Biomechanic Changes in Passive Properties of Hemiplegic Ankles With Spastic Hypertonia

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Objective: To investigate quantitatively biomechanic changes in the passive properties of hemiplegic spastic ankles.

Design: Evaluation of spastic hypertonia by moving the ankle joint slowly between dorsiflexion and plantarflexion extreme positions under controlled joint torque and position.

Setting: Institutional research center.

Participants: Twenty-four stroke patients with spastic ankles and 32 healthy controls.

Interventions: Not applicable.

Main Outcome Measures: Passive resistance torque at controlled dorsiflexion and plantarflexion positions, dorsiflexion and plantarflexion range of motion (ROM) at controlled torques, and quasistatic stiffness and energy loss in dorsiflexion and plantarflexion.

Results: Spastic hypertonic ankles showed significant alterations in the passive properties in plantarflexion (P < .041) as well as in dorsiflexion (P < .016) directions. Compared with healthy controls, spastic ankles showed higher resistance torque (9.51 ± 4.79 Nm vs 6.21 ± 3.64 Nm, P < .016), higher quasistatic stiffness (.54 ± 0.19 Nm/deg vs .35 ± 0.20 Nm/deg, P < .001) at 10° of dorsiflexion, larger normalized dorsiflexion energy loss (.068 ± 0.04 J/deg vs .04 ± 0.02 J/deg, P < .037), and decreased dorsiflexion ROM at 10 Nm of resistance torque (10.77° ± 8.69° vs 20.02° ± 11.67°, P < .014). The resistance torque, ROM, and stiffness of spastic hypertonic ankles in plantarflexion showed similar changes (P < .05) to those in dorsiflexion. The passive ROM, joint stiffness, and resistance torque at controlled positions correlated with each other and also correlated with the Modified Ashworth Scale (P < .01).

Conclusions: Various biomechanic changes in both plantar- and dorsiflexors are associated with spastic hypertonia of chronic stroke patients, and they can be evaluated quantitatively under well-controlled conditions. With simplifications, the various measures in this study can potentially be used to obtain more comprehensive and quantitative evaluations of spastic hypertonia in a clinical setting.

Key Words: Ankle; Contracture; Hemiplegia; Muscle spasticity; Rehabilitation.

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Despite the clinical significance of spastic hypertonia, its underlying mechanisms are often not clear.1-4 The increased mechanical resistance to passive movement may be related to hyperactive reflexes and/or caused by nonreflex biomechanical changes in muscles and connective tissues.2 Some investigators5-10 have shown that the increased resistance in spastic limb movement is mainly caused by hyperactive reflexes, as shown in exaggerated tendon jerks and increased H-reflex responses. On the other hand, other investigators11-17 believe that spastic hypertonia is independent of hyperactive reflexes, and mechanical changes of muscles are the main reasons for the increased muscle tone in spasticity. Furthermore, the nonreflex contributions include the dynamic component of viscous damping (dashpot-like property with resistance proportional to velocity) and static component of elastic stiffness (spring-like property with resistance proportional to displacement), and reflex changes may have both phasic (dynamic) and tonic (static) components. The different components may contribute to the increased resistance in passive movement of spastic limbs. It is often not clear whether each of these components is enhanced in spastic limbs or not.2,3,5,10,11,15,18

Spastic hypertonia at the ankle joint is a major source of disabilities after stroke. Both reflex and nonreflex changes in ankles with spastic hyper- tonia can substantially affect the functional performance of stroke patients. Several studies11,19-22 have suggested that nonreflex changes had more profound and consistent effects than did reflex changes. Moreover, some argued that changes in ankle passive biomechanic properties could contribute to the internal ankle joint torque in functional movement, depending on the severity of spasticity.22,24 There is a need for more precise evaluation and comprehensive understanding of the passive biomechanic changes in hemiplegic ankles. Although there have been many methods to evaluate reflex changes in spasticity such as the tendon reflex and H-reflex tests, less work has been done to quantify passive mechanical changes of spastic muscles and joints comprehensively over large samples in both plantarflexion and dorsiflexion. Only a few studies25-27 were carried out using small samples to evaluate some of the biomechanic changes in spastic ankles with focus on the plantarflexors.

