Isokinetic Strength Training of the Hemiparetic Knee: Effects on Function and Spasticity

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ABSTRACT. Sharp SA, Brouwer BJ. Isokinetic strength training of the hemiparetic knee: effects on function and spasticity. Arch Phys Med Rehabil 1997;78:1231-6.

Purpose: To determine whether isokinetic training can improve the strength of the hemiparetic knee musculature, functional mobility, and physical activity and to evaluate its effect on spasticity in long-term stroke survivors.

Design: Nonrandomized self-controlled trial.

Subjects: A volunteer sample of 15 community-dwelling stroke survivors of at least 6 months.

Intervention: A 6-week (3 days/week, 40 minutes/day) program consisting of warm-up, stretches, reciprocal knee extension and flexion isokinetic strengthening, and cool-down for the paretic limb.

Main Outcome Measures: Peak isokinetic hamstring and quadriceps torque, quadriceps spasticity, gait velocity, timed Up and Go, timed stair climb, and the Human Activity Profile (HAP) scores were recorded at baseline, after training, and 4 weeks after training cessation (follow-up).

Results: Paretic muscle strength improved after training ($p < .05$) while tone remained consistent ($p > .87$). Gait velocity increased after training ($p < .05$) and at follow-up ($p < .05$). Changes in stair climbing and timed Up and Go were not significant ($p > .37$; $p > .91$), although subjects perceived gains in their physical abilities at follow-up ($p < .01$).

Conclusions: Gains in strength and gait velocity without concomitant increases in muscle tone are possible after a short-term strengthening program for stroke survivors. The psychological benefit associated with physical activity is significant.

STROKE CAN BE CHARACTERIZED as an interruption of the blood supply to the brain or hemorrhage into the brain tissue,[1] commonly involving a disruption in the motor and sensory pathways. Clinical manifestations include impaired motor control that can be characterized by muscle weakness, altered muscle tone, and abnormal movement patterns. These impairments limit the ability to perform functional activities such as walking, climbing stairs, and self-care. Often, despite intensive rehabilitation in the first few months after stroke, survivors are left with significant disability.[2,3] These individuals are at risk for further functional decline secondary to the aging process or comorbidity.

Weakness is recognized as a limiting factor in the motor rehabilitation of patients after stroke. Bobath[4] advocated that weakness in an agonist was attributable to spastic restraint imposed by the antagonist muscle group. During rapid, reciprocal, or concentric contractions the force generated by the agonist may be reduced because of inappropriate (reflex) or prolonged activation of the antagonist when the agonist alone should be active.[5,6] The degree of agonist-antagonist cocontraction during isometric activity is also reportedly abnormally high in hemiparesis.[7] The resultant strength deficit may be further emphasized by reductions in muscle fiber number and increased fatigability[8,9] as well as decreased motor unit numbers, and altered recruitment order.[5]

Spasticity can also lead to secondary changes in muscle and the development of contracture.[10] Alterations in the viscoelastic properties and connective tissue of spastic, paretic muscle may contribute to significant passive restraint that can be limiting in terms of the opposing muscle's ability to produce torque.[5,11,12] The degree to which spasticity contributes to deficits in strength by virtue of opposing muscles actively and passively resisting each other is not well established. There is as yet no clear understanding of the relation between spasticity and strength.

Resistance training in healthy older adults can offset age-related declines in muscular strength,[13,14] which has been attributed to improvement in both motor unit recruitment and firing frequency.[15] There is some concern among clinicians that strength training may not be appropriate in the presence of spasticity or elevated muscle tone.[16] Such training may enhance the spastic restraint and thus interfere with coordination and reinforce abnormal muscle activation patterns.[4] In conditions other than stroke that present with spasticity, however, strength gains have been associated with improved gait performance and mobility without increased spasticity.[17,18] Furthermore, training programs designed to increase strength may well contribute to improved functional status and self concept.[19]

This study was designed to determine whether persons with chronic hemiparesis can improve function and muscle strength at an isolated joint of the "affected" lower extremity following a training program and whether gains are associated with alterations in muscle spasticity.

