

Ankle Stiffness and Tissue Compliance in Stroke Survivors: A Validation of Myotonometer Measurements

Sarah J. Rydahl, MS, Brenda J. Brouwer, PhD

ABSTRACT. Rydahl SJ, Brouwer BJ. Ankle stiffness and tissue compliance in stroke survivors: a validation of Myotonometer measurements. *Arch Phys Med Rehabil* 2004; 85:1631-7.

Objective: To determine the construct validity of Myotonometer measurements of tissue compliance as being reflective of ankle plantarflexor tone.

Design: Cross sectional.

Setting: Motor performance research laboratory.

Participants: Twenty-three stroke survivors (67.5 ± 10.9 y) and 24 control subjects (71.2 ± 9.0 y) recruited from the community.

Interventions: Not applicable.

Main Outcome Measures: Plantarflexor tone was measured using the Modified Ashworth Scale (MAS), ankle stiffness (total, passive, intrinsic and reflex components) was quantified using a torque motor, and tissue compliance during relaxation and activation of the plantarflexors was measured with the Myotonometer.

Results: MAS scores in the stroke group ranged from 1 to 4, whereas all control subjects had normal tone. Mean total ankle stiffness was higher in the stroke group than in the control group ($P < .02$), mainly due to elevated passive stiffness ($P < .03$). Compliance did not change as a function of muscle activation in stroke, but it decreased when control subjects contracted their plantarflexors ($P < .04$). The difference in Myotonometer measurements acquired during active and relaxed states was associated with total ankle stiffness and, specifically, intrinsic stiffness. The relationships were strongest when only stroke data were considered.

Conclusions: Stiffness and compliance measures distinguished between control subjects and persons with hypertonia secondary to stroke. Compliance differences in the relaxed and active gastrocnemius muscle reflected intrinsic stiffness associated with the contractile elements of the plantarflexor group of muscles as a whole.

Key Words: Muscle hypertonia; Muscle spasticity; Rehabilitation; Reliability and validity; Treatment outcome.

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ELEVATED MUSCLE TONE or hypertonicity is a common impairment after stroke and early rehabilitation often includes the normalization of tone among the treatment

goals.¹⁻⁴ Implicit in such an approach is that tone can be adequately quantified using methods that are reliable, valid, and responsive to progression and interventions.

Muscle tone is the resistance to passive muscle stretch and is the net result of several contributing mechanisms, including reflex excitability and gain, the mechanical (viscoelastic) properties of the musculotendinous unit, and the intrinsic properties or active resistance of the contractile elements.⁵⁻⁷ These contributors coexist, are interdependent, and in persons with neurologic conditions can change dramatically over time.^{8,9} For example, prolonged mobility restriction can lead to soft tissue contracture, which in turn can alter the point in the range of motion (ROM) at which the stretch reflex is initiated.¹⁰ To target interventions appropriately, it is important not only to quantify hypertonia, but also to identify the mechanisms contributing to disordered tone at any point in time. At a minimum, it is imperative that clinicians and researchers be knowledgeable about which factors or combination of factors they are measuring when they use a specific instrument or technique.

Servo-controlled torque motors have been used to quantify joint stiffness by measuring the change in resistance torque per unit joint displacement or muscle stretch.^{11,12} By moving a joint through a fixed range at a constant velocity under various conditions, stiffness can be separated into nonreflex (both passive and intrinsic) and reflex components.⁷ These measures yield excellent inter- and intrarater reliability (intraclass correlation coefficients [ICCs], $> .86$)¹³ and are highly responsive to alterations in tone.^{11,12} In subjects clinically identified as having spastic hemiparesis, passive ankle stiffness was found to be elevated compared with healthy control subjects, but reflex-mediated stiffness was similar in the 2 groups.¹⁴ The latter finding is paradoxical because spasticity is defined as a velocity-dependent increase in resistance to passive stretch and is described clinically as exaggerated tendon reflexes.¹⁴ Such discordance is likely attributable, at least in part, to the inaccuracies of clinical measures^{15,16} and their inability to differentiate between neural and other factors contributing to tone.^{6,8}

The Modified Ashworth Scale¹⁷ (MAS) is among the more commonly used clinical measures of muscle tone, even though its psychometric properties are poor. Scores tend to cluster in the lower ranges (slight increase in tone) of the 6-point ordinal scale, limiting its ability to discriminate between individuals or groups of patients.^{18,19} In terms of reliability, agreement between raters is good when testing the elbow flexors¹⁷ but poor when assessing the ankle plantarflexors.¹⁹ These findings, combined with the lack of association between plantarflexor MAS scores and dynamometric (quantitative) recordings of resistance torque,^{6,20} reaffirm the need for suitable clinical tools that reliably and accurately assess lower-extremity tone.

