Research Report

The Effect of Prolonged Static and Cyclic Stretching on Ankle Joint Stiffness, Torque Relaxation, and Gait in People With Stroke

Background and Purpose. Continuous passive motion (cyclic stretching applied to the subject's limb) has been used for the rehabilitation of some orthopedic impairments; however, few researchers have considered its application in the management of neurological disorders such as stroke. The purpose of this study was to examine the short-term effects of prolonged static and cyclic calf stretching on passive ankle joint stiffness, torque relaxation, and gait in people with ischemic stroke. Subjects. Ten community-dwelling people (mean age=64.6 years, SD=8.76, range=53–76) who were diagnosed with a cerebrovascular accident volunteered to be subjects. Methods. Participants engaged in one 30-minute static stretch and one 30-minute cyclic stretch of the calf muscle, using an isokinetic dynamometer that also collected torque and angle measurements. Before and after treatments, 10-m walking times were collected. Ankle joint stiffness was calculated from the slope of the torque and angle curves before and immediately after treatments, and torque relaxation was calculated as the percentage of decrease in peak passive torque over the 30-minute stretch durations. Results. Ankle joint stiffness decreased by 35% and 30% after the static and cyclic stretches, respectively. Stiffness values and 10-m walk times were not different between conditions. The amount of torque relaxation was 53% greater for static stretching than for cyclic stretching. Discussion and Conclusion. These preliminary data from a very small sample of people with stroke indicate that ankle joint stiffness decreases after both prolonged static and cyclic stretches; however, neither technique appears to be better at reducing stiffness in people with stroke. Torque relaxation is greater after static stretching than after cyclic stretching, and walking speed does not appear to be influenced by the stretching treatments used in our study. [Bressel E, McNair PJ. The effect of prolonged static and cyclic stretching on ankle joint stiffness, torque relaxation, and gait in people with stroke. *Phys Ther.* 2002;82:880–887.]

Key Words: *Biomechanics, Hypertonia, Rehabilitation, Spasticity.*

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People who have experienced a cerebrovascular accident (stroke) may exhibit an increased resistance to passive joint movement, decreased joint range of motion, and some exaggerated stretch reflexes. Researchers and clinicians have frequently described some of these symptoms as components of *spasticity*, defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex."[1(p485)]

The definition of spasticity implies that an increase in motoneuron response to muscle lengthening may be the primary mechanism for these symptoms of stroke.[2] Although evidence for increased motoneuron excitability is strongly supported,[3–5] some researchers[6–9] have argued that changes in passive mechanical properties of muscle may be the additional mechanisms responsible for symptoms observed in people with spasticity.

Passive mechanical properties of muscle (ie, how the muscle responds to applied loads) are related to the amount, type, temperature, and organization of structures such as muscle, collagen, elastin, proteoglycans, and water.[10] The mechanical properties are typically expressed as *stiffness*, which is the relationship between passive resistive torque and joint displacement, and *torque relaxation*, which is the decrease in peak passive torque (passive resistance) exhibited at a joint held in a non-neutral position over time.[11–13] Studies have shown that, in patients with spasticity, passive mechanical properties of muscle are to some extent responsible for impaired gait patterns[6,7,14] and enhanced passive joint resistance.[9,15] In our opinion, these findings suggest that interventions for stroke may need to focus on passive mechanical properties of muscle and not just motoneuron excitability.

Approaches for treating symptoms related to spasticity following stroke range from the use of modalities (eg, biofeedback) to complex neurosurgical procedures (eg, dorsal rhizotomies). There is, however, no consistent definition for spasticity. Therefore, these interventions may not always be aimed at decreasing the same symptoms. One common intervention that is used with most approaches is prolonged static stretching.[16–18] Investigators[16,18–21] have shown that stretching of the plantar-flexor muscles for periods ranging between 30 minutes and 6 weeks, imposed with a tilt table or cast, reduced passive ankle joint resistance, increased ankle joint range of motion, and improved gait characteristics (eg, stride length, stride width, angular joint displace-

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Both authors provided concept/research design, writing, and consultation (including review of the manuscript before submission. Dr Bressel provided data collection and analysis, project management, subjects, and clerical support. Dr McNair provided facilities/equipment.