METHODS

Subjects

Subjects with unilateral stroke who had residual weakness or spasticity of their affected lower extremity were recruited on a volunteer basis from the local Stroke Club and through newspaper advertising. All subjects were screened to ensure they were at least 6 months poststroke, were independently ambulatory with or without aids over a 12-meter distance, had not participated in any formal exercise or therapy program within the previous 4 weeks, and, on observation, would not be restricted in using the isokinetic device by contracture. Those with non–stroke-related disabilities were excluded. Subjects were also required to obtain their physicians' consent to be in the exercise

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program. Eligible subjects provided their signed consent before beginning the program. The experimental protocol was approved by the local research ethics review board.

Procedure

Demographic data were collected from all subjects to document age, time poststroke, side affected, and use of walking aids. Outcome measures relating to muscle performance (including strength and spasticity) and function (including a timed gait test, timed Up and Go test, a timed stair climb, and a Human Activity Profile score) were determined at baseline (preintervention), immediately postintervention (6 weeks), and 4 weeks after training cessation (follow-up).

Muscle Performance Testing

Peak knee extensor and flexor torques were recorded from the unaffected followed by the affected lower extremities at speeds of 30, 60, and 120°/sec in turn using the Cybex II isokinetic dynamometer.[a] Subjects were seated upright on the testing chair with a firm spacer board placed behind the back as required to position the hips sufficiently forward so the rotational axis of the knee joint could be aligned with the input shaft of the dynamometer. This position is comfortable and reliable for testing maximum knee flexor and extensor torques in the hemiparetic population.[20]

A velcro strap was secured around each subject's waist and a second strap placed just proximal to the knee to provide stabilization of the body and to reduce unwanted contribution by muscles other than those of interest. A padded strap at the distal end of the dynamometer's lever arm was secured around the subject's lower leg approximately 3cm above the malleoli. Subjects folded their arms across their torso throughout testing to minimize upper extremity involvement. Each subject performed a practice trial consisting of three submaximal knee extension-flexion combinations and three maximal efforts before recording the data. After a 30-second to 1-minute rest subjects were asked to "push and pull as hard and as fast as possible" for four repetitions. A 2-minute rest period was provided between each test speed, which progressed from slow to fast. A peak torque (adjusted for the effect of gravity) was determined at the given velocities for each of the quadriceps and hamstring muscle groups for the unaffected and affected limbs.

Spasticity of the knee extensor muscles was measured using the pendulum test,[21] which has been shown to differentiate well between normal and spastic subjects.[22,23] Subjects were positioned supine on a plinth with their legs overhanging the end such that knee flexion could freely occur. The leg not being tested was supported on a chair to minimize back strain. A battery powered electrogoniometer (elgon) was centered over the axis of rotation of the knee joint with the elgon's upper arm aligned with the greater trochanter of the femur and lower arm aligned with the lateral malleolus. Elasticized bandages secured the elgon arms in place. In addition, two gel-filled recording electrodes were placed approximately 5cm apart and taped to the skin overlying the belly of the rectus femoris muscle to record its electrical activity.

The examiner (SS) passively raised the leg to a horizontal starting position, instructed the subject to relax, then released the leg so it could swing freely. The electromyographic (EMG) signal (bandpass filtered at 10Hz to 1kHz and amplified to 0.2mV/cm) and the elgon signal were digitized (2kHz) and recorded on line for a period of 8 seconds using a laboratory computer. The signals were also viewed on an oscilloscope and the EMG signal was fed through a loudspeaker to provide

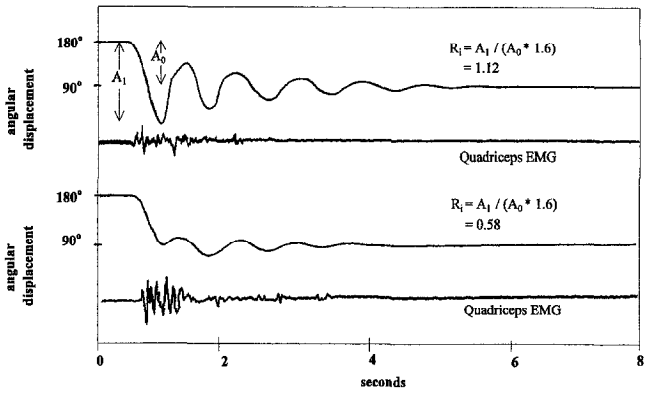

Fig 1. An example of the recordings obtained from the unaffected (top) and affected (bottom) limbs during the pendulum test.