Leonard et al^{16,21} tested the reliability of their recently developed computerized tissue compliance meter, the Myotonometer.^a The meter quantifies the amount of tissue displacement (stretch) per unit of force applied through a hand-held probe. To provide an indication of muscle tone, Leonard^{6,21} demonstrated high intra- and interrater reliabilities for measurements obtained from the biceps brachii and gastrocnemius muscles. Compliance measures were validated using the MAS

From the Motor Performance Laboratory, School of Rehabilitation Therapy, Queen's University, Kingston, ON, Canada.

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Reprint requests to Brenda Brouwer, PhD, Sch of Rehabilitation Therapy, Louise D. Acton Bldg, 31 George St, Queen's University, Kingston, ON K7L 3N6, e-mail: brouwerb@post.queensu.ca.

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Table 1: Subject Characteristics

Characteristics	Control (n=24)	Stroke (n=23)	Total (N=47)	P*
Age (y)	71.2±9.0	67.5±10.9	69.3±10.3	.210
Height (cm)	168.5±8.5	168.5±9.6	168.5±9.0	.987
Weight (kg)	70.7±10.3	76.3±14.1	73.5±12.6	.122
Time poststroke (y)	NA	4.6±3.3	NA	NA

NOTE. Values are mean ±1 standard deviation (SD).

Abbreviation: NA, not applicable.

*Based on independent Student *t* tests comparing control and stroke groups.

scores from the elbow flexors. Correlation coefficients were moderate to high, although it should be noted that both variables were reduced to categorical data before analysis due to clustering of the MAS scores.²¹ These findings suggest that the Myotonometer may be a useful clinical tool to characterize muscle tone, at least in the upper extremity. Validation of this device has not yet been reported for lower-limb muscles and cannot be inferred from data relating to the upper limb.

In the present study, we examined the construct validity of Myotonometer measurements of ankle plantarflexor tone in subjects with hypertonicity associated with hemiparesis and in control subjects of similar age. To determine to what degree compliance measures indicated total mechanical stiffness and, more specifically, reflex and nonreflex mediated stiffness, we correlated Myotonometer measurements with outcomes derived from a torque motor. As a clinical yardstick, MAS scores were also used as a correlate of tone. In this manner, we examined whether compliance measured by the Myotonometer was able to distinguish between groups of subjects with normal and excessive plantarflexor tone and to what extent the measures were associated with specific neuromuscular factors contributing to tone.

METHODS

This cross-sectional study required subjects to report to a motor performance laboratory once for approximately 1 hour.

Participants

Twenty-six stroke survivors from a hospital-based outpatient stroke clinic and stroke survivors support group currently living in the community responded to a request for volunteers having poststroke ankle stiffness on their paretic side and who had experienced their stroke at least 6 months before the study. Twenty-eight healthy volunteers were recruited from a spousal support group or the community at large. All were screened to exclude those reporting a history of orthopedic problems affecting the lower limbs (n=1) or neurologic disorders apart from stroke (n=2). Twenty-three people (14 men, 9 women) ranging in age from 45 to 86 years who had experienced a hemispheric stroke between 2 and 14 years before the study formed the stroke group. Eight had left hemispheric lesions and 15 had right hemispheric lesions. Twenty-four subjects (13 men, 11 women) between 52 and 83 years of age served as controls (table 1). All provided written informed consent prior to testing and protocols were approved by the Queen's University and Affiliated Hospitals Research Ethics Board.

Protocol

Subjects were seated comfortably on a padded plinth with their lower limbs hanging freely. The plantarflexor resistance felt in response to passive dorsiflexion was rated between 0 (no

increase in muscle tone) and 4 (test limb rigid) on the MAS. The same examiner (SJR) assessed all subjects.

For all remaining testing, subjects sat semireclined with their back supported and the test leg (randomly selected for control subjects and the paretic limb in stroke survivors) secured in a support frame such that the angle formed by the trunk and hip was approximately 90° and the knee was flexed at 45°. This position made it possible to test both muscle stiffness and compliance without having to reposition the subject. The lateral malleolus was aligned with the axis of rotation of the footplate, which was fitted with a potentiometer to measure angular position. Neutral position in which the foot formed a right angle with the shank was denoted as 0°. A strain gauge mounted on a cylinder coupling a torque motor to the footplate measured force.

The footplate was fixed such that the ankle was positioned at an angle of -17° (ie, plantarflexion). Subjects pushed against the footplate with the ball of their foot as hard as possible for 3 trials of approximately 5 seconds. The highest peak torque (in newton meters) generated during the 3 trials was recorded as the plantarflexor maximal voluntary contraction (MVC).

