

EFFECTS OF MUSCLE STRENGTHENING AND PHYSICAL CONDITIONING TRAINING ON TEMPORAL, KINEMATIC AND KINETIC VARIABLES DURING GAIT IN CHRONIC STROKE SURVIVORS

Luci Fuscaldi Teixeira-Salmela,¹ Sylvie Nadeau,² Ian McBride³ and Sandra Jean Olney³

From the ¹Universidade Federal de Minas Gerais, Brazil, ²University of Montreal, Quebec, ³School of Rehabilitation Therapy, Queen's University, Kingston, Ontario, Canada

The purpose of this study was to evaluate the impact of a combined program of muscle strengthening and physical conditioning on gait performance in subjects with chronic stroke, using a single group pre- and post-test design. Thirteen subjects were recruited for the 10-week program (3 days/week), which consisted of warm-up, aerobic exercises, lower extremity muscle strengthening and cool-down. Data from cinematographic film and a force plate obtained during multiple walking trials were used in a four-segment kinetic model to yield spatiotemporal, kinematic and kinetic variables. Gait analysis revealed that the 10 week training resulted in significant increases in gait speed associated with improvements in walking patterns as determined by increases in selected kinematic and kinetic measures. After training, subjects were able to generate higher levels of powers and demonstrated increases in positive work performed by the ankle plantar flexor and hip flexor/extensor muscles.

Key words: stroke, gait, biomechanics, muscle strength, physical conditioning.

J Rehab Med 2001; 33: 53–60

Correspondence address: Dr. Sylvie Nadeau, Professeure Adjointe, University of Montreal, Faculty of Medicine, School of Rehabilitation, C.P. 6128, Succursale Centre-ville, Montreal (Quebec), H3C 3J7 Canada

(Accepted July 7, 2000)

INTRODUCTION

Initial walking function is impaired in two-thirds of the stroke population and this deficit is the greatest contributor to functional disability after stroke (1). A single common pattern of hemiplegic gait does not exist and disturbances of the temporal, spatial and kinematic patterns are well documented (2, 3). The self-selected gait speed is a well-known indicator of overall gait performance and has been commonly used to monitor performance and evaluate the effects of treatment in stroke rehabilitation (4). However, when used alone, gait speed assists in neither understanding the nature of the gait deficiencies nor directing future training. By the same token, while the use of kinematic profiles is well known, it is generally agreed that they yield little information about the mechanisms underlying abnormal movement patterns (5). More recently, kinetic analyses for the diagnosis of pathological data have provided strategies for diagnosing the causes of abnormal motor patterns observed in

pathological gait profiles (2, 3, 5). Since kinetic variables are causal factors in kinematic and spatiotemporal outcomes of gait, this information is particularly useful in understanding not only abnormal gait patterns, but also the causes underlying improvements in performance (2). Measurement of mechanical work and power performed by major muscle groups has been used to predict gait performance in stroke subjects (3). Both the push-off power burst of the ankle plantar flexors and the pull-off burst of the hip flexors are of particular interest (2, 3) because of their importance in the required power generation for walking.

Muscle weakness has been implicated as a factor underlying deficits in gait performance in subjects with stroke (6). Muscle strength and associated declines in functional performance related to normal ageing can be ameliorated through strengthening programs and also may prove useful in promoting long-term independence for both stroke subjects and the elderly population (7). Previous studies have revealed the positive effects of strength training on gait speed for subjects with chronic stroke (4). Endurance training has also been recognized as an important component in rehabilitation. The effects of a combined strength and aerobic conditioning program have improved strength, muscular endurance and functional performance in the elderly (8). A previous randomized controlled trial study demonstrated the benefits of such a program in reducing impairment and disability in chronic stroke subjects (9). This study further assessed the effects of the training on gait performance in subjects with chronic stroke using spatiotemporal, kinematic and kinetic analyses, including joint moments, joint powers and work. It was expected that gains in gait speed would be associated with changes in kinematic profiles, higher joint moments and higher levels of power produced by major lower extremity muscle groups.

SUBJECTS AND METHODS

Subjects

Thirteen volunteer subjects with unilateral stroke, who had residual weakness and/or spasticity of the affected lower extremity, were recruited from a local stroke club and through newspaper and cable television advertisements. All subjects were screened to ensure that their mean time since onset of stroke was at least 9 months, were independently ambulatory with or without assistive devices for 15 minutes, had an activity tolerance of 45 minutes with rest intervals, and had no comprehensive aphasia. Those with non-stroke-related disabilities were excluded. Subjects were also required to obtain their physicians' consent to participate in the program and underwent dobutamine stress echocardiography (10) to rule out evident relative cardiac risk prior to program entry. Eligible subjects provided consent prior to their screening test based

on ethical approval that was previously obtained from the local research review board. Twenty age-matched healthy subjects were also recruited to provide reference values.

Design

A single group pre- and post-test group design was employed. Subjects participated in the 10-week training program immediately after the baseline tests, after which they were retested.

Procedure

Demographic data were collected on all subjects to document age, time since onset of stroke and side affected, as well as use of orthoses, walking aids and medication. Outcome measures regarding gait performance (spatiotemporal, kinematic and kinetic variables) were obtained through gait analysis at baseline and immediately after training.

Gait assessment

The Peak Motus 2D motion analysis system (Peak Performance Technologies, Englewood, CO, U.S.A.) with an AMTI force platform (Advanced Medical Technologies, Newton, MA, U.S.A.) were used to obtain kinematic and kinetic information. Data collection consisted of videotaping subjects walking in their own low-heeled shoes along an 8 m walkway containing an embedded force platform of standardized size. Three walking trials for each side were recorded at comfortable speed for a total of six trials. The subjects were also allowed to hold a straight cane or to touch an attendant's hand if extra guidance was required. The healthy subjects walked at a normal, slow and very slow speeds for three trials at each speed.