The Auckland University of Technology Ethics Committee approved this study.

This work was presented at the 2001 annual meeting of the American Society of Biomechanics; August 8–11, 2001; San Diego, Calif.

This article was submitted August 17, 2001, and was accepted March 19, 2002.

ment). Some researchers[22,23] have argued that the effects of static stretching are proportional to the amount of time a stretch is held at its end-range. Accordingly, some authors[18,20] have suggested that prolonged static stretching rather than short-term stretching (eg, <2 minutes) can be a convenient and cost-effective means of reducing symptoms of spasticity.

Research has indicated that continuous passive motion (ie, cyclic stretching) about the ankle joint may be more effective in reducing passive ankle joint stiffness than static calf stretching.[11,24] McNair and coworkers[11] examined passive ankle joint stiffness in volunteers without impairments after a single 60-second static calf stretch and after a 60-second cyclic calf stretch to determine the efficacy of the 2 stretching techniques. They reported a 16% decrease in ankle joint stiffness after cyclic stretching and no difference in stiffness after static stretching. Their findings supported earlier work[24] that showed ankle joint stiffness was more effectively decreased by jogging than by static stretching exercises, which suggests that cyclic motion may be more effective than static holds at decreasing stiffness at the ankle joint. Cyclic stretching also may increase joint range of motion according to Taylor et al,[25] who used an in vitro model.

Although cyclic stretching has been an accepted approach for the rehabilitation of some orthopedic problems (eg, total joint arthroplasty, contracture),[26] few researchers have considered its application in neurological disorders such as stroke. The purpose of our study, therefore, was to compare the short-term effects of prolonged static and cyclic calf stretching on passive ankle joint stiffness, torque relaxation, and gait in people with stroke who exhibit increased resistance to passive joint movement, decreased joint range of motion, and exaggerated stretch reflexes. We believe the results of our study may be clinically meaningful if cyclic stretching is more effective than static stretching in reducing the symptoms of stroke.

Method

Subjects

Ten community-dwelling people (9 men and 1 woman) who were diagnosed with spasticity due to a cerebrovascular accident volunteered for this investigation. The diagnoses were made by a neurologist, and patient records did not include the criteria used for the diagnoses of stroke. The subjects were recruited from the neurological rehabilitation clinic at Auckland University of Technology and from an advertisement placed in a local daily newspaper. The number of subjects chosen for the study was calculated using SamplePower software* and was based on a standard deviation of 0.21,[11] with an alpha level of .05 and power at 0.80. We hypothesized that a 10% difference in passive stiffness between stretching conditions would be clinically relevant.

Participants were included in the study if they met the following criteria: (1) they were not taking anti-spasticity medication, (2) they were free from contractures, (3) they had the mental capacity to perform the experimental tasks, (4) they were ambulatory with or without assistive devices, (5) their stroke occurred at least 3 months before the start of the study, (6) they had not been involved in a stretching program, and (7) they had sufficient ankle range of motion to perform the experimental task. The participants' time since stroke, mental state as determined with the Mini-Mental State Examination,[27] degree of spasticity as determined using a modified Ashworth Spasticity Scale,[28] ankle clonus response, ankle jerk response, and plantar response were evaluated and characterized. The subjects' physical characteristics are shown in Table 1, and a description of the assessments is reported in the Appendix. Before taking part in the study, participants read and signed an informed consent form approved by the Auckland University of Technology Ethics Committee.

Procedures

Participants attended a preliminary test session that included the measurement of physical characteristics and maximal dorsiflexion angle. Subjects relaxed in a supine position with their knees fully extended while their foot was passively moved from 10 degrees of plantar flexion into dorsiflexion until "firm" resistance was displayed[29] or the subject reported feeling discomfort. The greatest dorsiflexion angle out of 3 attempts was recorded for subsequent analyses.