auditory feedback to encourage subjects to relax. Data from ten trials were gathered from the unaffected, then affected, legs in turn with each trial separated by a 30-second rest to minimize intertrial effects.[23]

The data were analyzed off line using the technique described by Bajd and Vodovnik.[22] The angular displacements (in millivolts) from the horizontal start position to the final rest position (A_0) and from the start position to the first minima (A_1) were measured (fig 1). The relaxation index (R_i) was defined as $A_1/(A_0*1.6)$, where 1.6 is a constant reflecting the normal ratio of A_1/A_0. As such R_i would equal 1 in nonspastic subjects and approach 0 in the presence of extreme quadriceps spasticity. The mean and standard deviation (SD) of the R_i were calculated for the 10 trials. Relaxation index values which fell beyond $\pm$1SD were discounted and a new mean and SD calculated.[23]

Testing of Function

Subjects were instructed to walk at their most "comfortable speed" using their usual assistive device (if required) over a 12m distance. The time taken to traverse the central 8m (to avoid acceleration and deceleration) was recorded by stopwatch. The average time of three trials was determined and gait velocity calculated. Gait velocity measures yield strong interrater and test-retest reliability in hemiparetic subjects.[24] The timed Up and Go test[25] was also performed and averaged over three trials to examine subjects' ability to transfer from sitting to standing in addition to walking. This test has previously demonstrated high intrarater (ICC = .99) and interrater (ICC = .99) reliability.[25]

Stair climbing ability was determined as subjects climbed up a set of four standard steps (height, 17.7cm) at their "most comfortable" speed and using their normal pattern of foot placement and hand support. Both the time of ascent and descent were recorded for three trials and an average time and cadence (stairs/minute) were calculated. A reliability coefficient of .99 has been demonstrated for this measure in healthy subjects between 22 and 60 years of age.[26]

To gain insight into the subjects' general level of physical activity the Human Activity Profile (HAP)[27] was used. The HAP is a survey of 94 activities (including self-care, transportation, home maintenance, entertainment, and physical exercise) rated according to required metabolic equivalents. Subjects responded to each item on the scale by choosing one of three possible choices: *Still doing this activity, Have stopped doing this activity,* or *Never did this activity.* When subjects requested assistance in choosing between the first two options they were given the following guideline: "If you had the opportunity, would

you do that activity today?'' Scores were tallied to provide both a maximum activity score (MAS), which indicates the activity with the highest metabolic level that the subject still performs, and an adjusted activity score (AAS: difference between the MAS and the number of activities the subject has stopped doing) reflecting the average metabolic level in a typical day. Test-retest reliability for these outcomes are .84 and .79 for the MAS and AAS, respectively.[27]

Training Intervention

The training program required subjects to attend three sessions per week for 6 consecutive weeks (18 sessions). Training sessions began with a 5-minute warm-up on a stationary bicycle at low resistance followed by four 15-second stretches each for the quadriceps and hamstring muscles of the affected leg. Strength training involved reciprocal knee extension and flexion movements on the Orthotron[a] isokinetic machine that controls the velocity of concentric muscle action. Subjects were seated as described earlier (see muscle performance testing) and performed three sets of 6 to 8 repetitions of maximal effort at each of the three speeds approximating those used in testing. Progression was in all cases from the slowest to the fastest speed. Only the affected knee musculature was trained in order to specifically address the effects of selective training on strength, spasticity, and function.

Upon completion of the strengthening exercises, stretches were repeated as a cool-down.

Data Analysis

A multifactor repeated measures analysis of variance (ANOVA) was used to determine whether differences existed across the three testing periods (baseline, posttraining, and follow-up) for the variables of strength, spasticity, and stair climbing. A single factor repeated measures ANOVA was used to analyze the measures of gait velocity, timed Up and Go, and the AAS of the HAP. If significant variation was found between treatment time periods ($p < .05$) a post hoc multiple comparison test (the Dunn critical value[28]) was performed to determine between which test times the difference lay.

Pearson product correlations were used to determine the degree of association between peak torque values and the spasticity relaxation index. The significance of the relationship was evaluated at the $p < .05$ level.