Ankle passive ROM (PROM) was measured by slowly moving the footplate into plantarflexion and dorsiflexion and recording the end range angles as the position of the ankle at the point at which resistance to further motion registered 10Nm. We based our decision to use this criterion on observations that at lower torque levels there is a risk of underestimating the available range and that higher levels fail to produce more than a few degrees of additional movement yet are less well tolerated.²² PROM was the mean difference between the dorsiflexion and plantarflexion end range angles over 3 trials. The angle at which the subject's foot rested naturally (with gravity) after passive dorsiflexion was also recorded.

Muscle stiffness testing. Total ankle stiffness reflects contributions from reflex activation associated with a change in muscle length (reflex stiffness), the elastic properties of attached cross-bridges (intrinsic stiffness), and the noncontractile elements (passive stiffness).^{11,12,23} To quantify the relative contribution of each component, various test conditions were introduced. All incorporated a 5° perturbation (displacement in a dorsiflexion direction) at a constant velocity of 100°/s controlled by a torque motor system. The start position was standardized at -17° to ensure that all subjects could comfortably achieve the starting ankle position and had sufficient residual mobility to accommodate the 5° displacement without discomfort. The resistance torque associated with the start position was recorded to serve as a reference value for the relaxed condition. One or 2 practice trials during which subjects were instructed to relax and not resist the movement of the footplate were introduced so that subjects could experience the perturbation and their comfort postperturbation could be assured.

Position and torque data were acquired (1kHz per channel) over a 1050-ms period that included 500-ms pre- and postperturbation segments. Five acceptable trials (<.25Nm fluctuation in baseline plantarflexor torque) were recorded for each of 3 test conditions: rest, voluntary activation, and involuntary activation (the order determined randomly by card draw for each subject). During the rest condition, subjects remained relaxed as indicated by a flat, unchanging baseline plantarflexor torque corresponding to the previously recorded reference value. The perturbation (5° at 100°/s) was introduced without warning and the resultant increase in ankle joint torque reflected passive stiffness attributed to the elastic properties of noncontractile tissue.^{11,23} Electromyographic activity of the dorsiflexors and plantarflexors remains unchanged when the relaxed plantarflexors undergo stretches of up to 180°/s.²⁴ For the voluntary

activation condition, subjects contracted their plantarflexors to a level equivalent to 10% of MVC and were instructed to maintain the force level throughout the trial. Visual feedback of the required (target) force was provided on a computer screen along with the subject's actual force production. When the force levels were matched, the ankle was perturbed. The corresponding increment in torque reflected the sum of all contributing factors, thus indicating the total mechanical stiffness of ankle extensors (total stiffness).^{11,23} For the involuntary activation condition, we used an isolated electric stimulator (Digitimer model DS2^b) to stimulate the posterior tibial nerve in the popliteal fossa with 1-ms square-wave pulses delivered at a frequency of 30Hz controlled by a trigger generator (Digitimer model D4030^b). The stimulus amplitude was adjusted until a sustained plantarflexor torque equivalent to 10% of MVC was generated. At this point the perturbation was introduced. The subsequent increase in torque related to nonreflex contributions because nerve stimulation suppresses the stretch reflex in the homonymous muscle.^{7,11} The period of stimulation did not exceed 5 seconds and rest was provided between trials to avoid fatigue. The resultant torque profiles associated with these conditions are in figure 1.

Data from all conditions were visually inspected to ensure that baseline torque values corresponded to either a relaxed state or 10% of MVC as was required for the condition tested. Data were then smoothed offline using a 50-Hz low-pass filter and processed to eliminate intermittent large amplitude spikes associated with the torque motor. Ankle stiffness (in Nm/deg) was calculated as the difference in mean torque levels measured over a 50-ms period during baseline and another 150ms after the perturbation relative to angular displacement (5°) for each of the 3 testing conditions. The mean of 5 trials for each condition was recorded.

To isolate the contribution from intrinsic factors, the difference between nonreflex stiffness (electric stimulation) and passive stiffness was calculated.⁷ Reflex-mediated stiffness reflected the difference between total and nonreflex stiffness.^{7,23}

Muscle compliance testing. The Myotonometer is a handheld electronic device interfaced with a laboratory computer. It consists of a metal probe instrumented with a linear array of transducers that is housed in a plastic sleeve. The probe was placed perpendicular to the skin surface, 3 finger-breadths proximal to the flare of the gastrocnemius muscle and pushed onto the skin overlying the test muscle. The force applied to the tissue by the probe was monitored by the transducers and an electric measurement circuit detected the amount of displacement occurring between the inner probe and the outer sleeve, thus providing the force-displacement characteristics of the underlying tissue. Three trials were recorded while subjects were relaxed and another 3 trials were done while they maintained an isometric plantarflexor contraction corresponding to 10% of MVC (visual feedback provided). Computational software recorded the tissue displacement (in millimeters) at force intervals of .25kg (2.5N) over a range from .25 to 2.0kg (20.0N), and the percentage difference in compliance between relaxed and contracted conditions at each force level was calculated. The percentage difference of the area under the force-displacement curves for each condition was also computed. A similar procedure for analysis is described elsewhere.²¹