Participants attended two 1-hour experimental test sessions that were separated from each other by 1 week and were conducted at the same time of day. Subjects were instructed to not begin a stretching program between test sessions, and they were told to reschedule their session if symptoms of their stroke prevented testing. During the first test session, participants engaged in either a 30-minute cyclic stretching protocol or a 30-minute static stretching protocol. Subjects were randomly assigned to the 2 groups and performed the protocol on their involved lower extremity. The stretching protocol that was not performed during the first test session was performed during the second test session. The 30-minute stretch duration was chosen for comparative purposes and because it has been previously shown to reduce passive ankle joint resistance and motoneuron

* SPSS Inc, 233 S Wacker Dr, Chicago, IL 60606.

Table 1.
Physical Characteristics of Subjects With Stroke

Subject No.	Age (y)	Sex	Time Since Stroke (y)	Mini-Mental State Examination Score[a]	Ashworth Spasticity Scale Score[b]	Ankle Clonus Response (No. of Beats)[c]	Ankle Jerk Response[d]	Plantar Response[e]
1	57	M	1	27	1	1	++	−
2	67	M	8	29	4	2	++++	+
3	74	M	24	30	3	2	++	+
4	56	M	6	30	2	2	++++	−
5	54	M	5	28	3	1	+++	+
6	53	F	2	28	4	1	+++	+
7	71	M	4	21	1	0	++	−
8	71	M	4	29	2	3	++	−
9	67	M	5	28	1	1	++	−
10	76	M	8	29	2	1	++	−

[a] Scores range from 0 to 30. 30=no errors on test, a score of 20 was used to distinguish between people with cognitive impairments and people with "normal" levels of cognition.
[b] Scores range from 0 (no muscle tone) to 4 (limbs rigid in flexion or extension).
[c] Three or more beats indicates hyperreflexia.
[d] Ankle jerk response: 0=no response, +=diminished/slight response, ++=normal, +++=marked response, ++++=severe response.
[e] Plantar response: −=negative, +=positive.

excitability of the triceps surae muscle and to increase ankle joint range of motion.[16,18,20]

For the 30-minute static calf stretching protocol, the subject's foot was moved at a rate of 5°/s from 10 degrees of plantar flexion (neutral) to a static hold at 80% of the participant's maximal passive dorsiflexion angle. Immediately following the static stretch, the foot was returned to neutral, then back to 80% of maximal dorsiflexion angle, and again returned back to neutral. The last stretching sequence was necessary so that measurements of stiffness before the stretch could be compared with measurements of stiffness after the stretch.

For the 30-minute cyclic calf stretching protocol, continuous passive ankle joint motion between neutral and 80% of the participant's maximal passive dorsiflexion angle was performed. The angular velocity of the foot segment during the cyclic stretch was 5°/s. The angular velocity and end-ranges of motion selected for the static and cyclic stretching protocols have been shown in some subjects with spastic cerebral palsy and volunteers without impairments to not evoke a stretch reflex and to improve subject relaxation.[11,30]

During the stretching protocols, the participants were asked to lie in a supine position with their foot strapped to a footplate connected to a Kin-Com dynamometer[†] (Fig. 1). The Kin-Com dynamometer is a computerized device that was programmed to manually move the foot according to the stretching protocols and to collect force and angle measurements. The signals from the potentiometer and load cell of the Kin-Com dynamometer were sampled simultaneously at 500 Hz and stored for subsequent analyses using a computer. Previous studies[30,31] have shown that under selected conditions the Kin-Com dynamometer provides measurements that we would consider reliable and valid for ankle joint angle and passive torque. We did not examine the reliability of our measurements using our device on our subjects.