The purpose of this study was to investigate changes in passive biomechanic properties of both plantarflexors and dorsiflexors in ankles with spastic hypertonia by using a well-controlled device, including passive resistance torque at common ankle positions, passive range of motion (ROM) at controlled resistance torque, passive elastic stiffness, energy loss involving viscoelasticity, and correlations of the above quantitative measures with the Modified Ashworth Scale28-30 (MAS).
METHODS

Participants

Twenty-four stroke patients (15 men, 9 women) with a mean age \( \pm \) standard deviation (SD) of 55.3 \( \pm \) 10.1 years participated in the study. All patients had hemiparesis caused by cerebro-vascular accidents at least a year before the experiment (9\( \pm \)5.7y of mean duration of hemiparesis and evidence of supratentorial lesion in all cases, with hemorrhage in 11 and ischemia in 13 patients) and spastic hypertonia in ankles of the involved sides as determined by physical examination including motor, sensory, and reflex examinations and the MAS. \(^{28-30}\) The MAS was conducted at 60\(^{\circ}\) of knee flexion, the same position as in the experiment. Patients who did not have spastic ankles were excluded from the study if they had less than a grade 3 score of the Achilles’ tendon reflex and a score of 0 on the MAS (range, 0–4). Subjects who had previous ankle injury, surgery, or any kind of neurolytic injections were excluded. Thirteen subjects had left side weakness, and 11 subjects had right hemiparesis. Thirty-two healthy subjects (17 men, 15 women; mean age, 42.1\( \pm \)20.5y) were included as controls. None of the control subjects had sustained injury or had had surgery on the foot or ankle. The study was approved by the institutional review board of Northwestern University. All subjects gave informed consent before the experiment.

Experimental Setup

The evaluation was done by using a custom-designed joint driving device (fig 1). The joint driving device moved the ankle at a well-controlled speed, and it slowed down as resistance torque increased. In this way, the joint muscles were moved under controlled load, and reflex-mediated responses were minimized. \(^{31}\) Subjects were seated with the thigh and trunk strapped to the seat and backrest, respectively. The leg was strapped to the leg support and fixed at 60\(^{\circ}\) of knee flexion angle. The foot was held firmly to a footplate by using a premolded plastic cast and clamps. The footplate was mounted onto the motor shaft through a 6-axis force sensor that measured the torques at the ankle joint. The seat was adjusted and locked in 4 degrees of freedom, and the footplate could be adjusted in the toe-heel, mediolateral, and superior-inferior directions to align the ankle flexion axis with the motor shaft and the axis of the 6-axis force sensor. The ankle flexion axis was assumed to pass through the inferior tip of medial malleolus, perpendicular to the sagittal plane of lower leg. Surface electrodes were attached on the bellies of tibialis anterior, medial and lateral gastrocnemius, and soleus muscles to monitor electromyographic activities during the passive movement. Subjects were asked to relax as much as they could, and electromyographic signals were used to monitor muscle activation during the passive movement.

Protocol

Neutral ankle joint position was determined by positioning the sole of the foot at 90\(^{\circ}\) with respect to the long axis of the lower leg. To measure initial offset torque of the ankle joint, the footplate was fixed at the neutral position or at a position as close to neutral as possible without stretching the potentially stiff ankle joints. The initial offset torque at the neutral position was measured while the subject was asked to relax.

The ankle joint was moved passively in both dorsiflexion and plantarflexion directions by the joint-driving device, which was controlled digitally based on position/velocity and torque feedback. Torque limits were set for both directions of passive movement at 10Nm, with the initial torque offset subtracted. For safety purpose, position limits were determined by manual range of motion (ROM) measurement and set for both the dorsiflexion and plantarflexion directions. The joint-driving device moved the ankle passively and repeatedly in both directions in 90-second trials. The ankle flexion, 6-axis forces and torques, and dynamic electromyographic signals from the tibialis anterior, soleus, and medial and lateral gastrocnemius muscles were recorded at 500Hz, after antialiasing filtering with the cutoff frequency of 230Hz.
Data Analysis

**Resistance torque, gravity compensation, and offset adjust.**
The force and torque signals measured from the 6-axis force sensors were transformed into anatomic joint torques, with the passive resistance torque generated by ankle plantarflexors as positive. Initial torque offset measured at the beginning of the experiment was subtracted from the joint torque. The gravitational force of the foot and the footplate was calculated and compensated at each position within the ROM. The weight and center of mass of the foot were calculated from the anthropometric data including the body weight, foot length, and the width and height of the malleolus measured from the subject.  

