subjects in the third group received 45-minute sessions of over ground walking training. Mean walking speed significantly increased at the

end of training for all groups: from 0.61±0.32 m/s at pretest to 1.63±0.80 m/s at posttest ($p<.001$) in the STT group, from 0.66±0.39 m/s at pretest to 1.22±0.74 m/s at posttest ($p<.001$) in the LTT group, and from 0.66±0.42 m/s at pretest to 0.97±0.64 m/s at posttest ($p<.001$) in the OG training group. The STT group had greater improvement in walking speed than the other two groups ($p<.001$). The LTT group was similar to the OG training group in walking speed improvement. These results suggest that TM training at higher speeds may produce larger increases in gait speed. Unfortunately, the researchers did not assess long-term effects.

Similar to Pohl et al[40], Sullivan et al[43] found that TM training with BWS could significantly increase walking speed in people with chronic stroke living in the community. These researchers compared TM training with BWS at different speeds. Subjects in this study were divided into three groups: slow speed (0.5 mph), fast speed (2.0 mph) and variable speed (0.5, 1.0, 1.5, 2.0 mph). Training sessions for each group lasted 20 minutes, three times a week, for four weeks. During the training phase, subjects did not receive any physical training such as over ground ambulation or endurance training. Mean walking speed increased significantly for all three groups ($p<.001$) at posttest, and these gains were maintained at 3-month follow-up.

After stroke, many interventions can improve walking speed immediately and long term, including lower extremity strengthening exercises, TM plus OG training, TM training plus CPT, and TM training with BWS at a variety of speeds. Treadmill training with BWS can also facilitate independent walking and paretic lower extremity loading during walking. The greatest benefits appear to result from fast speed TM training .To promote long term

improvements in walking ability, therefore, physical therapists may need to incorporate not only conventional OG training, but also TM training with BWS at different speeds.

2.3 Tests and Measures of Gait

Observational gait analysis is regularly performed by physical therapists to determine treatment goals and to evaluate progress during rehabilitation. Clinically and scientifically robust measurement tools are needed to assess gait performance after stroke and changes following interventions, In particular, measurement tools must be reliable, where reliability refers to the consistency of measurements and the relative absence of measurement errors. A variety of gait performance tests have been used in stroke patients. Intra-rater reliability of several of the tests has been reported for stroke patients.[46, 47]

Green et al[47] assessed the within-assessment and between-assessment reliability of gait speed at home in patients with mobility problems at least one year post stroke. Gait speed was measured as the number of seconds needed to walk 10 meters at comfortable speed during two assessments one week apart. Intraclass correlation coefficients (ICCs) were 0.95-0.99 for within-assessment reliability, and 0.87-0.88 for between-assessment reliability.

Flansbjer et al[46] evaluated the intra-rater (between occasions) reliability of gait at comfortable and fast speeds in individuals with mild to moderate chronic stroke. Subjects were tested on 2 occasions at 7 days apart. The ICC values were 0.94 (95% confidence interval [CI] 0.90–0.97) for comfortable gait speed and0.97 (95% CI, 0.95–0.98) for fast gait speed.

3. Effects of Stroke on Sit-to-Stand Performance

3.1 Phases of Sit-to-Stand (STS)

Standing up from a chair is one of the most common functional tasks, and the ability to stand up without assistance is important for independent living. After stroke, many people have difficulty rising from a chair independently. Some demonstrate asymmetry of body-weight distribution during rising to stand, with more weight bearing through the non-paretic than the paretic lower extremity. Furthermore, individuals with stroke often demonstrate increased time to complete the movement of rising to stand. Further understanding of STS performance in people recovering from stroke will enable us to distinguish how their movement patterns differ from those of their peers without stroke.

Schenkman et al[48] described the STS movement as having 4 phases. Phase 1: Flexion-momentum phase; begins at initiation of movement and ends just before the buttocks are lifted from the chair seat. During this phase, the center of mass is within the base of support, and eccentric contractions of the erector spinae muscles are required to control forward motion of the trunk. Trunk forward flexion generates upper-body momentum, while the femurs, shanks, and feet are still stationary. Vertical projection of the body's center of mass remains over the base of support (buttocks on seat). Phase 2: Momentum-Transfer phase; begins as the subject leaves the chair seat and ends with maximum ankle dorsiflexion. The trunk remains flexed, and momentum of the upper body is transferred from a purely anterior (forward) direction to both anterior and upward. Anterior movement of the center of mass reaches a maximum close to the time of maximum ankle dorsiflexion. Momentum is transferred from the upper body to the whole body as the base of support changes from the chair to the feet. The maximum angles of hip flexion, trunk flexion, and ankle dorsiflexion

are reached. Maximum hip and knee torques are achieved at the same time at which the individual first becomes fully weight-bearing and while the hip and knee are close to maximum flexion.