RESULTS

Nineteen subjects were recruited into the study but four were withdrawn for the following reasons: two were unable to meet the training attendance requirements; one sustained a fall-related injury in the home that prevented further participation; and one became ill and required a period of restricted mobility. The remaining 15 (10 men) were community-dwelling stroke survivors (mean age ± 1 SD, 67 ± 10yrs) ranging from 0.9 to 18 years poststroke. Eight subjects used canes for walking, one used a rollator walker, and six did not require aids. The left side was affected in eight subjects; however, because there were no differences in baseline measures ($p > .39$), data from subjects with left and right hemiparesis were pooled.

Muscle Performance

All subjects tolerated the testing and training procedures well. Two were unable to return for follow-up testing; therefore, the multifactor repeated measures ANOVA was performed for the 13 complete data sets. Statistical findings from paired preintervention and postintervention analyses on the entire sample of 15 led to similar interpretations of the findings as the ANOVA; hence, the results of the latter are presented below.

Table 1: Summary of the Main Effects and Two-Way Interactions of the Multifactor ANOVA*

Source of Variance	SS	*df*	MS	*F* Value	*p* Value
Test period	2964.73	2	1482.36	8.76	.001
Side	69228.34	1	69228.34	41.84	.000
Muscle	352825.86	1	352825.86	203.83	.000
Speed	30320.40	2	15160.20	186.30	.000
Test period × side	415.72	2	207.86	4.42	.023
Test period × muscle	173.62	2	86.81	0.95	.401
Test period × speed	129.18	4	32.29	1.06	.384
Side × muscle	3644.52	1	3644.52	5.87	.032
Side × speed	242.08	2	121.04	1.76	.193
Muscle × speed	5379.62	2	2689.81	22.25	.000

Abbreviations: SS, sum of squares; *df*, degrees of freedom; MS, mean square.
* There were no significant three-way or four-way interaction effects ($p > .12$)

Significant main effects ($p < .001$) of the factors of time (baseline, posttraining, follow-up), side (affected, unaffected), muscle (quadriceps, hamstrings), and speed (isokinetic test velocity) were found with respect to the mean peak flexor and extensor torques produced in addition to marked interactions between two factors (table 1).

The magnitudes of the knee muscle torques improved over time ($F_{df=2} = 8.8$, $p < .001$), but the gains were limited to the affected (trained) side only ($F_{df=2} = 4.4$, $p < .024$). Both the affected hamstrings and quadriceps showed significant gains immediately following training when tested at 120°/sec ($p < .05$), and gains were also observed in hamstrings and quadriceps at 30°/sec and 60°/sec, respectively ($p < .05$) (fig 2). The improved torques were still evident at follow-up; however, they were no longer significant when compared to baseline values, with the exception of the quadriceps torque (affected leg), which demonstrated further increases in strength at a test velocity of 30°/sec ($p < .05$).

The torque-producing capabilities of the hamstrings and quadriceps muscles were greater on the unaffected side compared with the affected side ($F_{df=1} = 41.8$, $p < .0001$) and these differences were consistent at all speeds of testing and on each testing occasion ($p < .01$) as revealed from the post hoc analyses. The quadriceps muscles were notably stronger than the hamstrings ($F_{df=1} = 203.8$), and this was exaggerated on the affected side as evidenced by the interaction between the side and muscle factors ($F_{df=1} = 5.9$, $p < .033$). The expected decline in torque with increased speed ($F_{df=2} = 186.3$) was evident throughout the study period for each muscle group. This relationship was not influenced by the training ($F_{df=4} = 1.1$; $p > .38$), nor did it differ between affected and unaffected limbs ($F_{df=2} = 1.8$, $p > .19$). The effect was, however, more robust in the quadriceps muscles than the hamstrings ($F_{df=2} = 22.2$; $p < .0001$).

Table 2 summarizes the percentage change in muscle torque production over time and as a function of testing speed. At baseline, 5 to 7 subjects were unable to register a flexor torque (hamstrings) at any given speed of testing and 2 subjects could not produce an extensor torque on the affected side. Five of these individuals were able to do so after training. The degree of muscle spasticity did not appear to be a factor among the subjects who were unable to register a torque initially as their relaxation indices ranged from .19 to 1.03, where $R_i > .71$ was preselected to reflect mild hypertonicity and $R_i < .50$ to reflect severe hypertonicity.