**Torque-angle curves (hysteresis loops).** The anatomic joint torque and angle were plotted to get torque-ankle hysteresis loops. The number of hysteresis loops during each passive movement trial ranged from 4 to 6, depending on the ROM of the subject. Each hysteresis loop was divided into 2 limbs, the ascending limb for dorsiflexion direction movement and the descending limb for plantarflexion direction movement. Each limb of multiple hysteresis loops was averaged to generate 1 representative hysteresis loop (averaged torque-angle curve) for each subject (fig 2). The torques corresponding to every 1° of joint angle in either the upper or lower limbs of the hysteresis loops were averaged to reduce multiple hysteresis loops into a single representative hysteresis loop (averaged torque-angle curve) for each subject (see fig 2, row 4). Several parameters were obtained from the averaged torque-angle curves to characterize the passive biomechanic properties of spastic hypertonic ankles: the passive resistance torque at controlled positions, the passive dorsiflexion and plantarflexion ROMs at controlled resistances, quasistatic stiffness, and normalized dorsiflexion and plantarflexion energy loss related to the viscoelastic properties of the joint. All the parameters were measured in both the ascending and descending limbs of each hysteresis loop to evaluate the properties of ankle dorsiflexors and plantarflexors.

**Passive resistance torque.** Because the joint torque measured by the 6-axis force sensor was the resisting torque of ankle joints to the passive movement, the torque corresponding to each ankle angle of an averaged torque-angle curve for either
angular movement (passive torque increment during a certain amount of ankle where K is the quasistatic stiffness (spring-like property characterized by the elastic stiffness of the spring) and $\Delta \theta$ is the passive torque increment during a certain amount of ankle angular movement ($\Delta \theta$). As $\Delta \theta$ becomes infinitely small, the quasistatic stiffness approaches the slope of a tangential line of the torque-angle curve at a specific ankle position. Quasistatic stiffness was calculated at every 1° of ankle angle in the ROMs of the averaged torque-angle curves in both dorsiflexion and plantarflexion directions by taking the slope of the regression curve to fit 6 data points (3 points before and after) around a specific ankle angle. Quasistatic stiffness of ankle plantarflexor (stiffness in dorsiflexion direction movement) was evaluated at 10° of dorsiflexion and that of ankle dorsiflexor (stiffness in plantarflexion direction movement) at 30° of plantarflexion (fig 3). To assess the differences of quasistatic stiffness of the ankle joint in a continuous profile, quasistatic stiffness for either dorsiflexion or plantarflexion direction was compared between the stroke and control groups at each ankle angle at a 1° interval throughout ankle ROMs.

Normalized dorsiflexion and plantarflexion energy loss. Because the area under the upper limb of the hysteresis loop represents the energy needed to move the muscle-tendon unit in a circular arc around a specific ankle angle. Quasistatic stiffness of ankle plantarflexor (stiffness in dorsiflexion direction movement) was evaluated at 10° of dorsiflexion and that of ankle dorsiflexor (stiffness in plantarflexion direction movement) at 30° of plantarflexion (fig 3). To assess the differences of quasistatic stiffness of the ankle joint in a continuous profile, quasistatic stiffness for either dorsiflexion or plantarflexion direction was compared between the stroke and control groups at each ankle angle at a 1° interval throughout ankle ROMs.

**Statistical Analysis**

To examine whether passive biomechanic properties of ankle plantarflexors and dorsiflexors were changed in spastic hypertonia, the 4 parameters characterizing the passive properties of ankle joints—(1) the passive resistance torque at controlled positions, (2) the dorsiflexion and plantarflexion ROMs measured at controlled torques, (3) quasistatic stiffness, and (4) normalized energy loss—were compared between the spastic hemiplegic ankles and controls. Because spastic hyper...
Passive Properties in Dorsiflexion Direction Movement

The parameters of the passive properties in the dorsiflexion direction movement showed a significant difference between the stroke and control group by the MANOVA test (<i>P</i> pertaining to Pillai trace=.016). All of the subsequent univariate ANOVAs for each parameter also demonstrated meaningful differences between the 2 groups: spastic hypertonic ankles showed higher passive resistance torque at the common 10° of dorsiflexion, larger normalized dorsiflexion energy loss (0.06±.014J/deg vs .04±.02J/deg, <i>P</i>=.037), and decreased dorsiflexion ROM at a controlled 10Nm torque (10.77°±8.69° vs 20.02°±11.67°, <i>P</i>=.014) than the controls (fig 5).