Prior to data collection, reflective markers were placed on the following camera-side landmarks for joint positional information: lateral aspect of the neck at the level of C₇, greater trochanter, lateral epicondyle of the femur, ankle joint center and head of the fifth metatarsal. Two background markers attached to the middle of the walkway provided a level ground reference. Videotaping of each subject was conducted using a super VHS videocamera (Hitachi VM-6100A SVHS, Japan) set at a shutter speed of 1/1000 with a picture rate of 60 fields/second and located 480 cm from the walkway which operated perpendicular to the plane of movement.

A video synchronization unit was used to synchronize the video image and force-plate data. Force platform data, which operated in a voltage range of ± 10 V, were sampled at a rate of 1200 Hz and then converted to digital form using the Peak Performance Technologies A/D board with a range of ± 4096 units and stored on a desk computer. A light was recorded on the videotape to synchronize the digitizing software of the force-plate data. When combined with video data, information from the force plate permitted calculation of the vertical and fore-aft shear ground reaction forces and the center of pressure of the force vector. Video images were digitized using the Peak Motus software. At least 10 additional fields before the initial foot contact and after the final foot contact were digitized to provide data for the filtering process. The following events were identified: initial foot contact, opposite toe-off, opposite foot contact, toe-off and final foot contact. The coordinate data were digitally filtered using a second-order Butterworth filter with the cut-off frequency set at 5 Hz associated with the optimal method outlined by Jackson (11), whereas the force-plate data were also filtered with the cut-off frequency of 10 Hz.

A standard four-segment link segment model was used in an inverse dynamic analysis with anthropometric values recommended by Winter (12). The kinematic variables included relative angles of the hip, knee and ankle. The kinetic data contained the net moments, powers and work around each joints. Net joint powers (P) were calculated for each instant in time as the product of net moment across the joint (M) and the relative angular velocity between the adjacent limb segments (ω). The integrals of positive and negative portions of net joint power curves yielded positive and negative work performed across each joint. All kinetic data were normalized to body mass and all data across each stride were normalized to 10 points corresponding to each 1% of the gait cycle to facilitate ensemble averaging. Kinematic and kinetic profiles before and after training were compared and plotted against the normal elderly profiles walking at similar speeds.

Training program

The training program consisted of supervised exercise sessions by an exercise physiologist and a physiotherapist conducted on three mornings/

week for 10 weeks. Each session lasted between 60 and 90 minutes and all sessions were accompanied by appropriate music relevant for the participants' age. All missed sessions were made up at a later date unless continuation was not possible for medical or personal reasons. Heart rates (Polar Vantage XL heart rate monitor, Polar CIC, Port Washington, NY, U.S.A.), blood pressure and rating of perceived exertion were continuously monitored and recorded at rest, immediately after each aerobic exercise mode and post-exercise. The subjects were given a list of exercises that they could perform at home and were encouraged to do so at least three times/week. Each supervised training session included: (i) a 5–10 minute warm-up consisting of calisthenics, mild stretching and range of motion exercises; (ii) aerobic exercises, consisting of graded walking, plus stepping or cycling each at a target heart rate of 70% of the maximal heart rate attained from the exercise test; (iii) strength training; and (iv) a cool-down period, consisting of 5–10 minutes of muscular relaxation and stretching exercises. Emphasis was on stretching the trunk and lower extremity muscles. Attendance was monitored to ensure completion of all 30 sessions.

Aerobic conditioning was similar to methods previously recommended for healthy elderly adults, with appropriate modifications. During the first 5 weeks of the program, walking intensity was increased from 50% to 70% of aerobic working capacity to elicit the target heart rate and rating of perceived exertion monitored by Borg's 10-point psychometric scale (13). The walking duration was increased from 10 to 20 minutes/session, as were the duration and intensity for the stepping and cycling activities. Exercise intensity and duration were kept constant, at 70% and 20 minutes, respectively, during the second 5 weeks of the program, while 1–2 minute rest intervals were allowed when necessary. The strength training protocol used was an adaptation of standardized rehabilitation principles of progressive-resistance training, using isometric, concentric and eccentric muscle contractions with elderly subjects (14). The strength exercises were performed for about 30 minutes for hip flexors, extensors and abductors, knee flexors and extensors, and ankle dorsiflexors and plantar flexors. Apart from using body weight, sandbag weights and Therabands (Therabands, Hygienic Corporation, Akron, OH, U.S.A.) of eight different resistances, no special resistance equipment was employed. Programs were personally devised for each subject and 50% of the maximum weight that a subject could lift on a single occasion was used to set the load for the first week. Subjects were instructed to perform three sets of 10 repetitions for each exercise with a 1–2 minute rest period between sets. By the second week, or as subjects could tolerate, the load was increased to 80% of the single repetition maximum. This maximum repetition was reassessed every 2 weeks and the training stimulus was adjusted to keep the load at 80% of each subsequent maximum single repetition.

Data analysis

All statistical analyses were carried out using the software SPSS for Windows (Version 7.5, 1996, SPSS, Cary, NC, U.S.A.). Descriptive statistics and tests for normality (Shapiro-Wilk) were performed for all outcome variables. The training efficacy was assessed by paired Student's *t*-tests of outcome variables and determined by gains in gait speed. The gait data profiles at baseline and at post-training were compared (descriptively) with those of healthy elderly subjects walking at a similar speed (0.65 ± 0.19 m/second) and examined for training program effects.

