

# Biomechanic Changes in Passive Properties of Hemiplegic Ankles With Spastic Hypertonia

Sun G. Chung, MD, PhD, Elton van Rey, PT, Zhiqiang Bai, Elliot J. Roth, MD, Li-Qun Zhang, PhD

**ABSTRACT.** Chung SG, van Rey E, Bai Z, Roth EJ, Zhang L-Q. Biomechanic changes in passive properties of hemiplegic ankles with spastic hypertonia. *Arch Phys Med Rehabil* 2004; 85:1638-46.

**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 of 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 ( $9.51 \pm 4.79\text{Nm}$  vs  $6.21 \pm 3.64\text{Nm}$ ,  $P=.016$ ), higher quasistatic stiffness ( $.54 \pm .19\text{Nm/deg}$  vs  $.35 \pm .20\text{Nm/deg}$ ,  $P=.001$ ) at  $10^\circ$  of dorsiflexion, larger normalized dorsiflexion energy loss ( $.068 \pm .04\text{J/deg}$  vs  $.04 \pm .02\text{J/deg}$ ,  $P=.037$ ), and decreased dorsiflexion ROM at  $10\text{Nm}$  of resistance torque ( $10.77^\circ \pm 8.69^\circ$  vs  $20.02^\circ \pm 11.67^\circ$ ,  $P=.014$ ). 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.

© 2004 by the American Congress of Rehabilitation Medicine and the American Academy of Physical Medicine and Rehabilitation

**D**ESPITE THE CLINICAL SIGNIFICANCE of spastic hypertonia, its underlying mechanisms are often not clear.<sup>1-4</sup> 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.<sup>2</sup> Some investigators<sup>5-10</sup> 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 investigators<sup>3,11-17</sup> 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.<sup>2,3,5,10,11,15,18</sup>

Spastic hypertonia at the ankle joint is a major source of disabilities after stroke. Both reflex and nonreflex changes in ankles with spastic hypertonia can substantially affect the functional performance of stroke patients. Several studies<sup>11,19-22</sup> 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.<sup>22-24</sup> 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 studies<sup>25-27</sup> 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 (PRM) at controlled resistance torque, passive elastic stiffness, energy loss involving viscoelasticity, and correlations of the above quantitative measures with the Modified Ashworth Scale<sup>28-30</sup> (MAS).

From the Rehabilitation Institute of Chicago, Chicago, IL (Chung, van Rey, Bai, Roth, Zhang); Departments of Physical Medicine & Rehabilitation (Chung, van Rey, Bai, Roth, Zhang), Orthopaedic Surgery (Zhang), and Biomedical Engineering (Zhang), Northwestern University, Chicago, IL; and Department of Rehabilitation Medicine, Seoul National University, Seoul, South Korea (Chung).

Presented in part at the 4th World Congress of Biomechanics, August 4-9, 2002, Calgary, AB, Canada, and at the 2nd World Congress of the International Society of Physical and Rehabilitation Medicine, May 18-22, 2003, Prague, Czech Republic.

Supported by the National Institute on Disability and Rehabilitation Research and National Institutes of Health.