Passive Properties in Plantarflexion Direction Movement

In plantarflexion direction movement, the passive properties as a whole also showed a significant difference between the stroke and control groups (MANOVA, <i>P</i> pertaining to Pillai trace=.041). Subsequent univariate ANOVA tests for each parameter revealed significantly higher passive resistance torque (−1.90±1.84Nm vs −3.58±1.92Nm, <i>P</i>=.038), higher quasistatic stiffness (.20±.14Nm/deg vs .11±.09Nm/deg, <i>P</i>=.001) at 30° of plantarflexion, and decreased plantarflexion ROM at a −3Nm torque (36.23°±7.63° vs 46.01°±9.65°, <i>P</i>=.002) in the stroke group than in the control group. The
normalized plantarflexion energy loss was not statistically significant (.03±.01J/deg in stroke vs .02±.01J/deg in controls, P=.765; fig 6).

Continuous Profiles of Passive Torques and Stiffness Throughout Ankle ROMs

For investigation of continuous profiles of passive properties, the passive resistance torque during each direction movement was averaged and compared between the 2 groups at every 1° of ankle angle (fig 4). The stroke group showed higher passive resistance torque especially at 4° and higher of dorsiflexion ROMs in the upper graph (dorsiflexion direction movement) and showed higher resistance torque at −28° and lower of plantarflexion ROMs in the lower graph (plantarflexion direction movement) than control group (independent-samples t test, P<.05). Although the passive resistance torques at each joint angle differed statistically only in extreme ROMs, the quasistatic stiffness was significantly higher in the spastic ankles throughout almost the whole ROMs in both dorsiflexion and plantarflexion directions (fig 4).

Correlations Between the MAS Scores and the 4 Biomechanic Parameters

The MAS scores of the spastic hypertonic ankle plantarflexors showed significant correlations with the quantitative parameters of passive properties, except for the normalized energy loss (table 1). Among the significant correlations, the Kendall τ between the MAS and the passive resistance torque at 10° of dorsiflexion (τ=.255), dorsiflexion ROM at 10Nm (τ=.323), and quasistatic stiffness at 10° of dorsiflexion (τ=.312) were relatively low, whereas the 4 quantitative biomechanic parameters had strong correlations between each other with the Pearson r of −.895 between the dorsiflexion ROM and passive torque, r of .687 between the stiffness and passive torque, and r of −.721 between the stiffness and dorsiflexion ROM (P<.01).

DISCUSSION

Passive properties of spastic hypertonic ankles in stroke patients were investigated and compared with their counterparts in healthy subjects by moving ankle joints passively under precise control without provoking considerable reflex-mediated electromyographic responses. Spastic hypertonic ankles showed significant alterations of the passive properties in both dorsiflexion and plantarflexion directions. In the dorsiflexion direction, where the ankle plantarflexors were stretched, the spastic group showed increased quasistatic stiffness and passive resistance torques, decreased dorsiflexion ROMs, and larger normalized energy loss. In the plantarflexion direction movement, where ankle dorsiflexors were preferentially stretched, the 4 parameters of spastic hypertonic ankles showed similar changes as in the dorsiflexion direction movement, except the increased normalized energy loss was not significant. Continuous profiles of passive resistance torques and quasistatic stiffness showed that the passive resistance torques differed only at extreme ROMs, whereas the quasistatic stiffness differed across almost the whole ROM. The 4 parameters of altered passive properties had strong and significant corre-
lations with each other, whereas weaker but still significant correlations were found between the MAS of spastic hypertonic ankle plantarflexors and the quantitative parameters of passive properties in the dorsiflexion direction movement. These findings indicate that there are significant changes in the passive mechanical properties in spastic hypertonia of chronic stroke patients in both dorsiflexion and plantarflexion directions, which correlated with the routine clinical measurement of the MAS.