Statistical Analysis

Between-group (stroke, control) comparisons of clinical measures (ROM, MVC), ankle stiffness (total, passive, intrinsic, and reflex components), and computed Myotonometer measures (percentage differences) were performed using Stu-

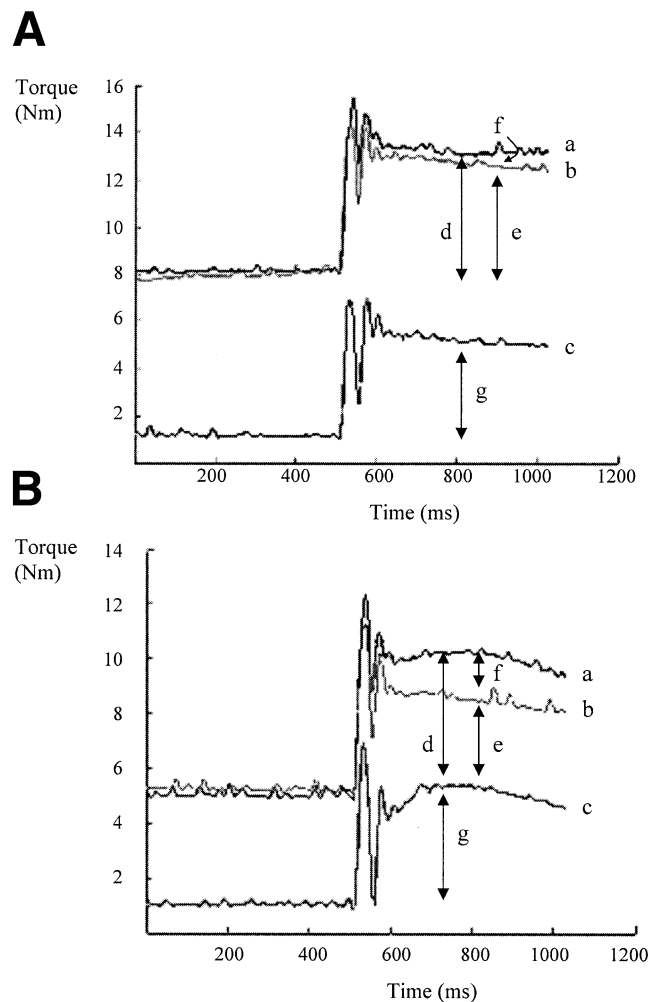


Fig 1. Resistance torque profiles from (A) a control subject and (B) a stroke survivor in response to a 5° perturbation introduced at 500ms. Ankle perturbations were introduced while subjects maintained a 10% of MVC (a) voluntarily and (b) involuntarily by stimulating the muscle nerve, and while they were (c) relaxed. Stiffness measurements derived from these profiles are illustrated: d, total mechanical stiffness; e, nonreflex stiffness; f, reflex stiffness ($f=d-e$); g, passive stiffness. Intrinsic stiffness was calculated as the difference between d and g (not shown). Note the different y-axis scales.

dent independent *t* tests, after statistical software^c confirmed that data were distributed normally. A multifactor, repeated-measures analysis of variance was used to analyze the Myotonometer data across force levels, groups (stroke, control), and condition (relaxed, contracted). We also compared percentage differences (relaxed vs contracted) across force levels and groups. Post hoc analysis (Tukey multiple comparisons test) was performed as appropriate to determine where differences lay. A Pearson correlation matrix was constructed to determine the degree of association between computed Myotonometer measures (percentage differences) and muscle stiffness. Spearman ρ coefficients indicated whether computed Myotonometer measurements correlated with MAS scores. For all analyses, the significance level was set at *P* less than .05.

RESULTS

All subjects completed the clinical assessment of plantarflexor tone and muscle function. Not surprisingly, all control

subjects scored 0 (no increase in tone) on the MAS. Their result was in contrast to the stroke group's in which a range of scores from 1 to 4 was observed, although clustering at the lower end was evident (19/23 subjects scored ≤ 2). Consistent with this finding was that the control group had 15° more PROM than the stroke group, which was mainly attributable to their greater mobility in dorsiflexion (control group: mean ROM ± 1 standard deviation [SD], $63.3^\circ \pm 16.1^\circ$; stroke group, $48.2^\circ \pm 11.1^\circ$; $P < .000$). However, the preferred resting ankle position was similar for both groups (control group, $-44.4^\circ \pm 8.9^\circ$; stroke group, $-41.3^\circ \pm 8.8^\circ$; $P = .234$).