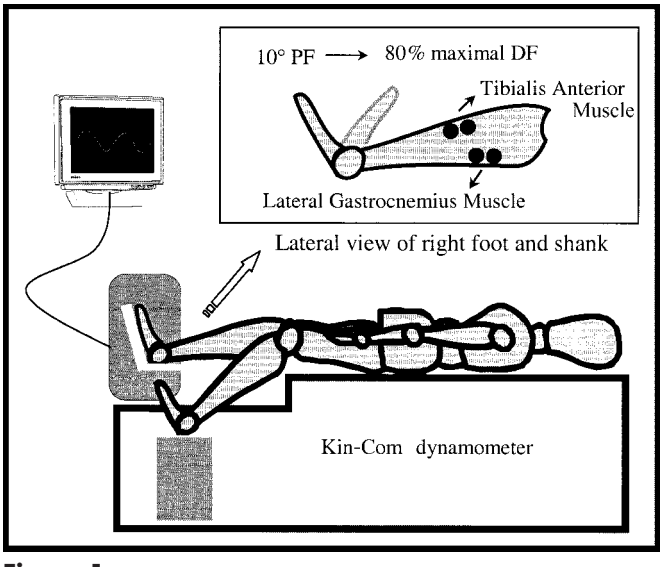

Figure 1.
Experimental setup used for measuring ankle joint angle and torque. The inset illustrates the plantar-flexion (PF) and dorsiflexion (DF) angles used and the electromyographic electrode positions.

Electromyographic (EMG) activity was monitored in an effort to ensure that passive torque measurements were not influenced by voluntary or involuntary muscle activ-

[†] Chattecx Corp, PO Box 4287, Chattanooga, TN 37045.

ity. The EMG signals were recorded with bipolar surface electrodes (DelSys 2.01)[‡] placed over the tibialis anterior and lateral gastrocnemius muscles (Fig. 1). A ground electrode was placed on the fibular head. Standardized techniques for surface electrode preparation and placement were used.[32] The EMG signals were recorded for 10 seconds, sampled at 500 Hz at a bandwidth of 20 to 450 Hz, and amplified using a DelSys Bagnoli 2 amplifier.[‡] The EMG signals were collected at 1-minute intervals beginning with the initial movement of the foot into dorsiflexion. The root mean square of the EMG data was calculated[32] and normalized to reflect a percentage of maximal voluntary contractions (MVCs). Subjects performed MVCs of the plantar flexors and dorsiflexors with their feet held in 10 degrees of plantar flexion, using the Kin-Com dynamometer. The subjects performed 3 maximal efforts, with verbal encouragement from the investigator (EB). The data of subjects exhibiting EMG values greater than 1% of the MVC during the passive measurements were not used for the data analysis.

Gait was assessed by having participants complete a 10-m walk test before and after treatments at a "comfortable speed." Walking speed is one of the most widely accepted measures of lower-limb recovery,[33] and studies of test-retest reliability of this measure yielded intraclass correlation coefficients of .94.[34] The intention of including a 10-m walk test in our study was to test the immediate effect of stretching treatments on a functional outcome measure. The average time of 3 walking trials was used for subsequent analyses. We did not examine the reliability of data obtained with the gait measure on our subjects.

Data Analysis

We defined *passive joint resistance* as resistive torque exerted through the foot by, among other structures, the plantar-flexor muscle-tendon unit, and it was calculated from the force data taken from the Kin-Com's load cell and the moment arm length. *Moment arm length* was defined as the distance from the lower edge of the load cell to the lateral malleolus and corresponded to 0.2 m.

Mean stiffness was calculated before and after treatments from the slope of the torque-angle curve (Δ torque/Δ angle) for the initial and final movements of the foot into dorsiflexion (ie, from neutral to 80% of the subject's maximal passive dorsiflexion angle). Torque relaxation was calculated from measurements of initial peak torque and final peak passive torque taken during the 30-minute static and cyclic calf stretching protocols. Torque relaxation was expressed as a percentage of initial peak passive torque. Figure 2 provides a schematic representation of the stiffness and torque relaxation measurements for the static condition.