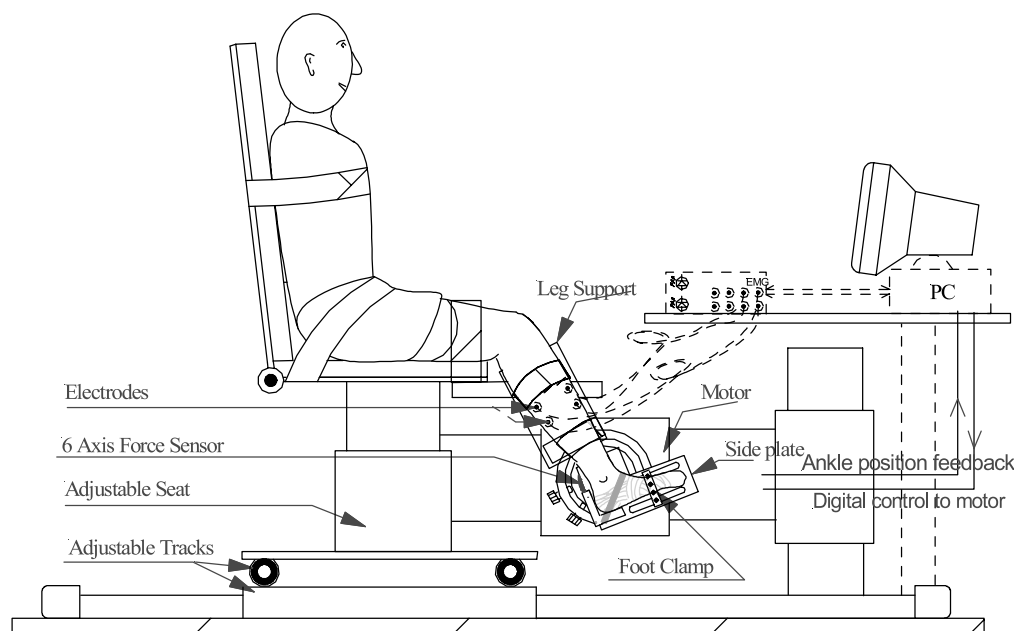
No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the author(s) or upon any organization with which the author(s) is/are associated.

Reprint requests to Sun G. Chung, MD, PhD, Sensory Motor Performance Program, Rehabilitation Institute of Chicago, 345 E Superior St, Ste 1406, Chicago IL, 60611, e-mail: [sungc@plaza.snu.ac.kr](mailto:sungc@plaza.snu.ac.kr).

0003-9993/04/8510-8651\$30.00/0

doi:10.1016/j.apmr.2003.11.041

**Fig 1. Experimental setup for evaluating ankle biomechanic properties.** The seat was adjusted in 4 degrees of freedom to align the ankle flexion axis with the motor shaft. A 6-axis force sensor was mounted between the motor shaft and the foot attachment. The foot and cast were clamped and strapped to the attachment with appropriate alignment. The leg was strapped to the leg support at 60° of knee flexion. The thigh and trunk were strapped to the seat and backrest, respectively, with the hip at 85° of flexion. Abbreviations: EMG, electromyograph; PC, personal computer.



## 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 cerebrovascular accidents at least a year before the experiment ( $9 \pm 5.7$  y 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.<sup>28-30</sup> The MAS was conducted at 60° 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,  $42.1 \pm 20.5$  y) 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