In terms of plantarflexor strength, the control group (49.1 ± 14.9 Nm) generated almost twice as much isometric plantarflexor torque as the stroke group (27.0 ± 16.1 Nm) ($P < .0001$). Normalizing torque to body mass did not alter the findings.

Muscle Stiffness

Two subjects in the stroke group did not complete the muscle stiffness testing; one was unable to tolerate electric stimulation due to heightened sensitivity, and the other could not sustain the 10% of MVC required to quantify total stiffness. A complete series of torque profiles was obtained from 21 stroke survivors.

Figure 1 displays representative torque profiles associated with the 3 test conditions from 1 control subject and 1 stroke survivor. The corresponding stiffness measurements are also illustrated for these subjects. It was important when decomposing stiffness measures into specific components that the level of contraction associated with electric stimulation and voluntary muscle activity (10% of MVC) were matched. The difference between the preperturbation torque levels associated with these 2 conditions was $4.6\% \pm 3.0\%$ for control subjects and $6.5\% \pm 3.9\%$ for stroke subjects; in all cases differences were less than 10%.

The mean total ankle stiffness was almost 40% higher in the stroke group than in the control group ($P = .018$). This finding reflected the tendency for the mean stiffness values associated with each contributing element to be higher in stroke subjects, with the notable exception of reflex-mediated stiffness, which was similar for both groups. These data are summarized in figure 2.

Muscle Compliance

Myotonometer measures of tissue compliance were obtained from all participants in the stroke group and all but 4 in the control group (equipment problems). As expected, there was greater tissue displacement at higher levels of force application ($F = 109.276$, $P < .000$) and the amount of tissue displacement was less when the plantarflexors were contracted than when they were at rest ($F = 10.49$, $P = .002$). This latter finding was especially evident in control subjects, as reflected by the significant interaction between condition and group factors ($F = 2.31$, $P = .034$). Relative to the stroke group, the control had a mean compliance that appeared higher at all force levels when the muscle was relaxed and lower when contracted (figs 3A, 3B), although there was no main effect of group ($F = .046$, $P = .498$). Examination of the percentage difference in compliance measured with the muscle at rest and contracted did, however, reveal a significant difference between stroke and control subjects (fig 3C; $F = 7.98$, $P = .007$). Similarly, analysis of the area under the force-displacement curves generated under the 2 muscle conditions (relaxed and 10% of MVC) revealed greater percentage differences in the control group ($24.5\% \pm 13.7\%$) than the stroke group ($13.1\% \pm 10.7\%$) ($P = .022$).

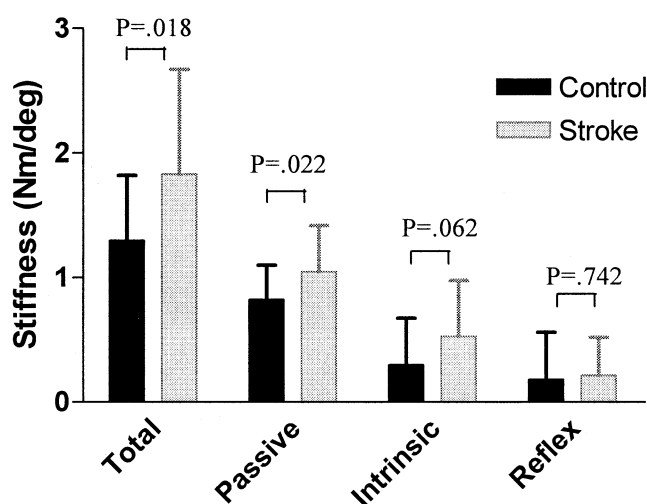


Fig 2. Mean +1 SD total mechanical stiffness of the ankle and the breakdown into contributing components. The *P* values indicate the significance of the between-group comparison.

Associations Between Myotonometer Measures and Other Tone Indicators

The percentage differences of the Myotonometer data recorded at rest and during a 10% of MVC were significantly associated with total ankle stiffness (r range, $-.428$ to $-.477$) for each force level. Smaller differences in compliance between muscle conditions reflected higher joint stiffness. This finding was primarily attributed to the contractile or intrinsic properties, because passive and reflex contributions were not related to differences in compliance to any significant degree (r range, $-.117$ to $-.289$). When only the stroke group's data were considered, the strength of the correlations generally increased (table 2).

Lower computed Myotonometer measures (differences between relaxed and active conditions) were associated with higher MAS scores (ρ range, $-.329$ to $-.490$). In the stroke group alone, the correlation coefficients were slightly higher, but only at the lower force levels. Table 2 summarizes these data.