RESULTS

Thirteen subjects (6 females, 7 males) completed all testing and training. Their age ranged from 54 to 80.2 years (mean \pm S.D. 67.7 ± 9.2 years) and their strokes had occurred between 1.0 and 34.1 (7.7 ± 9.4) years before testing. Seven had the left side of the body affected, whereas six had the right side. Five subjects used a standard or a quad cane for ambulation and five wore ankle-foot orthoses. All subjects were taking oral medication, primarily anticoagulants, β -blockers or antihypertensive drugs.

Effects of training on gait parameters

Gait speed at post-training averaged 0.76 ± 0.37 m/second,

Table I. Temporal and kinematic variables at baseline and at post-training (n = 13)

Variables	Unaffected side		Affected side	
	Baseline	Post-training	Baseline	Post-training
Spatiotemporal				
Cadence (steps/minute)*	84.2 ± 22.0	95.3 ± 24.4	84.3 ± 22.9	93.5 ± 24.6
Stride length (m)*	0.84 ± 0.33	0.93 ± 0.30	0.81 ± 0.36	0.93 ± 0.30
Double support (%)	34.8 ± 14.0	31.6 ± 8.8	33.8 ± 12.4	31.6 ± 12.4
Stance (%)	71.0 ± 8.6	69.1 ± 5.8	64.6 ± 7.6	63.1 ± 6.2
Symmetry ratio ^a	1.36 ± 0.43	1.22 ± 0.20	1.18 ± 0.24	1.20 ± 0.22
Kinematics (°)				
Max. plantar flexion ^b	-0.8 ± 6.9	-5.8 ± 6.5	-4.9 ± 8.3	-5.7 ± 8.6
Max. knee flexion ^b	58.0 ± 10.6	62.2 ± 9.0	39.8 ± 12.5	44.5 ± 13.1
Max. hip extension ^b	-1.0 ± 12.7	-4.0 ± 13.1	2.9 ± 12.8	1.9 ± 14.9

Data are means ± 1 S.D.

^a Symmetry ratio = time taken for the swing phase of affected leg/unaffected leg.

^b Positive values indicate ankle dorsiflexion, knee flexion and hip flexion.

* $p < 0.001$.

which was significantly faster than the speed observed at baseline (0.60 ± 0.39 m/second), an improvement of 37.2%. Associated with improved speed, increases in cadence and stride length were observed, while stance time, double support time and symmetry ratio, defined as time taken for the swing phase affected/swing phase unaffected, remained unchanged (Table I). There was a trend towards an improvement in maximum plantar flexion angle during push-off and in knee flexion angle over the swing phase on both sides at post-training (Table I), as well as higher moment values and increases in power generation and positive work performed by the hip and ankle joints (Table II).

Effects of training on gait profiles

The joint-angle, joint-moment and joint-power profiles are shown in superimposed graphs in Figs. 1–3, respectively. Each figure represents the means of the profiles for both unaffected and affected limbs at baseline and at post-training compared with those of elderly subjects performing at a similar speed (0.65 ± 0.19 m/second). In general, the profiles were similar in shape to those of the healthy subjects but the amplitudes were lower and the events tended to occur earlier on the affected side and later on the unaffected side.

Joint-angle profiles

The hip profiles demonstrated a decrease in maximum hip extension during the late stance phase for both affected and unaffected sides at baseline (Fig. 1). On the affected side, this difference persisted at post-training. However, on the unaffected side, the post-training profiles showed an increase in hip extension during late stance and the timings of the curves were comparable to those of healthy elderly subjects. The knee profiles of the affected side at post-training revealed clear increases in knee flexion during swing phase, but the timing remained unchanged. For the unaffected side, the profiles at post-training indicated that both amplitude and timing reached similar values to those of the control group. At baseline, there was less than normal ankle plantar flexion at push-off and a shift towards late timing of events for both affected and unaffected sides. Post-

training profiles showed at push-off that the ankle moved into plantar flexion earlier and more steeply.

Joint-moment profiles

On average, the profiles on the affected side showed an increase in the peak ankle plantar flexor moment, and in the hip extensor and hip flexor moments at post-training (Fig. 2). The knee moment profile throughout the gait cycle showed, on average, a similar shape to the normal profile. The knee moment during early stance (0–10%) and at 50–100% of the gait cycle (swing phase) was similar to normal values. However, between 10 and 50% of the gait cycle (stance phase), the moment profile did not show a normal extension pattern and reversed into flexion during the last part of the stance phase. At post-training, the profile followed the same pattern, showing further increases in the flexor moment. On the unaffected side, the joint-moment profiles remained unchanged, except for the hip joint, which demonstrated an increase in the hip flexor moment.

Joint-power profiles

Lower than normal values of power bursts for all joints were seen for both the affected and unaffected sides at baseline, which were more pronounced at the ankle joint (Fig. 3). At baseline, hip power profiles of both affected and unaffected sides were similar to normal in shape and magnitude, with a minor shift in timing of the bursts. After training, there was a considerable increase in power generation by the hip extensors (H1) on the affected side and increases in both H1 and H3 power-generation phases on the unaffected side. The knee power profiles for both affected and unaffected sides at baseline were also similar to those of healthy subjects, except for an increase in power absorption at K3 and an earlier shift of events on the affected side. At post-training, the affected side profiles showed that the timing remained unchanged, but the amplitude reached normal values. However, on the unaffected side, the amplitudes of the power absorption phases (K3 and K4), which were already higher than normal at baseline, increased even more after training. The ankle power profiles of the affected side at baseline revealed both a decrease in