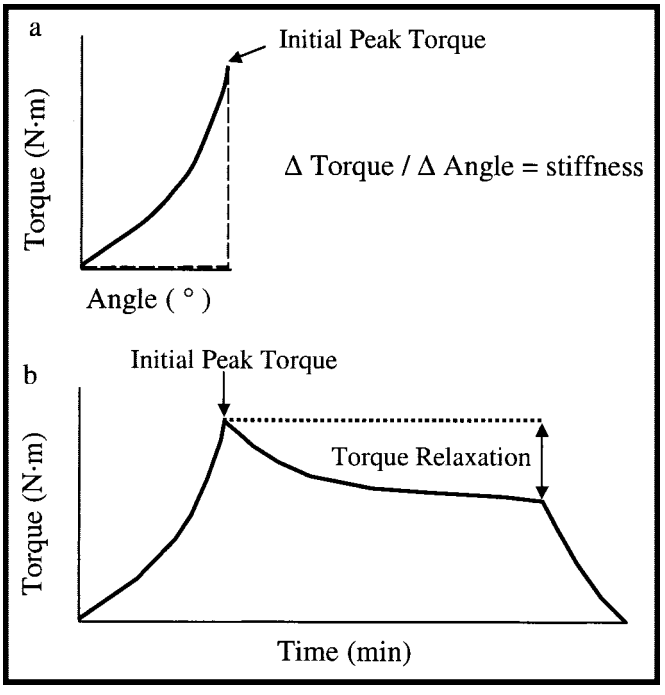

Figure 2.
Schematic representation of the stiffness and torque relaxation measurements: (a) stiffness reflects the change (Δ) in torque to the change in angle ratio, and (b) torque relaxation reflects the percentage of decrease in passive torque over the 30-minute passive calf stretch.

The within-subject design of this study included 2 independent variables (stretching protocol and treatment time) and 3 dependent variables (mean stiffness, percentage of torque relaxation, and 10-m walking times). Given the number of dependent variables, we used a multivariate Hotelling T^2 procedure to determine the effect that cyclic and static stretching had on the group of dependent variables. If the main multivariate effect for group was statistically significant, a Wilcoxon signed rank test value was then calculated for each dependent measure. Given the high variability expected among subjects, we chose to analyze differences between conditions with the Wilcoxon nonparametric test. The probability associated with a Type I error was set at .05 for all observations.

Results

All subjects underwent testing as planned, and no subjects exhibited an EMG value greater than 1% of MVC. Therefore, data from all subjects were included in the analyses. The Wilks' criteria for the Hotelling T^2 procedure revealed that the combined group of dependent variables were affected by stretching protocols ($P<.001$) and treatment time ($P<.001$). Follow-up univariate statistics showed that mean stiffness values were different after static and cyclic stretching, whereas no differences

[‡] DelSys Inc, PO Box 15734, Boston, MA 02215.

Table 2.
Comparison of Dependent Variables Within and Between Prolonged Static and Cyclic Stretching

	Pre-stretch Values						Post-stretch values					
	Static			Cyclic			Static			Cyclic		
Variable	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range
Stiffness (N·m/°)	1.50	0.42	0.80–2.30	1.54	0.61	0.50–2.60	0.98	0.33[a]	0.55–1.51	1.08	0.44[a]	0.35–1.83
Torque relaxation (%)							35.12	2.44	30.84–38.84	23.02	8.75[b]	13.51–39.53
10-m walk times (s)	20.25	11.89	8.27–50.99	20.16	12.07	7.18–50.99	20.70	12.08	6.76–51.55	20.24	12.22	6.68–51.00

[a] Significantly different from pre-stretch values ($P<.05$).
[b] Significantly different from static stretching ($P<.05$).