Phase 3: Extension phase; begins after maximum ankle dorsiflexion and continues with the hip reaching full extension and the knee reaching almost full extension. The stability requirements in this phase are not as great as in the momentum-transfer phase because the COM is well within the base of support of the feet. Therefore, this phase is mechanically distinct from the first 2 phases because the major task of this phase is to translate the body vertically while in a stable position. Phase 4: Stabilization phase; begins after hip extension velocity reaches 0^0/sec and continues until all motion associated with stabilization from rising is completed. The end point of this phase is not easily defined because the individual normally experiences some anterior-posterior and lateral sway during quiet stance. In Schenkman's study, the mean time to complete all 4 phases of STS was 1.95 sec. The mean times to complete each phase of STS were 0.5±0.08 sec for the flexion-momentum phase (phase 1), 0.33±0.08 sec for the momentum-transfer phase (phase 2), and 0.98±0.2 sec for the extension phase (phase 3).

Other studies have categorized phases of the STS movement in different ways. Millington et al[49] and Cheng et al[50] defined three STS movement phases. In a study of older adults (age range 65-76 years) with no known pathology, Millington et al[49] defined STS movement phases by the onset of muscle activity. Muscles activated in the weight shift phase (phase 1) were: erector spinae, rectus femoris, and vastus medialis; in the transition phase (phase 2): biceps femoris, gluteus maximus, and rectus abdominis; and in the lift phase (phase 3): rectus femoris, gluteus maximus, and biceps femoris. In Millington's study, the

mean time to complete STS was 2.03±0.34 sec (range = 1.62-2.54 sec). The characterization of STS motion in this study can help clinicians identify problems in elderly patients who have difficulty rising from a chair.

Cheng et al 2004[50] studied the STS task in patients with stroke according to changes in vertical ground reaction forces (vGRFs) measured by force plates. The initiative phase (phase 1) begins when the vGRF decreases at the start of trunk flexion. The executive phase (phase 2) begins with the sharpest rise in vGRF and ends when peak vertical momentum is achieved. The standing phase (phase 3) begins at the moment of peak vertical momentum and ends with the stabilization of the total body vertical force on the force plates, when the subject achieves stable standing.

These studies provide information about kinematic, kinetic and electromyographic aspects of the STS movement. Taken together, they provide a comprehensive understanding of how the task is performed. Description of STS phases facilitates communication among clinicians and identification of why patients may have difficulty performing the task.

3.2 Characteristics of STS Performance after Stroke

Because the STS task is important for everyday function, impairment of STS performance may lead to loss of independence in people who have had a stroke. Many investigators have reported impaired STS performance after stroke. Mean peak vGRF beneath the paretic lower extremity during STS in individuals post stroke is less than that in healthy subjects.[51, 52] Other STS measures, such as time to complete STS, mean difference in body weight (BW) distribution, and BW distribution ratio between lower limbs during STS are different for individuals post stroke compared to healthy subjects.[51, 52] Furthermore,

among individuals who are post stroke, values of some of these measures are lower for those with a history of falls compared to those with no history of falls.[50, 51]

People post stroke demonstrate greater variability in their sequence of leg muscle activation during STS than do healthy subjects.[50] The order of muscle activation in most subjects with no known pathology is as follows: tibialis anterior (TA), quadriceps (QUA), hamstrings (HAM), and soleus (SOL) muscles. In contrast, the SOL and HAM muscles activate almost simultaneously with TA and QUA muscles on the paretic side in people post stroke. In some people post stroke, especially those with a history of falls, the SOL muscle is activated before the TA and QUA muscles.[50] Many of these same individuals also exhibit QUA and TA muscle activations in the non-paretic leg earlier than in the paretic leg. Cheng et al[50] reported that 70% of stroke fallers exhibited no or low-amplitude activity of the TA muscle on the paretic side during STS. In addition, 50% of stroke fallers exhibited premature or excessive activation of the paretic SOL muscle during STS.

Engardt and Olsson[52] reported that BW distribution during rising from a chair in patients with stroke was less symmetrical than in healthy adults. The BW distribution ratio between paretic and non-paretic legs during rising from a chair was 0.60 in patients with stroke and 0.99 in healthy adults. In addition, the peak vGRF beneath the paretic leg in patients with stroke (37.5%BW) was less than in healthy adults (49.7%BW) in this study.[52]

Cheng et al[51] used force plates to perform kinetic analysis during STS in stroke fallers, stroke non-fallers, and healthy subjects. The mean time needed to complete STS for each of these groups was 4.32±2.22 sec, 2.73±1.19 sec, and 1.88±0.48 sec, respectively. Mean peak vGRF for each of these groups was 103.26±6.49%BW, 107.19±8.75%BW, and 114.32±9.06%BW, respectively. Mean difference in BW distribution between paretic and

non-paretic legs while rising from a chair was 52.87±18.42%BW in stroke fallers, 41.86±20.87%BW in stroke non-fallers, and 17.41±5.96%BW in healthy subjects. In addition, center of pressure (COP) displacement in the mediolateral (ML) direction during STS was much greater in patients with falls than in patients without falls or healthy subjects.