Table II. Moment, power and work variables at selected phases of the gait cycle at baseline and at post-training ($n = 13$)

Variables	Unaffected side		Affected side	
	Baseline	Post-training	Baseline	Post-training
Moments (Nm/kg)				
Max. ankle plantar flexion	1.17 ± 0.41	1.22 ± 0.48	0.91 ± 0.26	1.17 ± 0.32
Max. ankle dorsiflexion	-0.11 ± 0.12	-0.12 ± 0.10	-0.08 ± 0.10	-0.06 ± 0.06
Max. knee extension	0.50 ± 0.33	0.48 ± 0.23	0.32 ± 0.31	0.31 ± 0.26
Max. knee flexion	-0.20 ± 0.11	-0.23 ± 0.12	-0.25 ± 0.21	-0.37 ± 0.32
Max. hip extension	0.60 ± 0.17	0.71 ± 0.29	0.59 ± 0.23	0.70 ± 0.24
Max. hip flexion	-0.50 ± 0.43	-0.61 ± 0.24	-0.34 ± 0.22	-0.42 ± 0.27
Powers (W/kg)				
Max. ankle plantar flexion	2.28 ± 2.06	2.87 ± 2.02	0.97 ± 0.63	1.71 ± 1.06
Max. knee extension	0.34 ± 0.36	0.39 ± 0.29	0.29 ± 0.23	0.42 ± 0.33
Max. hip extension (H1)	0.57 ± 0.35	0.84 ± 0.77	0.48 ± 0.41	0.79 ± 0.61
Max. hip flexion (H3)	0.63 ± 0.45	0.90 ± 0.47	0.42 ± 0.31	0.51 ± 0.33
Work (J/kg)				
Positive ankle	0.22 ± 0.18	0.28 ± 0.19	0.11 ± 0.05	0.17 ± 0.09
Negative ankle	0.16 ± 0.08	0.17 ± 0.09	0.12 ± 0.06	0.16 ± 0.07
Positive knee	0.06 ± 0.06	0.05 ± 0.03	0.05 ± 0.03	0.07 ± 0.04
Negative knee	0.22 ± 0.14	0.30 ± 0.13	0.10 ± 0.08	0.14 ± 0.09
Positive hip	0.27 ± 0.13	0.31 ± 0.19	0.17 ± 0.10	0.26 ± 0.15
Negative hip	0.06 ± 0.06	0.10 ± 0.09	0.04 ± 0.05	0.04 ± 0.05

Data are means ± 1 S.D.

power generation (A2) and an earlier shift in the timing during the cycle. After training, the timing remained unchanged, but the amplitude of the power burst increased substantially, although it did not reach the values observed for healthy subjects. For the unaffected side, there were lower than normal values for power generation and a shift to later timing during the cycle at baseline. Post-training profiles indicated that both the amplitude and timing of the power burst reached normal values.

DISCUSSION

The present findings indicate that the 10 week combined program of muscle strengthening and aerobic conditioning resulted in significant improvements in gait velocity, cadence and stride length, and suggested improvements in walking patterns as determined by changes in selected kinematic and kinetic measures. The gait of the stroke subjects is characterized by low values for speed, cadence and stride length, and high values for cycle duration and double support phase, which supports the results of previous studies (15–17). The percentage of the total double support at baseline showed a trend towards lower values at post-training. However, the proportion of the gait cycle occupied by the stance phase remained unchanged. Increases in speed occurring without changes in interlimb symmetry also support research involving sub-acute and chronic stroke patients (17). Wall & Turnbull (17) also failed to show changes in gait symmetry after a 6-month strength training program with chronic stroke subjects. Such a program would be expected to increase subjects' ability to transfer weight through the paretic limb and improve gait symmetry, but this hypothesis was not supported. However, it must be remembered that these subjects walked more slowly than healthy subjects and they may have been more concerned with walking quickly at the expense of gait symmetry. Furthermore, since the present subjects attained most of their

recovery potential and established their abnormal gait pattern, the objective of improving symmetry would demand a change in their habitual gait pattern that would necessitate acquiring a new skill. Although gait symmetry is a main goal of physiotherapists working with stroke patients, there is no clear evidence that symmetry enhances performance. As found in the present study, symmetric patterns seem to be unrelated to the severity of hemiplegia (16) or to gait performance in chronic stroke subjects (16–18).

The major changes in joint angular profiles observed at post-training were represented by overall increases in ankle plantar flexion during late stance, bilateral knee flexion during swing and hip extension in late stance on the unaffected side. Increases in hip extension in late stance phase may be functionally important because these changes are associated with moving the trunk forward over the stance foot, thus providing the hip flexors with better mechanical advantage to generate power to pull-off the limb, resulting in a larger contralateral step length and an increase in speed. The magnitude of hip extension in late stance has been previously reported to be positively related to walking speed (2). The angular excursion of the knee on the affected side showed two distinct variations from normal profiles. It was usually more flexed at initial contact and peak knee flexion during mid-swing phase was less than that of healthy subjects. These findings are consistent with other reports (3) and are likely to be attributable to reduced levels of power generation and/or quadriceps spasticity. At post-training, the only noticeable change was the peak knee flexion during mid-swing, which increased considerably but was less than that of healthy subjects. It is likely that improvements were associated with increases in power generation by the hip and ankle muscles, resulting in increases in gait speed and peak knee flexion during swing (3). The ankle profiles for both sides demonstrated less peak plantar flexion during push-off than in healthy subjects and this deficiency improved after training.