DISCUSSION

The main findings of this study were that Myotonometer measurements of compliance distinguished between subjects with normal plantarflexor tone and strength and those with stroke-related hypertonicity and paresis. Further, computed Myotonometer measurements correlated with laboratory and clinical measures of ankle stiffness. Compared with control subjects, tissue compliance in subjects with chronic stroke was lower when the plantarflexors were relaxed and higher when the muscles were contracted. These findings are similar to those reported by others in relation to the biceps brachii muscle²¹ and account for the relatively small difference in area under the force-displacement curves generated at rest and during contraction compared with control subjects. As hypothesized by Leonard et al,²¹ these values best illustrate differences between the 2 subject groups (compare figs 3A–C) and reflect hypertonicity and weakness.

Muscle compliance (displacement per unit force) is the inverse of muscle stiffness (change in force per unit displacement). It therefore follows that if Myotonometer measurements distinguish between stroke and control groups, then quanti-

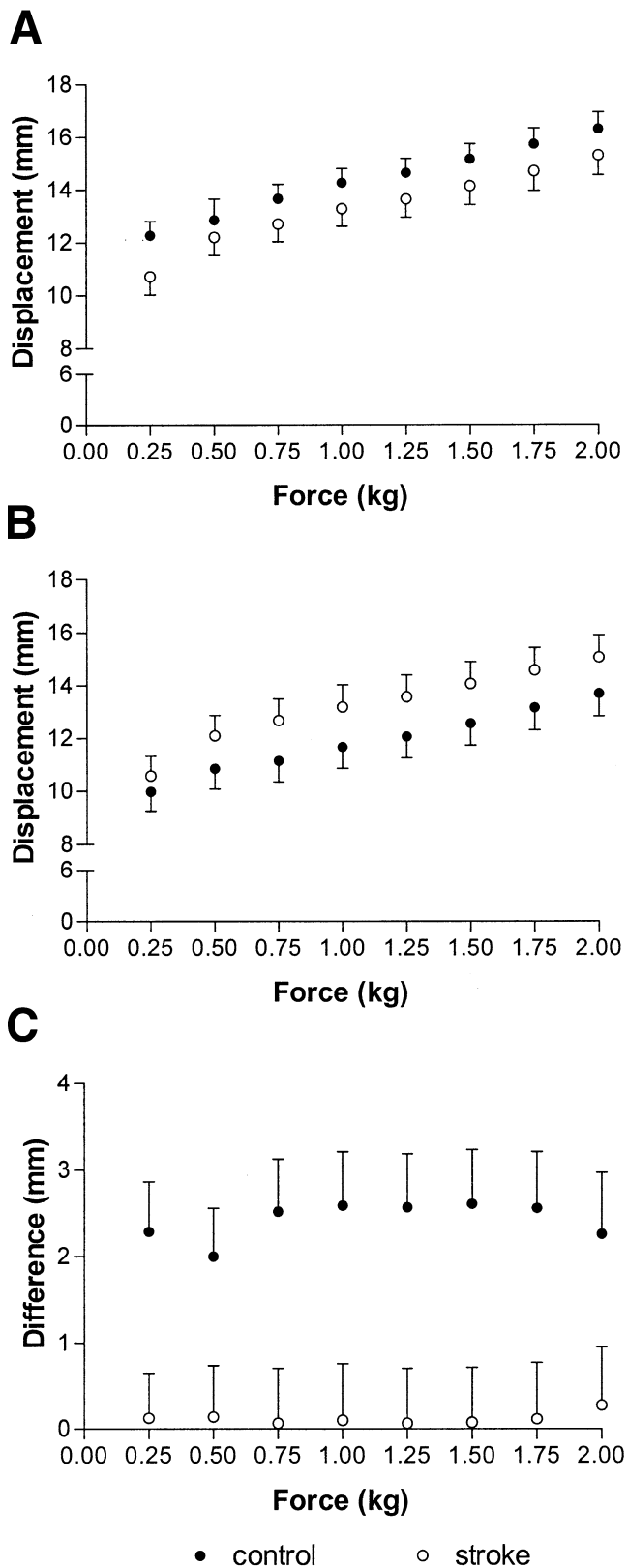


Fig 3. A comparison of the mean (1 SD) muscle compliance recorded at increasing levels of force for each group while the plantarflexors were (A) relaxed and (B) tonically active; and (C) the difference in compliance between the 2 muscle activation states.

cation of mechanical stiffness would yield similar findings. Furthermore, an inverse relationship between the 2 measurements would be expected. Indeed this was the case, although the strength of the association between compliance and stiffness measures was only moderate, suggesting that the 2 outcomes measured different aspects of tone or were adversely influenced by error.