3.3 Interventions to Improve STS Performance

The inability to rise from a seated to a standing position can limit independent function during activities of daily living. Hence, the rehabilitation of STS movement is a critical goal after stroke. To promote evidence-based practice, knowledge about the effectiveness of interventions to improve STS performance following stroke is important.

Many investigators have reported on the effects of intervention programs to improve STS performance in people post stroke. The interventions included use of biofeedback and repetitive practice,[53, 54] strengthening of paretic leg muscles,[44] home-based strengthening and flexibility exercises,[38] and changes in foot placement during STS.[55] Most studies reported that significant improvement occurred in some STS measures. Positive effects of these interventions included improved symmetry of BW distribution during STS,[53, 54] increased peak vGRF beneath the paretic lower extremity,[44] decreased time to peak vGRF,[38] and decreased time to complete the STS task.[53]

Biofeedback and repetitive STS practice

Researchers who have implemented training programs involving repetitive practice of the STS task often have used biofeedback to try to improve symmetry of BW distribution. In one study by Engardt & Olsson,[54] subjects who were one week to three months post stroke

performed a training program with auditory biofeedback three times a day, five days a week, for six weeks. In this study, BW distribution was computed as the ratio between the time integrals of the vertical ground reaction forces of the paretic and non-paretic legs. Mean BW distribution on the paretic leg during STS significantly increased from 34.7%BW to 47.8%BW, and mean BW distribution ratio between paretic and non-paretic legs significantly increased from 0.55±0.18 to 0.95±0.25. A control group that practiced without auditory feedback also improved, but to a lesser extent, with an increase in mean BW distribution on the paretic leg from 39.0%BW to 44.0%BW, and an increase in mean BW distribution ratio between paretic and non-paretic legs from 0.66±0.17 to 0.81±0.18. These improvements were not maintained over time. In a follow-up study by the same researchers,[56] mean BW distribution on the paretic leg during STS significantly decreased from immediate post-tests to re-testing performed an average of 33.2±6.6 months after the training period. Values for the paretic leg decreased from 47.8±6.7%BW to 38.7±7.1%BW ($p<0.001$) in the training group and from 44.0±6.6%BW to 39.5±7.0%BW ($p<0.05$) in the control group[56].

Cheng et al[53] reported significant improvements in several STS measures, including symmetrical BW distribution, COP displacement, and time to complete the STS task, after training in individuals who were 2-4 months post stroke. The training program consisted of 30 minutes of symmetrical standing practice and 20 minutes of repetitive STS practice with feedback, including use of a postural correction mirror and a dual force platform with real-time visual and auditory feedback. Training sessions lasted a total of 50 minutes a day and continued for five days a week for three weeks. The mean difference in BW distribution between the paretic and non-paretic legs during STS decreased significantly from

49.5±18.9%BW at initial testing to 38.6±15.8%BW at 6-month follow-up (p<.005). Mean COP displacements during STS also decreased significantly (p<.05) in the mediolateral direction (from 10.9±5.0 cm to 7.8 ± 4.2 cm) and anteroposterior direction (from 10.8±4.1 cm to 8.8±3.0 cm). Furthermore, subjects with stroke in the Cheng et al[53] study demonstrated a significant decrease in the time required to complete the STS task, from 4.1 ± 1.3 sec at initial testing to 2.7 ± 1.1 sec at 6-month follow-up (p<.001).

Lower extremity strengthening exercises

Two similar studies, one by Dean et al[44] and the other by Monger et al,[38] examined the effects of strengthening exercises on STS performance in individuals with chronic stroke. The training programs included exercises such as repetitive STS practice, heel lifting and reciprocal leg flexion and extension in standing, step-ups and other stepping exercises, calf stretching, and walking on a treadmill. Training sessions were three times a week for at least three weeks. A significant increase in mean peak vGRF beneath the paretic lower extremity (from 48.6±8.8%BW at pretest to 62.2±7.8%BW at posttest; p< .05) was found after training for subjects in the Dean et al[44] study, but not in the Monger et al[38] study. The lack of an effect in the latter study may be attributable to shorter duration of each training session (20 minutes, as compared to 60 minutes in the Dean et al[44] study) or the exercise setting (home-based, as compared to clinic in the Dean et al study). Monger et al[38] , however, did report a significant decrease in mean TTP vGRF through the paretic leg during STS, from 0.12±0.03 sec before training to 0.09±0.02 sec at post-test (p<.05).