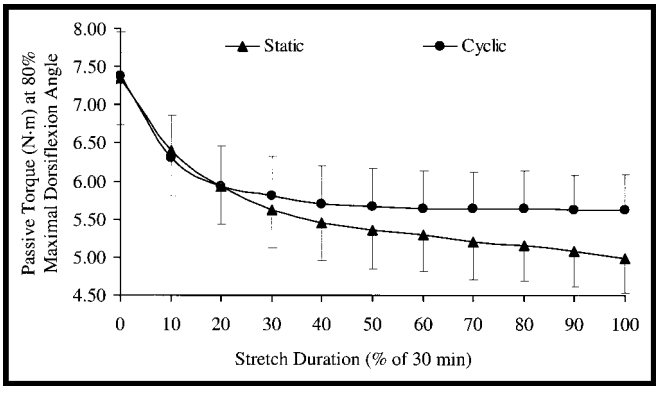

Figure 3.
Passive torque values (mean ± SD) during the 30-minute static and cyclic calf stretches while the foot was held at 80% maximal dorsiflexion angle.

were observed between conditions (Tab. 2). Stiffness values decreased by 35% and 30% after static and cyclic stretching, respectively.

The amounts of torque relaxation were different between conditions ($P<.01$). The amounts of torque relaxation are reported in Table 2 and were 53% greater for static stretching than for cyclic stretching (Fig. 3). Univariate analyses of mean walking times revealed no main effect within or between conditions (Tab. 2).

Discussion and Conclusions

Previous research[11] has suggested that cyclic stretching about the ankle joint may be more effective than static stretching at decreasing ankle joint stiffness. A comparison of stretching interventions has not been examined in people with stroke, where the potential benefits of decreased stiffness might be considered highly desirable. An aim of our investigation, therefore, was to compare the short-term effects of prolonged static and cyclic calf stretching on passive ankle joint stiffness in people with stroke.

Stiffness values decreased after prolonged static stretching, and we found this consistent with the passive torque data reported by Tremblay and coworkers.[20] Our results also support the contention that prolonged static stretching is effective at reducing symptoms of spasticity such as passive joint resistance.[16,20] Our results further suggest that prolonged cyclic stretching may be equally effective as prolonged static stretching at decreasing ankle joint stiffness (Tab. 2).

According to Enoka,[35] a mechanism by which stiffness may decrease after a passive static or cyclic stretch is related to the thixotropic property of muscle. *Thixotropy* has been defined as the physical change of a substance after being mechanically agitated.[36,37] For example, a gel substance such as ketchup may become less viscous (ie, more fluid) if mechanically agitated.[35] In this study, the gel component of muscle (eg, water and proteoglycans) may have become less viscous after being stretched, resulting in less passive stiffness. Indeed, for this scenario to be plausible, the muscle must not receive neural input because this may also modulate stiffness. Because EMG activity was less than 1% of the MVC during the conditions of our study, we do not consider neural mechanisms as contributing to the passive mechanical measures.

In general, our stiffness values were greater than those reported for people without a stroke[12,38] and are consistent with data reported by other researchers.[9,15] Vattanasilp et al[15] investigated factors contributing to muscle stiffness after stroke, including thixotropy and contracture. They concluded that contracture was a major contributor to exaggerated ankle joint stiffness and that thixotropy was not different in patients with stroke versus subjects without impairments who were in a control group. Their results[15] and ours showing greater passive stiffness may provide further evidence to support the research of Dietz et al[39] that indicated a morphological and not just a motoneuron transformation of spastic muscle over time.

Our results, which showed no difference in stiffness between stretching conditions, do not agree with the

results of previous work examining the effect of short-term stretches (eg, 60 seconds) in people without impairment.[11] McNair and coworkers[11] suggested that thixotropic properties of muscle (eg, collagen, water, proteoglycans) may become less viscous during cyclic motion than during static stretching because of the continuous nature of the cyclic stretch. In our study, constituents of muscle that contribute to thixotropy may not have responded the same as they did in middle-aged subjects without impairment, and this may be due in part to the age of our subjects and the condition of the patients' tissues as a result of their stroke.