As a tissue compliance gauge, the Myotonometer measures the penetration distance of the probe as a function of the force applied. Although muscle stiffness would certainly limit penetration, this stiffness would only occur after first compressing the more superficial tissues, including skin, vasculature, and subcutaneous fat, and would therefore require relatively higher forces.^{25,26} It is not known how much force is required to compress the superficial tissues to the point where displacement of muscle tissue occurs, although our data suggest that .25kg (2.5N) of force may be sufficient. This belief is based on the fact that in control subjects compliance decreased with muscle activation even at low levels of force applied through the probe and on the assumption that the muscle's state of activation (10% of MVC) would have a negligible effect on the compliance of superficial tissue. By extension, calculation of the difference in compliance between relaxed and contracted muscle states (residual displacement) reflects primarily alterations in muscle stiffness, because the remaining nonmuscle contributors (error source) would be removed. This explains why differences in compliance measured at rest and during contraction best distinguish between groups. Further, as the level of contraction during which compliance is measured increases to 100% of MVC, the paresis attributable to stroke would likely accentuate differences between hemiparetic subjects and controls, as shown by Leonard et al.²¹

In the stroke group, muscle activation did not produce a significant decrease in compliance. One explanation is that there may be a higher degree of subcutaneous adiposity in the paretic limb. This adiposity may have primarily accounted for the tissue displacement measured at all force levels. Iversen et al²⁷ presented functional magnetic resonance imaging data that demonstrated that adiposity thickness in the paretic limbs of hemiparetic subjects significantly exceeded that associated with the nonparetic side, although differences were more pronounced in the upper limbs. Although we did not measure the thickness of subcutaneous tissue overlying the plantarflexors in our subjects to provide an indication of fat content, we have no reason to believe that it differed between the 2 groups. Calculation of mean body mass indices, an indirect measure of overall adiposity, revealed similar values for the control ($25.0 \pm 2.9 \text{ kg/m}^2$) and stroke ($26.4 \pm 4.1 \text{ kg/m}^2$) groups. These values are slightly lower than the 27.7 kg/m^2 reported by Ryan et al²⁸ for chronic stroke survivors in whom no difference was found in the amount of subcutaneous fat present in the paretic and nonparetic legs. Also of note, if the compliance measures we obtained reflected mainly the displacement of nonmuscle tissue, thus accounting for the negligible difference between activation states, then no relationship with mechanical stiffness would be expected. The data state otherwise, and the correlations were consistently higher when only the stroke data were analyzed. The distinction between the control and stroke groups based on compliance differences likely reflects true differences in muscle stiffness.

It is axiomatic that validity can be compromised by inaccuracies of the measurement device or by poor reliability. The generally weak associations between computed Myotonometer measurements and MAS scores in stroke subjects likely reflect this notion. The qualitative nature of the MAS, poor reliability in testing plantarflexor tone, and clustering of ratings^{19,21} di-

Table 2: Correlations Between Computed Myotonometer Data (Percentage Difference Between Relaxed and Contracted Conditions) and Other Measures of Tone

Force (kg)	MAS*		Total		Passive		Intrinsic		Reflex	
	All	Stroke	All	Stroke	All	Stroke	All	Stroke	All	Stroke
0.25	-.397(.008)	-.453(.030)	-.476(.001)	-.568(.005)	-.248(.100)	-.253(.243)	-.310(.041)	-.540(.010)	-.182(.232)	-.436(.043)
0.50	-.311(.040)	-.404(.056)	-.455(.002)	-.526(.010)	-.141(.355)	-.215(.323)	-.377(.012)	-.543(.009)	-.283(.059)	-.360(.100)
0.75	-.378(.011)	-.412(.050)	-.463(.001)	-.505(.014)	-.235(.117)	-.180(.410)	-.354(.019)	-.536(.010)	-.255(.094)	-.360(.100)
1.00	-.386(.010)	-.412(.050)	-.469(.001)	-.517(.012)	-.249(.099)	-.196(.370)	-.353(.019)	-.532(.011)	-.259(.089)	-.393(.071)
1.25	-.412(.005)	-.364(.088)	-.472(.001)	-.529(.009)	-.272(.071)	-.228(.296)	-.364(.015)	-.542(.009)	-.227(.138)	-.375(.086)
1.50	-.399(.007)	-.372(.080)	-.474(.001)	-.523(.010)	-.278(.065)	-.221(.311)	-.366(.015)	-.541(.009)	-.224(.143)	-.378(.083)
1.75	-.364(.015)	-.319(.138)	-.477(.001)	-.529(.009)	-.289(.054)	-.243(.264)	-.365(.015)	-.534(.010)	-.221(.149)	-.374(.086)
2.00	-.323(.032)	-.285(.188)	-.428(.003)	-.493(.017)	-.278(.065)	-.207(.343)	-.306(.043)	-.510(.015)	-.188(.223)	-.343(.118)
AUC	-.316(.034)	-.305(.179)	-.348(.017)	-.444(.039)	-.114(.457)	-.065(.767)	-.324(.030)	-.470(.027)	-.206(.186)	-.363(.097)

NOTE. Significant coefficients are in bold face. *P* values are in parentheses.