The results of the pendulum test were sensitive to the presence of hypertonicity on the affected side as noted by the difference between the affected and unaffected lower extremity R_i

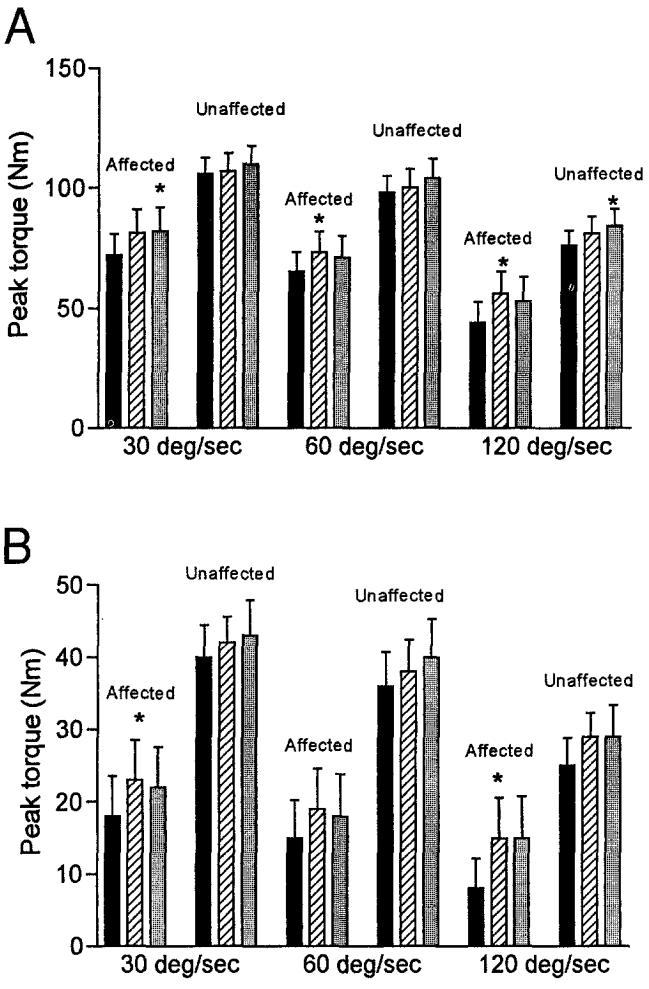

Fig 2. Mean (+1SEM) peak torque values generated by (A) the quadriceps muscle group and (B) the hamstring muscle group across time (■, baseline; ▨, posttraining; ▩, follow-up). Asterisks denote significant differences from baseline (* $p < .05$). Note also the main effects of velocity of testing ($p < .0001$), muscle (quadriceps vs hamstrings, $p < .0001$) and side (affected vs unaffected, $p < .0001$).

values over time ($F_{df=1} = 13.35, p < .004$; see figs 1 and 3). The mean R_i on the affected side remained consistent throughout the study period ($F_{df=2} = .14, p > .87$).

There was some degree of association between the baseline relaxation index and torque production at 120°/sec for the quadriceps ($r = .37$) and the hamstring ($r = .49$) muscles but the

Table 2: Mean Percentage Change in Peak Torque Values Relative to Baseline for Each Muscle Group at Three Isokinetic Test Speeds ($n = 13$)

	Posttraining			Follow-Up		
	30°/sec	60°/sec	120°/sec	30°/sec	60°/sec	120°/sec
Quadriceps						
Unaffected leg	1.15	1.19	6.38	3.55	5.94	11.02‡
Affected leg*	15.84	16.64†	19.53‡ ($n = 11$)	16.99†	9.84	5.47 ($n = 11$)
Hamstrings						
Unaffected leg	9.04	11.54	27.24	8.00	11.47	20.11
Affected leg*	39.22 ($n = 8$)	37.53 ($n = 8$)	153.89‡ ($n = 6$)	32.03 ($n = 8$)	21.30 ($n = 8$)	159.87† ($n = 6$)

* When subjects were unable to generate torque at baseline, a percentage increase could not be determined; therefore, the number of data points included is indicated in parentheses.
† $p < .05$ relative to baseline.
‡ $p < .01$ relative to baseline.