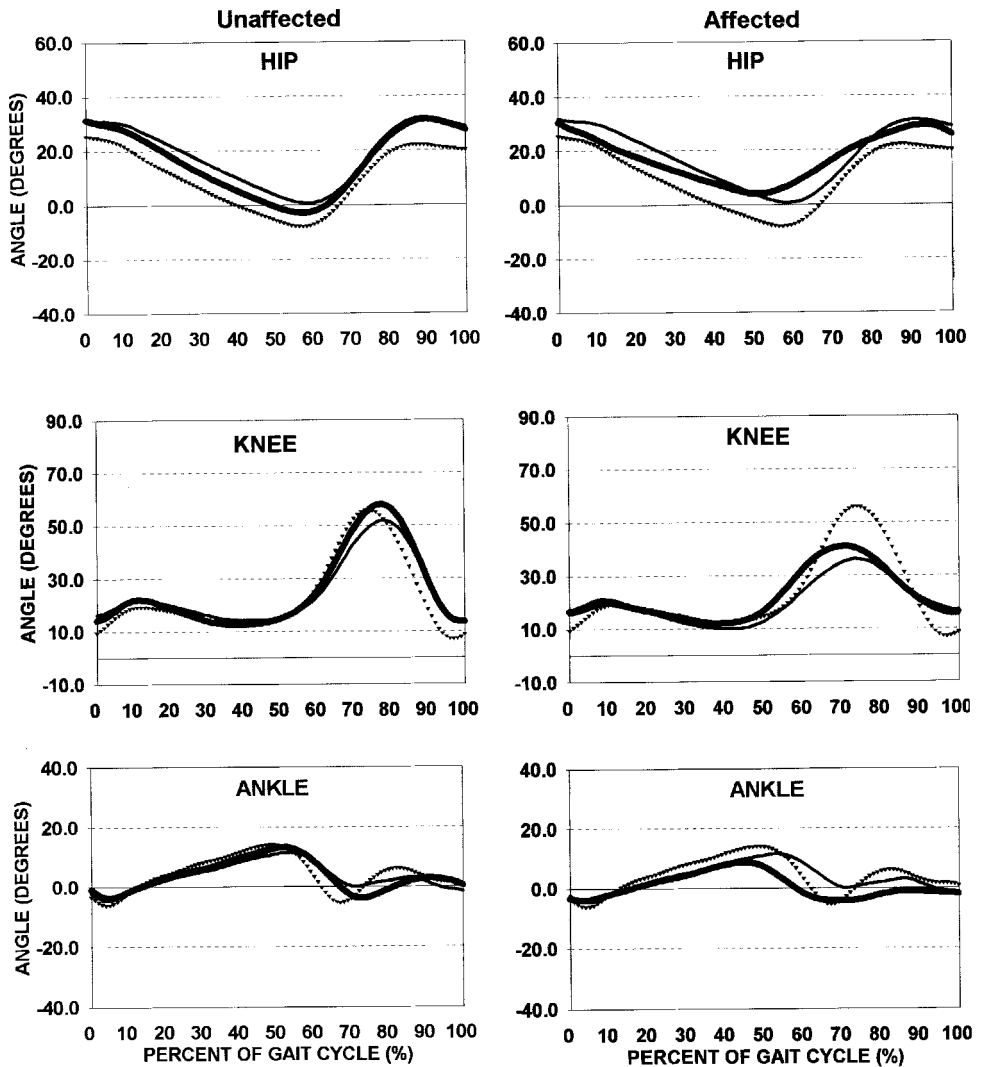


Fig. 1. Joint-angle profiles ($^{\circ}$) of the hip, knee and ankle joints of the stroke subjects at baseline (thin line) and at post-training (thick line) in comparison with healthy subjects walking at a similar mean speed (dotted line). Positive values indicate hip flexion, knee flexion and ankle dorsiflexion.

Profiles at post-training revealed increases in maximum ankle plantar flexion over the stance phase for both sides.

Reports of kinetic variables for stroke subjects which assess changes associated with therapeutic intervention are scarce. These findings are consistent with Winter (5), who reported that increases in hip moments were associated with faster cadences. It has been reported that fast walkers demonstrate, on average, exaggerated flexor profiles at the hip during late stance (15). These increases observed in late stance on both sides have been associated with changes in walking speed and high correlations between the affected peak hip flexor moment and the self-selected speed of walking (2). Significant correlations between walking speed and the unaffected peak hip extensor moment were also reported. Furthermore, the affected peak hip flexor moment accounted for 74% of the variance in walking speed. Since hip pull-off power is determined by the product of the hip flexor moment and the angular velocity of the hip joint, and such increases are a means of compensating for deficient ankle plantar flexor push-off power, it is reasonable to expect that a large flexor moment is simply a byproduct of this compensatory strategy.

The knee moment profiles during early stance and swing phases showed distinct differences between the affected and unaffected sides, and the latter were similar in both shape and timing to normal profiles and persisted after training. However, from mid- to late stance, the profile on the affected side showed a flexor moment, which increased even further at post-training. The results of Lehmann et al. (19) did not demonstrate a continuous flexor moment; however, the mean internal knee flexor moment was much larger in the stroke subjects than in healthy subjects walking at similar speeds. This high flexor moment was attributed either to the anterior orientation of the gravity line or to vertical forces related to the knee joint center (20), suggesting that increased hip flexion in mid-stance may have caused forward displacement of the center of gravity and its vertical force (19). The knee flexor moments may have been the result of kinematic abnormalities such as knee hyperextension patterns of hamstring overactivity for large extensor moments at the hip to balance for forward lean, or of the anterior displacement of the center of gravity associated with the hip flexion bias throughout the gait cycle. At baseline, the affected ankle moment profiles followed