Foot placement during STS

Brunt et al[55] reported that specific changes in foot placement during STS increased paretic lower limb muscle activity and decreased the difference in peak vGRF between paretic and non-paretic lower limbs in individuals with chronic stroke. Subjects in this study were asked to perform the STS task under three different conditions: 1) normal condition: both legs were placed in 100^0 knee flexion; 2) limb-extended condition: non-paretic leg was extended to 75^0 knee flexion; 3) limb-elevated condition: non-paretic leg was placed on a dense foam support. Mean peak vGRF was greater beneath the non-paretic than the paretic leg during STS under normal and limb-elevated conditions, but not in the limb-extended condition. In the limb-elevated and -extended conditions, electromyographic amplitudes of the quadriceps and tibialis anterior muscles on the paretic side increased significantly compared to the normal condition. These findings suggest that placement of the non-paretic foot in a position relatively anterior to the paretic foot can encourage more symmetrical lower extremity loading during STS.[55]

Many intervention studies demonstrate improvements in symmetrical BW distribution during STS in people after stroke. However, intervention effects may not be sustained long term because many of the interventions are short in duration (e.g., three weeks). Furthermore, interventions performed at home may not have sufficient intensity or be sustained over a sufficient period of time to achieve a therapeutic threshold. To maintain long term improvements in BW distribution during STS, therefore, type, intensity, and duration of the training program are important considerations.

3.4 Tests and Measures of Sit-to-Stand Performance

The STS test is used for multiple purposes, including as an indicator of postural control, fall risk, lower extremity strength, and physical function. A number of different measures of STS performance have been used in both clinical and research settings. As with any measures of physical performance, reliability and validity are critical psychometric properties.

Cheng et al[51] examined COP displacement in people post stroke during rising from a chair by use of two force plates (AMTI force platform). The researchers found that COP displacement in the mediolateral direction during STS was much greater in stroke fallers than stroke non-fallers.[51]

Yamada and Demura[57] examined test–retest reliability of vGRF measurement during STS and the relationships between vGRF parameters and knee extension muscle strength using a force platform (GRAVICORDER G5500; Anima, Japan). The researchers found that the reliability of vGRF parameters was high, with ICCs of 0.70 to 0.95. The vGRF at hip-off and knee-hip extension phase was significantly correlated with knee extension strength (r = 0.29–0.64).

In another study, Yamada and Demura[58] examined the test-retest reliability of vGRF and EMG parameters in young adults while rising from a chair. Intraclass correlation coefficients (ICCs) for peak EMG amplitudes of rectus femoris and tibialis anterior muscles during STS in this study ranged from 0.55 to 0.88. The researchers reported that the ICC for vGRF was 0.95.

Usuda and Yamaji59 examined the test-retest reliability of EMG analysis of STS in healthy subjects who had a mean age of 20±0.5 years. Subjects performed two trials of STS

at each of five different chair heights. The test-retest reliability of peak EMG of rectus femoris and tibialis anterior muscles increased with decreasing chair height (ICCs = 0.68 - 0.96). This study suggested that both muscles have an important role in moving the body's center of mass forward during STS. Furthermore, the reliability of the EMG activity was high.

Researchers have investigated both the reliability and the validity of the five-times-STS-test (FTSST). FTSST is a simple clinical measure of STS performance in which subjects rise from a chair five times as fast as possible with their arms folded across their chests. The examiner records the time required (in seconds) to complete the 5 chair stands. Lord et al[60] reported the test-retest reliability of the FTSST in 30 older people as ICC = 0.89 (95% CI = .79–.95). Whitney et al[61] described the discriminative and concurrent validity of the FTSST by comparing this measure with scores on the Activities-specific Balance Confidence Scale (ABC) and the Dynamic Gait Index (DGI) in people with balance disorders. People with balance disorders performed the FTSST more slowly than those without balance disorders. The discriminative analysis revealed that the FTSST correctly identified 65% of subjects with balance dysfunction, the ABC identified 80%, and the DGI identified 78%. The ability of the FTSST to identify people with balance dysfunction was better for people younger than 60 years of age (81%). The concurrent validity of the FTSST was supported by a Spearman rho of –0.68 (P<.001) between the FTSST and the DGI and of –0.58 (P<.001) between the FTSST and the ABC. Jones et al[62] reported the test-retest reliability and the construct validity of a 30-s chair stand as a measure of lower body strength in older adults. The test score is the number of chair stands completed in 30 seconds. The ICC for the 30-s chair-stand measure was 0.95 (95% CI = .84-.97). Construct validity of the chair stand was demonstrated by the

test's ability to detect differences between various age and physical activity level groups. Chair-stand performance decreased significantly across age groups in decades--from the 60s to the 70s to the 80s ($p < .01$) and was significantly lower for low-active participants than for high-active participants ($p < .0001$). These results suggest that the 30-s chair stand provides a reasonably reliable and valid indicator of lower body strength in older adults.