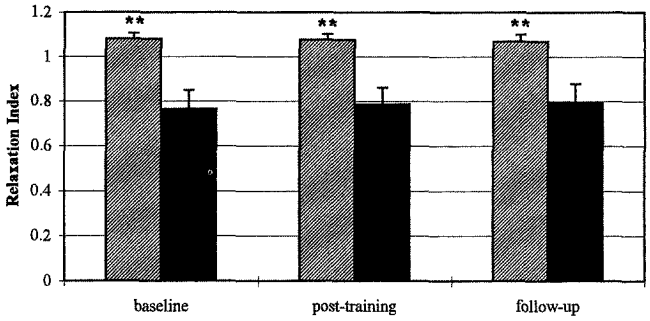

Fig 3. Mean (+1SEM) of the calculated relaxation indices measured over time: ▨, unaffected side; ■, affected side (significant [$p < .01$] differences between unaffected and affected limb R_i values).**

significance of these associations was not borne out statistically ($p > .07$). The relation between the relative gains in torque production after training and the posttraining relaxation index values was poor (quadriceps $r = .26, p > .37$; hamstrings $r = .35, p > .24$).

Functional Outcomes

Gait velocity improved after training ($F_{df=2} = 6.77, p < .005$), with speed increasing by 5.3% after training ($p < .05$) and 6.8% at follow-up ($p < .05$) relative to baseline. In contrast, there was little change over time in the results of the timed Up and Go test ($F_{df=2} = .09, p > .91$), and stair climbing ability remained unchanged ($F_{df=2} = 1.01, p > .37$). Subjects did, however, ascend the stairs more quickly than they descended ($F_{df=1} = 17.75, p < .002$). Upon observation of individual subjects' data, there was no apparent relation between changes in performance and the use of aids. The scores from the tests of functional performance are summarized in table 3.

In terms of overall physical activity, subjects improved their scores on the HAP over time ($F_{df=2} = 8.12, p < .003$). There was an average gain of 25% in the AAS scores after training relative to baseline ($p > .05$), and this increased further to a 36% improvement from baseline at follow-up ($p < .01$). The raw score values are presented in table 3.

DISCUSSION

The main finding of this study was that a 6-week training program of the hemiparetic knee musculature resulted in significant increases in muscle strength without any detectable change in extensor spasticity. After 4 weeks of detraining the strength gains were, for the most part, no longer significant; however, absolute strength performance remained higher than initially measured. Interestingly, the perceived ability to perform physical activity continued to improve even at follow-up testing.

The subjects in this study demonstrated significant weakness on the affected side and knee flexors were more impaired than

Table 3: Mean Scores (±1SD) Achieved on Functional Performance Measures at Baseline, Posttraining, and at Follow-Up ($n = 13$)

Variable	Baseline	Posttraining	Follow-Up	p Value
Gait velocity (m/sec)	.66 ± .38	.70 ± .42*	.69 ± .40*	$p < .05$
Timed Up and Go (sec)	30.69 ± 26.50	29.90 ± 27.37	31.11 ± 30.69	$p > .91$
Stair climb (stairs/min)				
Ascent	44.5 ± 23.3	43.9 ± 23.8	44.7 ± 22.6	$p > .39$
Descent	40.3 ± 25.0	37.0 ± 21.1	38.0 ± 22.7	$p > .37$
HAP (AAS)	42 ± 21	48 ± 19	51 ± 18†	$p < .01$

* Significant difference from baseline, $p < .05$.
† Significant difference from baseline, $p < .01$.

the knee extensors. This pattern is typical of the dynamic strength deficits in persons with hemiparesis.[6,29,30] It has been shown, however, that isokinetic training can result in increased strength and improved walking performance in adults after stroke.[30,31] Average gains in knee extensor torques of 12N-m and 14N-m at speeds of 60°/sec and 120°/sec, respectively, have been reported after 6 weeks of concentric knee extensor training in stroke patients.[30] These values are consistent with those of the present study although the relative strength gains (15% to 20%) were somewhat less than those reported by Engardt et al,[30] likely reflecting differences in initial strength status. Muscles that were weaker initially tended to show the greatest improvements.