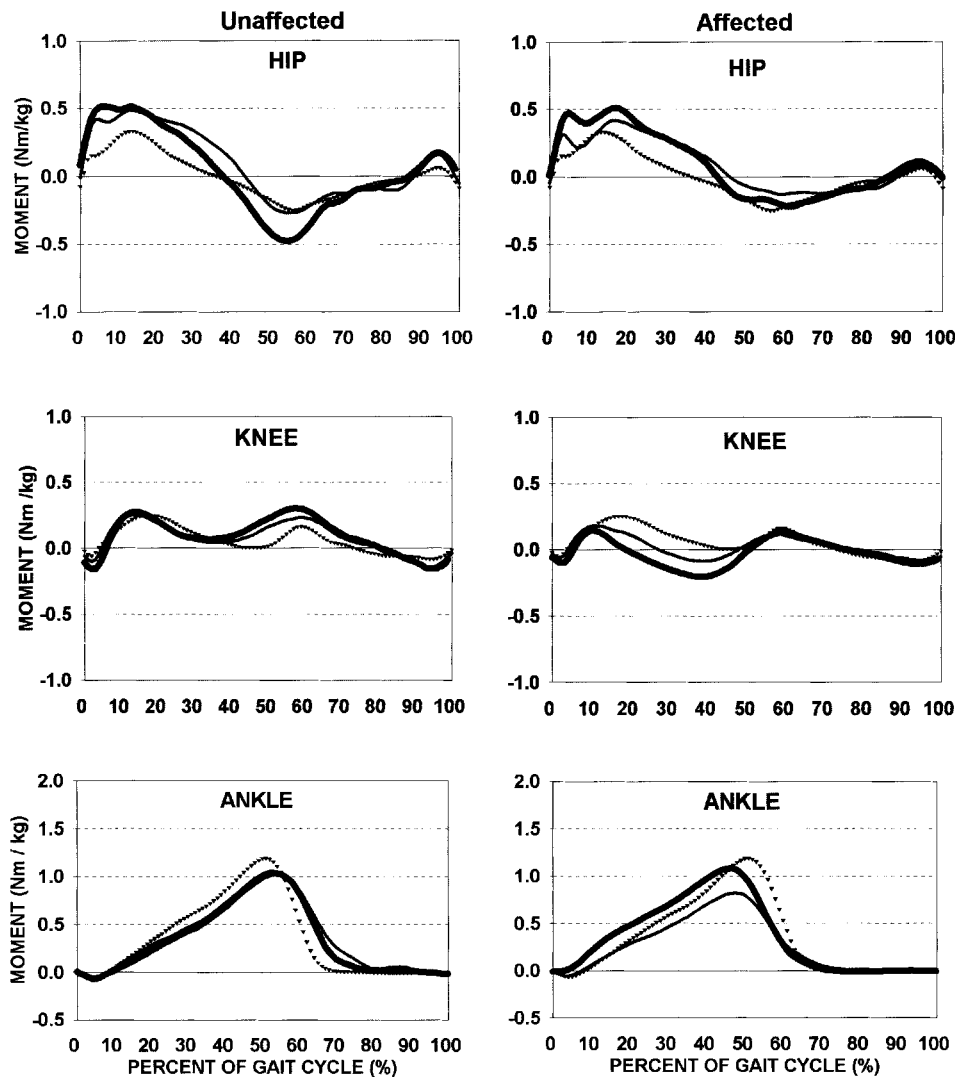


Fig. 2. Joint-moment profiles (Nm/kg) of the hip, knee and ankle joints of the stroke subjects at baseline (thin line) and at post-training (thick line) in comparison with healthy subjects walking at a similar mean speed (dotted line).

similar patterns to those of healthy subjects, but were smaller in magnitude. Increases in peak values indicated a more efficient push-off and thus increased speed and power generation (5). There was also an earlier shift in timing during the gait cycle, which tended to occur even earlier after training.

The most striking characteristics of the power profiles across all three joints of both the affected and unaffected sides were their similarities in shape compared with normal profiles (5), with major discrepancies in decreases in amplitude, indicating deficits in power generation or absorption. In addition, power bursts tended to occur later on the unaffected and earlier on the affected side, which is consistent with overall temporal abnormalities.

The three major reported power generators of the lower limb for A2, H3 and H1 (2, 5) have shown that there are important links between the performance of the muscle groups in the limb and between limbs (3, 5), which is consistent with the present findings. The major difference in the power profiles compared with those of Olney et al. (3) was related to the H1 power-generation phase. Certain compensations, as indicated by above-

normal profile values, were evident at H1 for both affected and unaffected sides. This adaptation by the hip extensors was a direct result of the high hip extension moment and is a compensation for the lack of ankle push-off power (5). In some cases, this increase in H1 may have been a postural adaptation, since some subjects demonstrated an increased forward lean, to help to increase the muscle length and enable the production of greater tension for forward propulsion.

Olney et al. (3) compared the peak power values for three groups of stroke subjects categorized as fast, medium and slow walkers. The power values on the affected side in the present study were lower than those reported earlier (8) and were paralleled with lower walking speed. Interestingly, the maximum power values for the unaffected ankle, knee and hip joints found in the present study were higher than those of the reported values (3), which may be indicative of a greater compensation by the unaffected side of the present subjects.