McCarthy et al[63] examined relationships between muscle strength and STS performance in older adults. Muscle strength was assessed isometrically, and STS performance was assessed by use of the FTSST and 30-second chair stand. Both the FTSST and 30-second chair STS test presented high degrees of stability of testing ($r = .95$, $p = .0001$ and $r = .93$, $p = .0001$, respectively). Muscle strength explained 48% and 35% of the variance in FTSST scores and 30-second chair STS scores, respectively. Ankle plantar flexor, hip flexor, and knee extensor strength were the strongest predictors for both STS tests.

4. Relationships between STS Performance and Functional Ability after Stroke

The ability to perform STS is an important component of functional ambulation. The STS task has been associated with multiple variables, including postural control during standing, walking ability, independence in ADL, lower-extremity motor function, and fall risk. Asymmetrical lower extremity weight bearing during STS may affect functional performance in patients following stroke.

4.1 ADL and Gait

The ability to transfer BW onto the paretic limb while rising from a chair is indicative of walking performance. Chair rise (3 repetitions) has been shown to correlate with gait

speed (r=.54).[64] STS control is also related to ambulation ability. Chou et al[65] found that people with chronic stroke who could stand up within 4.5 sec or who had a vGRF difference of less than 30%BW between paretic and non-paretic legs during STS had better gait performance (velocity, cadence, stride time, single support).[65]

4.2 Functional Independence Measure

Weight-bearing ability correlates with functional performance in individuals with stroke. The degree of weight-bearing asymmetry during rising from a chair has been correlated with motor function and level of self-care independence. Degree of paresis correlated with weight bearing differences between lower extremities during the STS task. (Lee 1997). Stroke patients who scored lower on the Functional Independence Measure (FIM) locomotion and mobility tests had greater weight bearing differences.[66] In another study, Engardt et al[54] found that symmetry of body-weight distribution during rising from a chair is positively correlated ($p<0.001$) with scores on the lower extremity motor scale of the Fugl-Meyer Assessment ($r = 0.54$), motor function in STS as assessed by the Motor Assessment Scale (MAS, 1-6 points) ($r = 0.73$), and functional ability as assessed by the Barthel Index ($r = 0.53$).[54]

4.3 Balance and Falls

Asymmetrical body-weight distribution during STS may be an indicator of increased risk of falls in individuals with stroke.[51] Cheng et al[50] reported that 70% of stroke fallers exhibited no or low-amplitude activity in the tibialis anterior muscle of the paretic leg during STS. In addition, 50% of stroke fallers exhibited premature or excessive activation of the

soleus muscle on the paretic side during STS. At the 6-month follow-up, the number of falls was lower in the training group (16.7%) compared to the control group (41.7%) ($p<.05$).[53] In another study, Dean and Shepherd[67] reported that improved loading of the paretic limb during STS after a 2-week intervention was associated with improved ability to perform the STS task[67] and increased standing symmetry.[68] In another study, Eng et al[69] reported a high correlation between paretic limb loading during STS and standing balance, $r = 0.739$ at $p<.001$.

SUMMARY

Impairments after stroke may involve the motor, sensory, perceptual, and/or cognitive systems. These impairments, in turn, affect functional abilities after stroke. Many people who are post stroke have difficulty shifting weight onto the paretic lower extremity during functional tasks, such as rising from a chair and walking. Asymmetrical BW distribution during STS is associated with slower walking speed and longer time to complete the STS task. Various interventions have been shown to improve STS performance after stroke, and associated improvements in functional abilities, such as reaching while in a sitting position, are sometimes observed.

Because STS performance has been related to other functional abilities and to risk of falls, understanding of how this performance changes during stroke recovery is important. The STS task has been divided into different phases, depending on the type of measurement used. Kinetic, or force platform, data are used commonly to investigate paretic lower extremity weight bearing or "loading" during STS. Deficits in paretic lower extremity loading do not appear to resolve completely in many individuals who are post stroke.

REFERENCES

1. Heart Disease and Stroke Statistics: 2009 Update: *American Heart Association*. 2009.

2. Kelly-Hayes M, Beiser A, Kase CS, Scaramucci A, D'Agostino RB, Wolf PA. The influence of gender and age on disability following ischemic stroke: the Framingham study. *J Stroke Cerebrovasc Dis.* May-Jun 2003;12(3):119-126.