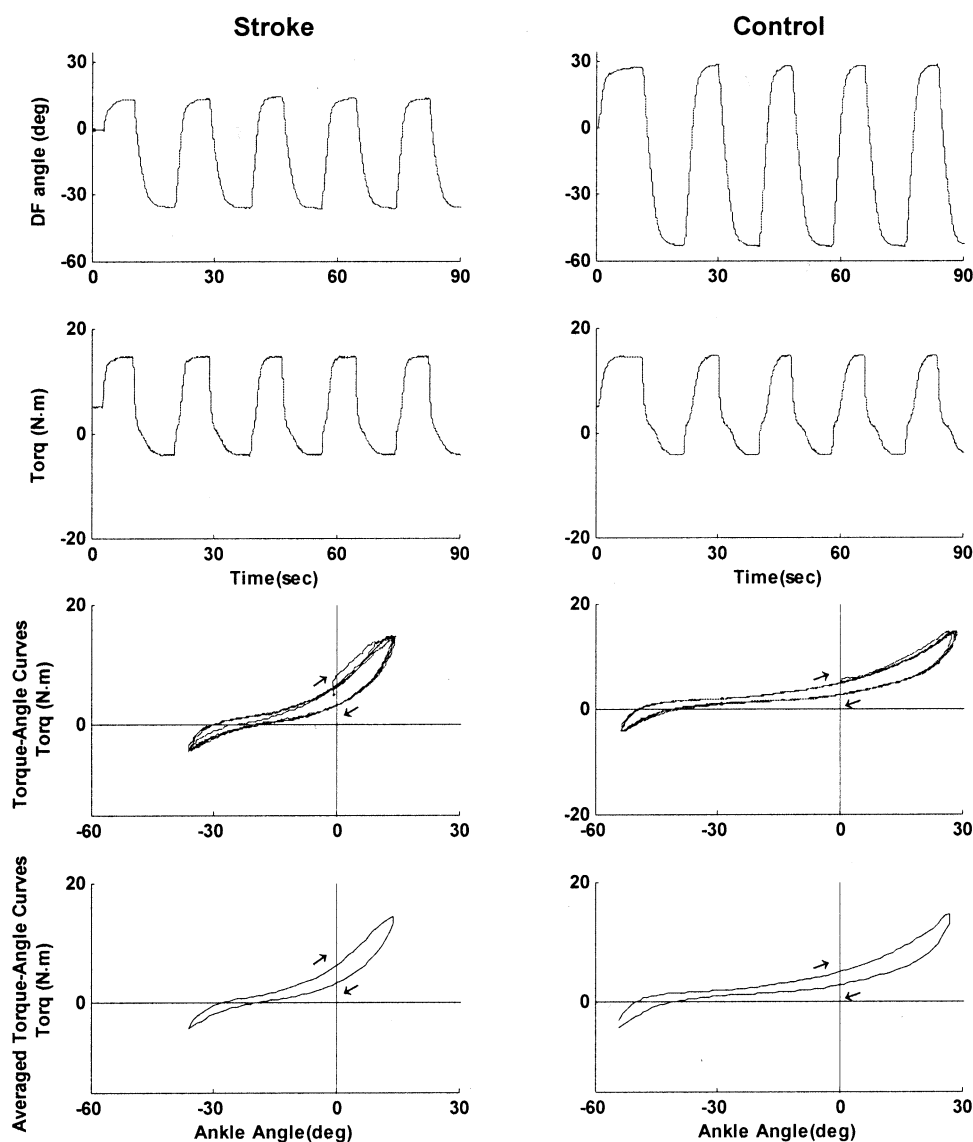
The evaluation was done by using a custom-designed joint driving device (fig 1). The joint driving device moved the ankle at a well-controlled speed, and it slowed down as resistance torque increased. In this way, the joint muscles were moved under controlled load, and reflex-mediated responses were minimized.<sup>31</sup> 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° 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<sup>a</sup> 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° 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.



**Fig 2.** Representative signals during passive movement trials on a stroke patient (left column) with a spastic ankle and an age- and sex-matched control (right column). The top and the second rows correspond to the ankle joint angle (positive for dorsiflexion [DF]) and ankle joint torque (positive for plantarflexion resistance torque), respectively. The joint torque is gravity compensated and offset (at the 0° dorsiflexion angle) adjusted. The trial lasted 90 seconds and the joint was held at the extreme ROMs for 3 seconds. The plots at the third and bottom rows show the raw (over multiple cycles) and averaged torque-angle curves, respectively. The x axis is the dorsiflexion angle and the y axis is the average resistance torque. Averaged torque-angle curves were calculated by averaging the torque values at every 1° of the ankle position to represent the viscoelastic properties of each ankle with a hysteresis loop. The curve moves clockwise as time progresses as indicated by the arrows.