Reflex and/or Nonreflex Changes of Spastic Hypertonia
It has been reported that spastic hypertonia is associated with reflex5-10 and/or nonreflex changes.11-13 The former is supported by the exaggerated tendon jerks and the increased H-reflex response5-10 and the latter is associated with mechanical changes.11-13 Several reports supported the predominant contribution of nonreflex component. Dietz et al11,12 have suggested that changes in mechanical muscle properties were mainly responsible for muscle hypertonia. Lee et al13 reported that, for voluntarily activated muscles of spastic hemiparetic patients, the stretch reflex gains of spastic and contralateral limbs did not differ significantly. O’Dwyer et al14,15 reported that hypertonia in the upper limbs of stroke patients within 13 months of their stroke was associated with contracture but not with reflex hyperexcitability. Sinkjaer et al16,17 reported that spastic muscles in stroke patients had an increased nonreflex

<table>
<thead>
<tr>
<th>Measures</th>
<th>MAS</th>
<th>Resistance Torque at 10° of DF</th>
<th>DF ROM at 10Nm</th>
<th>Stiffness at 10° of DF</th>
<th>DF Energy Loss</th>
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<tr>
<td>MAS</td>
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<td>-.380†</td>
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<td>.230*</td>
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<tr>
<td>Resistance torque at 10° of DF</td>
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<tr>
<td>DF ROM at 10Nm</td>
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<td>DF energy loss</td>
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<td>.438†</td>
<td>-.407†</td>
<td>.223*</td>
<td>1.00</td>
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NOTE. The Kendall ρ values are in italics (calculated for the correlations between the MAS scores and quantitative parameters), and the Pearson coefficients are in roman (for the correlations among the quantitative parameters). The correlation analysis was done only with the parameters in the dorsiflexion (DF) direction, because the MAS was measured in the ankle plantarflexors. A Pearson r= .353 or a Kendall ρ= .346 with P<.01 was considered significant.

*P<.05.
†P<.01.
stiffness but that reflex-mediated stiffness during sustained voluntary contraction did not differ significantly from normal subjects. In our study, passive properties were measured under well-controlled conditions by moving the ankle without activating the reflex component, which showed significant alterations of passive properties in the spastic hypertonic ankles in hemiparesis. Furthermore, the alterations were found in both dorsiflexion and plantarflexion.

Correlations Between Clinical Measurements and Altered Passive Properties

The MAS is the most widely used method for assessing muscle spasticity in clinical practice and research. However, controversial results were reported with regard to the properties being measured by the MAS. Although it was reported that the MAS was influenced more by a velocity-dependent response of spasticity than passive structure,14,28 a conflicting result has been reported recently that the MAS measures muscle hypertonia rather than spasticity.35 In our study, the Pearson correlation coefficients among the 4 parameters were significantly strong, except for the energy loss, but the correlations (by the Kendall τ) between the MAS and the passive properties were significant but not as strong as the relationships among the quantitative parameters when we considered the level of a strong correlation defined as a Pearson r greater than .512 or a Kendall τ greater than .340 at P less than .01. These findings indicate that the MAS as a clinical measurement could reflect the alterations in the passive properties of spastic hypertonic ankles in part but not as good as quantitative measurements would. Better ways to quantify passive biomechanic properties are needed whether they would be simple or sophisticated.

Comparison With Previous Studies

The PROM of hemiplegic ankles in our study, 10.77°±8.69° at a 10Nm resistance torque, was comparable to the results reported by Singer et al.27 The PROM of the control group (n=18; 19.0°±1.9°) of Singer27 was also similar to our result (n=32; 20.02°±11.67°). The quasi-static stiffness of ankle plantarflexor measured at 10° of dorsiflexion (.54Nm/deg for stroke, .35Nm/deg for control) was comparable to the 2 previous studies, which reported 4.4Nm/10°25 and .53Nm/deg27 in hemiplegic ankles and 3.6Nm/10°25 and .44Nm/deg27 in controls. Harlaar et al25 reported lower stiffness in both hemiplegic and unaffected contralateral ankles than that reported by Singer27 or by our study, possibly because they measured the stiffness in a wider ROM that included neutral ankle position.

On the other hand, little work has been published about the stiffness or PROM of ankle dorsiflexors in hemiplegic ankle joints, which was investigated in our study (table 2).

CONCLUSIONS

Spastic hypertonic ankles showed significant alterations of passive biomechanic properties in dorsiflexors as well as in plantarflexors, including decreased ROM at controlled torques, increased resistance at controlled positions, and increased stiffness and energy loss. The biomechanic measures also correlated with the routine clinical measurement of the MAS. With simplifications and using a portable device, the various measures in this study can potentially be used to obtain more comprehensive and quantitative evaluation of spastic hypertonia in a clinical setting.

References


Suppliers
a. JR3 Inc, 22 Harter Ave, Woodland, CA 95776.

b. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.