Mean ankle, knee and hip maximum powers for the affected side over the stride at baseline were lower than those reported for chronic stroke subjects (3). Power generation at the knee joint of

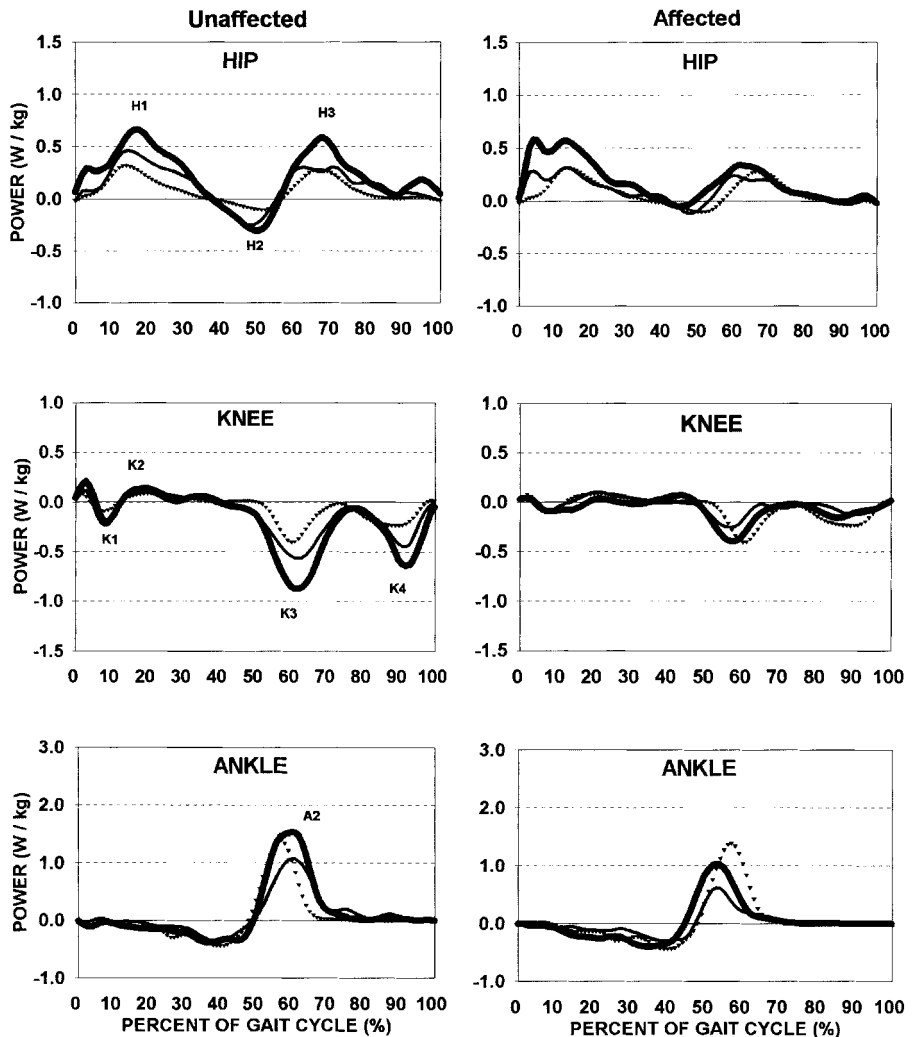


Fig. 3. Joint-power profiles (W/kg) of the hip, knee and ankle joints of the stroke subjects at baseline (thin line) and at post-training (thick line) in comparison with healthy subjects walking at a similar mean speed (dotted line).

both sides was minimal compared with that at the ankle and hip joints. On the affected side, K1 and K2 bursts were negligible, possibly owing to locking of the knee by the hip extensors. The knee muscle activity was mainly eccentric, indicating that the knee acted mainly by absorbing, rather than generating energy (3, 5). Interestingly, when comparing these power values with healthy subjects walking at a similar speed, the stroke subjects showed higher peak hip and lower peak ankle power values, indicating that power needs were met by using a larger proportion of hip and a smaller proportion of the ankle power than for the healthy subjects. It was evident that a trade-off between the hip and ankle joints existed, resulting in the use of differential compensatory strategies.

Present increases in the major bilateral hip power-generation phases were evident after training. Both the hip extensors during early stance and hip flexors at pull-off showed remarkable increases after training, with values reaching or surpassing those of healthy subjects. Although the affected side showed considerable increases in plantar flexor power, average peak values never reached healthy reference values and may reflect compensation for the affected side. Profiles at the knee reflected increases after

training only in K3 for the affected side and in K3 and K4 for the unaffected side. Although there were substantial gains in power generation by both H1 and H3 bilaterally, increases in H1 were more evident on the affected side, and in H3 on the unaffected side. It is likely that owing to functional weaknesses, the affected hip flexors could not generate enough power to pull-off the limb and, thus, the hip extensors were engaged to activate and initiate forward propulsion.

These increases in power generation by the major muscle groups were indicative of improvement in gait performance after training. The extent to which the ankle plantar flexors contributed to positive work was consistent between baseline and post-training measures and was somewhat lower than other findings (3). At the hip, both H1 and H3 contributed significantly to the positive work. For the affected side, the most significant actions were performed by the hip extensors in early stance, whereas for the unaffected side, the activity of both hip extensors during early stance and flexors at pull-off was dominant. This finding somewhat disagrees with others (3) who reported that the most important hip power-generation phase was performed by the hip flexors at pull-off. In the present study, H3 also played a

considerable role, but less than that of the hip extensor, especially on the affected side.

The negative work supplied by the knee (K3), which has a large eccentric phase at push-off, has also been reported to be highly correlated with walking speed (3), indicating that faster walkers flex their knees at termination of the stance, while weight is still on the foot. It appears that increased cadence and velocity are achieved by accelerating these gains so that the muscles generate and absorb energy at a faster rate (5).