Abbreviation: AUC, area under the curve.

*Due to the ordinal nature of the MAS, a Spearman ρ statistic was determined; a Pearson product-moment correlation coefficient was calculated for all other variable pairs.

minish the degree to which MAS ratings indicate the severity of tonal disorder. With respect to compliance measures, Leonard at al¹⁶ reported excellent intrarater (ICCs, $\geq .84$) and interrater (ICCs, $\geq .75$) reliability of Myotonometer measures from the lateral gastrocnemius muscle with probe forces ranging from 0.5 to 2.0kg (5.0–20.0N). Torque motor measurements of ankle stiffness yielded similarly high reliability coefficients. Two studies,^{13,22} each involving 10 subjects who were tested on multiple occasions, revealed that total mechanical ankle stiffness and its passive, intrinsic and reflex contributions were highly reliable (ICCs, $\geq .83$). These studies attest to the reliability of the compliance and stiffness measurement procedures and tools used in the present study. The significance of the associations between these outcomes indicates that they measure similar constructs, but perhaps not identical components.

The decomposition of total mechanical stiffness revealed that stroke survivors had abnormally high passive stiffness—a finding that has been attributed to alterations in connective tissue and the accumulation of collagen increasing fibrosis within the high tone muscle.^{29–31} It was this factor more than reflex stiffness or the intrinsic properties of muscle that contributed to the elevated total stiffness detected among stroke survivors. However, in the absence of recording electromyograms from ankle muscles, it is possible that subjects may not have been fully relaxed, resulting in the overestimation of passive resistance. In view of the consistency of the preperturbation resistance torque in the relaxed condition paired with the findings from others that electromyographic signals are negligible at corresponding speeds of stretch,²⁴ we do not consider unrelaxed muscle a viable explanation. Furthermore, our present findings are compatible with those from other studies,^{7,20} but suggest that, in chronic stroke, spasticity (increased reflex sensitivity) may not be a major contributor to hypertonia. Our data further indicate that reflex stiffness contributes little to the explained variance in compliance measures.

The difference in Myotonometer measures from relaxed and active plantarflexors was most strongly associated with intrinsic stiffness. This is reasonable, because it is the muscle's contractile properties that are reflected by calculating the difference measure. Intrinsic stiffness increases linearly with cross-bridge formation, which, in turn, is proportional to the amount of force generated by the muscle.^{32,33} Although stroke survivors were weaker than controls, at 10% of MVC the elastic properties attributable to the cross bridges formed did not differ statistically between the groups ($P=.062$), but this

may have been a function of the sample size. Indeed, the relation between compliance differences and intrinsic stiffness was strengthened when only stroke data were included in the analysis.

One limitation in validating Myotonometer measures with those derived from a torque motor is that the latter measures the *net* resistance torque. Such measures could include any combination of properties associated with gastrocnemius, soleus, and/or other tibial nerve-innervated muscles that assist plantarflexion. In contrast, the probe penetration of the Myotonometer was specific to the medial gastrocnemius muscle, and therefore changes in compliance attributed to activation would be detected only if the medial gastrocnemius was involved in generating the plantarflexor torque, which it clearly was in the current study. With the knee positioned at 45° of flexion, both gastrocnemius and soleus likely contributed to plantarflexor torque generation³⁴ and joint stiffness, but not Myotonometer measures of compliance. Electromyographic recordings would have enabled us to identify muscles actively contributing to plantarflexor torque production. Arguably, if the gastrocnemius was working in isolation, the strength of its relationship with Myotonometer measures would be expected to improve. Muscle substitution can compromise tests of validity¹⁶ and that factor probably contributed to a portion of the unexplained variance between muscle stiffness and Myotonometer measures in the present study.

CONCLUSIONS

Differences in tissue compliance measured by the Myotonometer in relaxed and active muscle distinguished between normal muscle tone and hypertonic muscle tone due to chronic stroke. The Myotonometer measures were related to total ankle stiffness and reflect primarily the intrinsic properties of the muscle tested. Because it is a portable device and relatively inexpensive, the Myotonometer could be a useful clinical tool to quantify tonal disorders associated with the contractile properties of muscle.

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Suppliers

- a. Neurogenic Technologies Inc, 5000 Pattee Canyon St, Missoula, MT 59803.
- b. Digitimer Ltd, 37 Hydeway, Welwyn Garden City Hertfordshire, AL7 3BE, England.
- c. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.