3. Benaim C, Perennou DA, Villy J, Rousseaux M, Pelissier JY. Validation of a standardized assessment of postural control in stroke patients: the Postural Assessment Scale for Stroke Patients (PASS). *Stroke.* Sep 1999;30(9):1862-1868.

4. Smith MT, Baer GD. Achievement of simple mobility milestones after stroke. *Arch Phys Med Rehabil.* Apr 1999;80(4):442-447.

5. Neckel N, Pelliccio M, Nichols D, Hidler J. Quantification of functional weakness and abnormal synergy patterns in the lower limb of individuals with chronic stroke. *J Neuroeng Rehabil.* 2006;3:17.

6. Newham DJ, Hsiao SF. Knee muscle isometric strength, voluntary activation and antagonist co-contraction in the first six months after stroke. *Disabil Rehabil.* Jun 15 2001;23(9):379-386.

7. Miller M, Flansbjer UB, Lexell J. Voluntary activation of the knee extensors in chronic poststroke subjects. *Am J Phys Med Rehabil.* Apr 2009;88(4):286-291.

8. Tyson SF, Chillala J, Hanley M, Selley AB, Tallis RC. Distribution of weakness in the upper and lower limbs post-stroke. *Disabil Rehabil.* Jun 15 2006;28(11):715-719.

9. LeBrasseur NK, Sayers SP, Ouellette MM, Fielding RA. Muscle impairments and behavioral factors mediate functional limitations and disability following stroke. *Phys Ther.* Oct 2006;86(10):1342-1350.

10. Corriveau H, Hebert R, Raiche M, Prince F. Evaluation of postural stability in the elderly with stroke. *Arch Phys Med Rehabil.* Jul 2004;85(7):1095-1101.

11. Connell LA, Lincoln NB, Radford KA. Somatosensory impairment after stroke: frequency of different deficits and their recovery. *Clin Rehabil.* Aug 2008;22(8):758-767.

12. Tyson SF, Hanley M, Chillala J, Selley AB, Tallis RC. Sensory loss in hospital-admitted people with stroke: characteristics, associated factors, and relationship with function. *Neurorehabil Neural Repair.* Mar-Apr 2008;22(2):166-172.

13. Robertson IH, Marshall JC. Unilateral neglect: clinical and experimental studies. Hove [England]; Hillsdale [N.J.]: L. Erlbaum Associates; 1993.

14. Heilman KM, Valenstein E. *Clinical neuropsychology*. 2nd ed. New York: Oxford University Press; 1985.

15. Siekierka-Kleiser EM, Kleiser R, Wohlschlager AM, Freund HJ, Seitz RJ. Quantitative assessment of recovery from motor hemineglect in acute stroke patients. *Cerebrovasc Dis.* 2006;21(5-6):307-314.

16. Bonan IV, Colle FM, Guichard JP, et al. Reliance on visual information after stroke. Part I: Balance on dynamic posturography. *Arch Phys Med Rehabil.* Feb 2004;85(2):268-273.

17. de Haart M, Geurts AC, Huidekoper SC, Fasotti L, van Limbeek J. Recovery of standing balance in postacute stroke patients: a rehabilitation cohort study. *Arch Phys Med Rehabil.* Jun 2004;85(6):886-895.

18. Laufer Y, Sivan D, Schwarzmann R, Sprecher E. Standing balance and functional recovery of patients with right and left hemiparesis in the early stages of rehabilitation. *Neurorehabil Neural Repair.* Dec 2003;17(4):207-213.

19. Tatemichi TK, Desmond DW, Stern Y, Paik M, Sano M, Bagiella E. Cognitive impairment after stroke: frequency, patterns, and relationship to functional abilities. *J Neurol Neurosurg Psychiatry.* Feb 1994;57(2):202-207.

20. Patel MD, Coshall C, Rudd AG, Wolfe CD. Cognitive impairment after stroke: clinical determinants and its associations with long-term stroke outcomes. *J Am Geriatr Soc.* Apr 2002;50(4):700-706.

21. Lomaglio MJ, Eng JJ. Muscle strength and weight-bearing symmetry relate to sit-to-stand performance in individuals with stroke. *Gait Posture.* Oct 2005;22(2):126-131.

22. Ring H, Feder M, Schwartz J, Samuels G. Functional measures of first-stroke rehabilitation inpatients: usefulness of the Functional Independence Measure total score with a clinical rationale. *Arch Phys Med Rehabil.* Jun 1997;78(6):630-635.

23. Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients: the Copenhagen Stroke Study. *Arch Phys Med Rehabil.* Jan 1995;76(1):27-32.

24. Wade DT, Wood VA, Heller A, Maggs J, Langton Hewer R. Walking after stroke: Measurement and recovery over the first 3 months. *Scand J Rehabil Med.* 1987;19(1):25-30.

25. Shumway-Cook A, Woollacott MH. Motor control: theory and practical applications. Baltimore: Williams & Wilkins; 1995.

26. Perry J. Gait analysis: normal and pathological function. Thorofare, N.J.: SLACK Incorporated; 1992.

27. Sandra J. Olney CR. Hemiparetic gait following stroke. Part I: Characteristics. *Gait & Posture.* 1996;4(2):136-148.

28. Dunsky A, Dickstein R, Marcovitz E, Levy S, Deutsch JE. Home-based motor imagery training for gait rehabilitation of people with chronic poststroke hemiparesis. *Arch Phys Med Rehabil.* Aug 2008;89(8):1580-1588.

29. 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.* Aug 2003;84(8):1185-1193.

30. Laufer Y, Dickstein R, Chefez Y, Marcovitz E. The effect of treadmill training on the ambulation of stroke survivors in the early stages of rehabilitation: a randomized study. *J Rehabil Res Dev.* Jan-Feb 2001;38(1):69-78.

31. Regnaux JP, Pradon D, Roche N, Robertson J, Bussel B, Dobkin B. Effects of loading the unaffected limb for one session of locomotor training on laboratory measures of gait in stroke. *Clin Biomech (Bristol, Avon).* Jul 2008;23(6):762-768.

32. Trueblood PR. Partial body weight treadmill training in persons with chronic stroke. *NeuroRehabilitation.* 2001;16(3):141-153.

33. 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.* Jul 2001;82(7):879-884.

34. Solomon NA, Glick HA, Russo CJ, Lee J, Schulman KA. Patient preferences for stroke outcomes. *Stroke.* Sep 1994;25(9):1721-1725.

35. 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(3):173-180.

36. Ada L, Dean CM, Morris ME. Supported treadmill training to establish walking in non-ambulatory patients early after stroke. *BMC Neurol.* 2007;7:29.

37. Ada L, Dean CM, Lindley R, Lloyd G. Improving community ambulation after stroke: the AMBULATE Trial. *BMC Neurol.* 2009;9:8.

38. Monger C, Carr JH, Fowler V. Evaluation of a home-based exercise and training programme to improve sit-to-stand in patients with chronic stroke. *Clin Rehabil.* Jun 2002;16(4):361-367.

39. Ada L, Dean CM, Hall JM, Bampton J, Crompton S. A treadmill and overground walking program improves walking in persons residing in the community after stroke: a placebo-controlled, randomized trial. *Arch Phys Med Rehabil.* 2003;84(10):1486-1491.

40. Pohl M, Mehrholz J, Ritschel C, Ruckriem S. Speed-dependent treadmill training in ambulatory hemiparetic stroke patients: a randomized controlled trial. *Stroke.* Feb 2002;33(2):553-558.

41. Liston R, Mickelborough J, Harris B, Hann AW, Tallis RC. Conventional physiotherapy and treadmill re-training for higher-level gait disorders in cerebrovascular disease. *Age Ageing.* Jul 2000;29(4):311-318.

42. Werner C, Bardeleben A, Mauritz KH, Kirker S, Hesse S. Treadmill training with partial body weight support and physiotherapy in stroke patients: a preliminary comparison. *Eur J Neurol.* Nov 2002;9(6):639-644.

43. Sullivan KJ, Knowlton BJ, Dobkin BH. Step training with body weight support: effect of treadmill speed and practice paradigms on post stroke locomotor recovery. *Arch Phys Med Rehabil.* May 2002;83(5):683-691.

44. Dean CM, Richards CL, Malouin F. Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. *Arch Phys Med Rehabil.* Apr 2000;81(4):409-417.

45. Visintin M, Barbeau H, Korner-Bitensky N, Mayo NE. A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. *Stroke.* Jun 1998;29(6):1122-1128.

46. Flansbjer UB, Holmback AM, Downham D, Patten C, Lexell J. Reliability of gait performance tests in men and women with hemiparesis after stroke. *J Rehabil Med.* Mar 2005;37(2):75-82.

47. Green J, Forster A, Young J. Reliability of gait speed measured by a timed walking test in patients one year after stroke. *Clin Rehabil.* May 2002;16(3):306-314.

48. Schenkman M, Berger RA, Riley PO, Mann RW, Hodge WA. Whole-body movements during rising to standing from sitting. *Phys Ther.* Oct 1990;70(10):638-648; discussion 648-651.