Considering the power-generating role of the hip and ankle joints during gait, increases in H1, H2 and A2 work and power values observed at post-training indicated the effectiveness in enhancing performance of both the affected hip flexor and extensors and ankle plantar flexors. However, increased A2 power was not translated into reduced compensatory strategies adopted by the other joints of both sides. Both hip maximum powers, which were above normal values at baseline on the unaffected side, increased further after training on both sides and sooner on the unaffected side. These findings suggest the possible adoption of differential strategies to achieve higher levels of gait performance.

It is interesting to note that at baseline, 37% of the positive work was performed by the affected side, and this did not change substantially with the recorded increases in speed following treatment. At post-training, the amount of positive work performed by the affected side was similar to other reported research (3), where most of the positive work was performed by the affected side, regardless of gait competence. This finding challenges the assertion that improvement after stroke is largely attributed to increased use of the remaining unaffected musculature (3).

The findings of the present study have several implications for the field of the rehabilitation sciences. The results revealed that a combined program of muscle strengthening and physical conditioning proved beneficial for improving the gait performance of chronic stroke subjects. Moreover, the dependence of gait performance on the function of the affected limb suggests that greater consideration should be directed to the planning of exercises for stroke patients. The training of the ankle plantar flexors is of particular importance since it has been reported that they accomplish comparable gains for both the hip flexors and extensors (3). Although this finding is true for the unaffected side where the ratio of positive work between ankle and hip was similar at post-training, this was not the case for the affected side, which was different at post-training. This suggests that the power-generating capability of the hip flexors/extensors is functionally very relevant, since it was responsible for about 60% of the work performed on the affected side. However, the contribution of the ankle plantar flexors should not be underestimated because of the interplay between muscle group performance both within the same limb and between the two sides of the body. The present study supports evidence of a trade-off between ankle and hip muscles. However, the relevant mechanisms, and the nature, extent and degree to which adaptations can be developed require further investigation.

ACKNOWLEDGMENTS

Staff members of Stat Lab, Queen's University, are gratefully acknowledged for their assistance with the study design and data analysis. This research was supported by grant no. ST2714 from the Heart and Stroke Foundation of Ontario. L. F. Teixeira-Salmela was supported by a PhD studentship from CNPQ (Brazilian Government Agency) and S. Nadeau by fellowships from MRC and REPAR of Quebec.

REFERENCES

- Jorgensen H, Nakayama H, Raaschou H, Olsen T. Recovery of walking function in stroke patients: the Copenhagen study. *Arch Phys Med Rehabil* 1995; 76: 27–32.
- Olney S, Griffin M, McBride I. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. *Phys Ther* 1994; 74: 872–885.
- Olney S, Griffin M, Monga T, McBride I. Work and power in gait of stroke patients. *Arch Phys Med Rehabil* 1991; 67: 92–98.
- Sharp SA, Brouwer BJ. Isokinetic strength training of the hemiparetic knee: effects of function and spasticity. *Arch Phys Med Rehabil* 1997; 78: 1231–1236.
- Winter D. The biomechanics and motor control of human gait: normal, elderly, and pathological. Waterloo: University of Waterloo Press; 1991.
- Bohannon R, Andrews A. Correlation of knee extension torque and spasticity with gait speed in patients with stroke. *Arch Phys Med Rehabil* 1990; 71: 330–333.
- Nakamura E, Moritani T, Katenaka A. Effects of habitual exercise on physiological age in men aged 20–85 years as estimated using principal component analysis. *Eur J Appl Physiol* 1996; 73: 410–418.
- Sauvage L, Myklebust B, Crow-Pan J, Novack S, Millington P, Hoffman M, Hartz A, Rudman D. A clinical trial of strengthening and aerobic exercises to improve gait and balance in elderly male home residents. *Am J Phys Med Rehabil* 1992; 71: 333–342.
- Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B. Reducing impairment and disability in chronic stroke survivors through muscle strengthening and physical conditioning. *Arch Phys Med Rehabil* 1999; 8: 1211–1218.
- Sawada S, Segar D, Ryan T. Echocardiographic detection of coronary artery disease during dobutamine infusion. *Circulation* 1991; 83: 1605–1614.
- Jackson KM. Fitting of mathematical functions to biomechanical data. *IEEE Trans Biomed Eng* 1979; 26: 122–124.
- Winter D. Kinetics: forces and moments of forces. In: Biomechanics and motor control of human movement. New York: John Wiley & Sons 1990; 75–100.
- Borg G. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 5: 377–381.
- Fiatarone MA, Marks EC, Ryan ND, Meredith CN, Lipsitz LA, Evans WJ. High-intensity strength training in nonagenarians. *JAMA* 1990; 263: 3029–3034.
- Olney S, Richards C. Hemiparetic gait following stroke. Part I: characteristics. *Gait Posture* 1996; 4: 136–148.
- Tyson S. Hemiplegic gait symmetry and walking aids. *Physiother Theory Prac* 1994; 10: 153–159.
- Wall J, Turnbull G. Evaluation of out-patient physiotherapy and a home exercise program in the management of gait asymmetry in residual stroke. *J Neuro Rehabil* 1987; 1: 115–123.
- Griffin M, Olney S, McBride I. Role of asymmetry in gait performance of stroke subjects with hemiplegia. *Gait Posture* 1995; 3: 132–142.
- Lehmann J, Condon S, Price R, DeLateau B. Gait abnormalities in hemiplegia: their correlation by ankle-foot orthosis. *Arch Phys Med Rehabil* 1987; 68: 763–771.
- Andriacchi T, Ogle J, Galante J. Walking speed as a basis for normal and abnormal gait parameters. *Biomechanics* 1977; 10: 261–268.