There are few reports of studies that examined the effects of strength training on the knee flexors in isolation or in conjunction with the knee extensors in the presence of extensor spasticity. Concentric contractions of the nonspastic knee flexors at fast movement speeds have been associated with the initiation of a stretch reflex in the spastic muscles, thus limiting the ability to produce maximal flexor torques.[6,32] The results of the present study suggest that the magnitude of the torque production of the flexors can be improved by 37% to 154% after training. This occurred in conjunction with gains in extensor strength and in the absence of tonal change in the knee extensors. Arguably, some degree of coactivation of the knee extensors and flexors may have been present during reciprocal movements, but evidently not to the extent of masking gains in strength or preventing subjects from achieving the desired isokinetic speeds. In the absence of recording electromyograms the presence (or absence) of coactivation cannot be determined.

The presence of moderate to severe hypertonus is believed causal to the inability of persons with hemiparesis to produce recordable isokinetic knee flexor torques.[6,33,34] Nakamura et al[29] found no association between hyperactivity of the patellar tendon reflex with either isometric or isokinetic knee extensor strength. In the present study, the relaxation index had some degree of association (though not statistically significant) with flexor torque production ($r = .49$). However, individuals with mild spasticity ($R_i > .71$) were among those who were unable to produce measurable flexor torque. Furthermore, the association was poor between the relative gain in flexor torque and the relaxation index ($r = .35$), suggesting that the ability to improve flexor strength is only weakly associated with the level of extensor spasticity. One might argue that the static and dynamic stretching incorporated into the training program may have led to a reduction in reflex excitability[35] or an increase in muscle compliance,[36] enabling more effective torque production. In future studies, it would be valuable to evaluate the contribution of these potential restraints during active movement. In the present findings, however, prolonged alteration in tone was not detected using the pendulum test.

The physiological mechanisms underlying the increases in strength cannot be determined on the basis of the current data, although it is probable that improved motor unit recruitment[15,37] and motor learning (the development of neuromotor patterns of coordination between agonist and antagonist muscles through practice of a skill)[38,39] may have contributed to some degree. It may be argued that subjects' improved strength after completing the program reflected a familiarity with isokinetic devices rather than a true gain in strength. However, improvements in gait velocity suggest that the effects were not merely task specific.

The mean walking speed at baseline (.66m/sec) indicated that, after stroke, subjects ambulate approximately 50% slower than healthy adults of the same age group (1.2m/sec).[40] After training, walking velocity increased by 5.8% and 6.8% at follow-up. The continued improvement in gait velocity beyond program cessation concomitant with a slight decline in knee muscle strength is interesting given the significant correlation between these two variables.[29,33] Clearly, other variables must be considered. Regression analysis of temporal, kinematic, and kinetic variables describing hemiparetic gait revealed that the power generated by the hip flexors and the ankle plantarflexors of the paretic lower extremity are the best predictors of walking speed.[41]

The lack of improvements in the stair climbing and Up and Go tests may reflect that other muscle groups may determine functional performance to a greater degree than does the knee alone. Functional tasks are comprised of various components and require balance[42] and coordination. The effects, therefore, of training specific muscle groups may not be sufficient to invoke changes in more complex tasks. The power to detect significant changes in functional abilities is also limited when the intersubject variation is high, as was the case in the present study.

The ability to retain some strength gain even after the end of the training program, as well as the continued improvement in gait speed, may be secondary to subjects feeling better about their physical abilities and perhaps leading more active lifestyles. The HAP scores were highest at follow-up, suggesting that the training program yielded a prolonged positive effect. Whether the scores reflected actual performance of activities by the subjects, or their perceived ability that they could perform certain activities given the opportunity, is not known. It is, however, unlikely that subjects scored higher simply by virtue of being involved in the study because a progressive increase beyond the termination of the program would be inconsistent with such a hypothesis.

Other studies that have investigated the psychological effects of exercise have demonstrated a general positive trend between physical activity and psychological well-being in both disabled and control groups.[43,44] It has further been suggested that physiological improvement is not a criterion for psychological improvement to be experienced during exercise training.[44] Unsolicited comments offered by the majority of participants in the present study indicated that they had better tolerance for activities, could walk faster, or felt more confident walking longer distances than before starting the program.

The results of this study are encouraging in terms of chronic stroke survivors being able to significantly improve their strength without concomitant increases in tone. Although the carryover from the strengthening of specific muscle groups to an improvement in functional abilities was limited, the gains in actual or perceived ability to perform physical activities was marked. Whether this translates into long-term enrichment in stroke survivors' quality of life remains to be determined, and longer term follow-up is warranted.

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