49. Millington PJ, Myklebust BM, Shambes GM. Biomechanical analysis of the sit-to-stand motion in elderly persons. *Arch Phys Med Rehabil.* Jul 1992;73(7):609-617.

50. Cheng PT, Chen CL, Wang CM, Hong WH. Leg muscle activation patterns of sit-to-stand movement in stroke patients. *Am J Phys Med Rehabil.* Jan 2004;83(1):10-16.

51. Cheng PT, Liaw MY, Wong MK, Tang FT, Lee MY, Lin PS. The sit-to-stand movement in stroke patients and its correlation with falling. *Arch Phys Med Rehabil.* Sep 1998;79(9):1043-1046.

52. Engardt M, Olsson E. Body weight-bearing while rising and sitting down in patients with stroke. *Scand J Rehabil Med.* 1992;24(2):67-74.

53. Cheng PT, Wu SH, Liaw MY, Wong AM, Tang FT. Symmetrical body-weight distribution training in stroke patients and its effect on fall prevention. *Arch Phys Med Rehabil.* Dec 2001;82(12):1650-1654.

54. Engardt M, Ribbe T, Olsson E. Vertical ground reaction force feedback to enhance stroke patients' symmetrical body-weight distribution while rising/sitting down. *Scand J Rehabil Med.* Mar 1993;25(1):41-48.

55. Brunt D, Greenberg B, Wankadia S, Trimble MA, Shechtman O. The effect of foot placement on sit to stand in healthy young subjects and patients with hemiplegia. *Arch Phys Med Rehabil.* Jul 2002;83(7):924-929.

56. Engardt M. Long-term effects of auditory feedback training on relearned symmetrical body weight distribution in stroke patients. A follow-up study. *Scand J Rehabil Med.* Jun 1994;26(2):65-69.

57. Yamada T, Demura S. The relationship of force output characteristics during a sit-to-stand movement with lower limb muscle mass and knee joint extension in the elderly. *Arch Gerontol Geriatr*. 2009 (In Press).

58. Yamada T, Demura S. Influence of instruction on reliability and magnitude of evaluation parameters in each phase of a sit-to-stand movement. *Percept Mot Skills.* 2005;101:695–706.

59. Usuda S, Yamaji T. Reliability of computer-based video motion analysis and electromyographic analysis of sit-to-stand movement. *J Phys Ther Sci.* 1996;8:49-54.

60. Lord SR, Murray SM, Chapman K, Munro B, Tiedemann A. Sit-to-stand performance depends on sensation, speed, balance, and psychological status in addition to strength in older people. *J Gerontol Med Sci.* 2002;57:M539-M543.

61. Whitney SL, Wrisley DM, Marchetti GF. Clinical measurement of sit-to-stand performance in people with balance disorders: validity of data for the Five-Times-Sit-to-Stand Test. *Phys Ther*. 2005;85:1034 –1045.

62. Jones CJ, Rikli RE, Beam WC. A 30-s chair-stand test as a measure of lower body strength in community-residing older adults. *Res Q Exerc Sport.* 1999 Jun;70(2):113-119.

63. McCarthy EK, Horvat MA, Holtsberg PA, Wisenbaker JM. Repeated chair stands as a measure of lower limb strength in sexagenarian women. *J Gerontol Med Sci*. 2004; 59(10):1207–1212

64. Thapa PB, Gideon P, Fought RL, Kormicki M, Ray WA. Comparison of clinical and biomechanical measures of balance and mobility in elderly nursing home residents. *J Am Geriatr Soc.* May 1994;42(5):493-500.

65. Chou SW, Wong AM, Leong CP, Hong WS, Tang FT, Lin TH. Postural control during sit-to stand and gait in stroke patients. *Am J Phys Med Rehabil.* Jan 2003;82(1):42-47.

66. Lee MY, Wong MK, Tang FT, Cheng PT, Lin PS. Comparison of balance responses and motor patterns during sit-to-stand task with functional mobility in stroke patients. *Am J Phys Med Rehabil.* Sep-Oct 1997;76(5):401-410.

67. Dean CM, Shepherd RB. Task-related training improves performance of seated reaching tasks after stroke: a randomized controlled trial. *Stroke.* Apr 1997;28(4):722-728.

68. Winstein CJ, Gardner ER, McNeal DR, Barto PS, Nicholson DE. Standing balance training: effect on balance and locomotion in hemiparetic adults. *Arch Phys Med Rehabil.* 1989;70(10):755-762.

69. Eng JJ, Chu KS. Reliability and comparison of weight-bearing ability during standing tasks for individuals with chronic stroke. *Arch Phys Med Rehabil*. 2002;83(8):1138-1144.