An additional purpose of our study was to compare the effect of prolonged static and cyclic calf stretching on torque relaxation. The patterns of decline in torque between these 2 modes of stretching were similar to those of previous research.[11] McNair and coworkers[11] reported 17% and 11% declines in torque over 60-second static and cyclic stretches, respectively, at the ankle joint. The difference in torque relaxation between stretching modes (ie, 54%) that they observed corresponds to the 53% difference we observed. Torque relaxation values were substantially greater in our study (23%–35%), probably because of the prolonged stretch duration (60 seconds versus 30 minutes). Theoretically, the torque relaxation response could continue indefinitely because of the viscoelastic properties of biological tissues.[25] Our static stretch data supported this contention and continued to decline up to 30 minutes, whereas no appreciable decreases were observed after the initial 15 to 20 minutes of the cyclic stretching condition (Fig. 3). On the basis of these findings and those of other researchers,[11] it appears, in our opinion, that static stretching may be more effective than cyclic stretching at decreasing peak passive torque in people with stroke.

It is important to examine whether an intervention is useful to the patient and therapist. Researchers[19,21] have shown that long-term static stretching of the plantar flexors (>30 minutes), using a casting boot, improved characteristics of gait in subjects with spastic cerebral palsy. In our study, we used a common measure of mobility (ie, 10-m walk times) and found no improvement after or between stretching interventions (Tab. 2). These data imply that neither the 30-minute cyclic intervention nor the 30-minute static stretch intervention may improve gait speed after a one-time treatment.

The results of our study concern the immediate effects after a one-time 30-minute stretch intervention. We did not measure any long-lasting effects. Researchers in this area, in our opinion, should compare the lasting effects of each treatment after a training period. This would be more clinically relevant. We believe it may also be of value to assess force measurements before and after interventions, because there is recent evidence to support 12% and 25% decreases in MVC of the quadriceps femoris and plantar-flexor muscles, respectively, after a prolonged 20- to 30-minute passive stretch in volunteers without impairments.[40,41] Reduced strength of the quadriceps femoris and plantar-flexor muscles of patients with stroke may further decrease performance on selected functional outcomes such as gait because of their already weakened condition.

Ankle joint stiffness decreased after both prolonged static and cyclic stretching, although neither technique appeared to be better at reducing stiffness in people with stroke. Torque relaxation was greater after static stretching than after cyclic stretching, and walking speed did not appear to be influenced by the stretching treatments used in this study.

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Appendix.

Assessment Measures Used in the Study

Mini-Mental State Examination: examines cognitive aspects of mental health. Subjects responded to a series of assessments that include orientation, memory, naming, verbal instruction, and written commands.[27] Scores range from 0 to 30, with 30 indicating no errors on the test. A score of 20/30 was used in this study to distinguish between people with cognitive impairments and people with "normal" levels of cognition.[a]

Modified Ashworth Spasticity Scale: grades the passive resistance (or tone) encountered at a joint during a passive stretch. The 5-point scale ranges from 0 (no muscle tone) to 4 (limb is rigid in flexion or extension).[28]

Ankle Clonus Response: tests for hyperreflexia. Clonus was measured by quickly moving and releasing the relaxed foot into dorsiflexion and counting the number of beats or rhythmic plantar-flexion movements. Three or more beats is an indication of hyperreflexia.[b]

Ankle Jerk Response: tests for hyperreflexia. The ankle jerk response was elicited by tapping the Achilles tendon with a reflex hammer to produce a sudden stretch of the plantar-flexor muscle tendon unit. The response was graded as no response, diminished/slight, normal, marked, or severe depending on the plantar-flexion response.[b]

Plantar Response: tests for hyperreflexia. The plantar response was elicited by moving a fingertip up the lateral side of the foot from the heel to the base of the little toe (digit 5) then across the ball of the foot. The response was graded as "positive" when digit 1 (great toe) plantar flexed or "negative" when digit 1 extended and digits 2 to 5 abducted. A positive test is considered normal.[b]

[a] Folstein M, Anthony JC, Parhad I, et al. The meaning of cognitive impairment in the elderly. *J Am Geriatr Soc.* 1985;33:228–235.

[b] O'Sullivan SB, Schmitz TJ. *Physical Rehabilitation Assessment and Treatment.* 4th ed. Philadelphia, Pa: FA Davis Co; 2000.