## 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.<sup>32</sup>

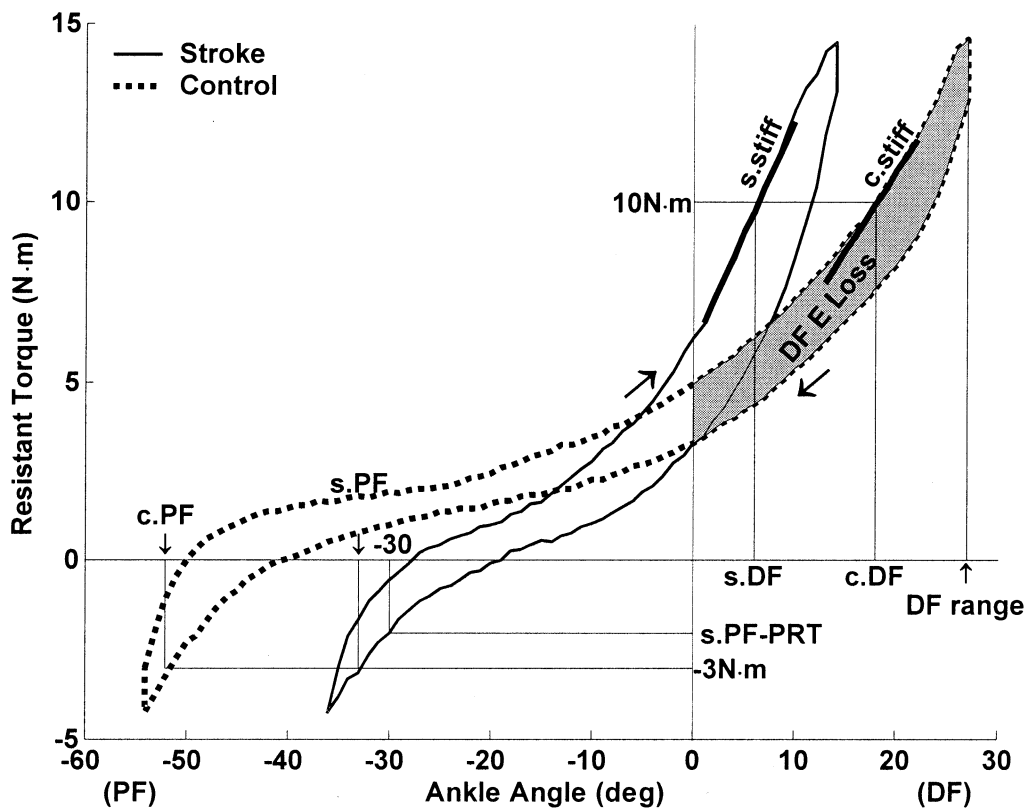
**Torque-angle curves (hysteresis loops).** The anatomic joint torque and angle were plotted to get torque-ankle hysteresis loops. The number of hysteresis loops during each passive movement trial ranged from 4 to 6, depending on the ROM of the subject. Each hysteresis loop was divided into 2 limbs, 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

Fig 3. Representative averaged torque-angle curves (hysteresis loops) from a stroke and a healthy subjects. The x axis is the dorsiflexion angle, and the y axis is the passive resistance torque. The curve moves clockwise as time (and the passive movement) progresses, as indicated by the 2 bigger arrows. Dorsiflexion ROMs (s.DF [stroke] c.DF [control]) taken at 10Nm of the passive plantarflexor resistance torque and plantarflexion (PF) ROMs (s.PF [stroke] c.PF [control]) at -3Nm of the passive dorsiflexor resistance torque are shown. The s.PF-PRT point is an example of passive resistance torque measured at a -30° (plantarflexion) ankle angle in the stroke subject. The slopes of 2 curve-fitted lines (s.stiff, c.stiff) are the quasistatic stiffness of the plantarflexor at the corresponding torque levels. The shaded area (DF E Loss) is the energy loss in dorsiflexion position, which is divided by the dorsiflexion ROM (dorsiflexion range) to get normalized dorsiflexion energy loss. Normalized plantarflexion energy losses are calculated similarly.



direction of movement was regarded as the passive resistance torque at that ankle angle. The passive resistance torque of a stroke subject at -30° ankle angle, for example, was indicated by the s.PF-PRT point (as defined in fig 3). The passive resistance torque of ankle plantarflexor (resistance torque to dorsiflexion direction movement) was measured at 10° of dorsiflexion and that of ankle dorsiflexor (resistance torque to plantarflexion direction movement) was sampled at 30° of plantarflexion for comparison between the groups. In addition to these sampled passive resistance torques, torque-angle curves for dorsiflexion and plantarflexion directions were compared between the stroke and control groups at each ankle angle at a 1° interval to assess the alterations of passive torques in a continuous profile (fig 4).

**Passive dorsiflexion and plantarflexion ROM.** PROMs were defined as the ankle joint angles at controlled dorsiflexion and plantarflexion torques. The dorsiflexion ROM was taken from the upper limb of the representative hysteresis loop (dorsiflexion direction movement) at the 10Nm torque level (the ankle angle at s.DF and c.DF points in fig 3) and the plantarflexion ROM from the lower limb of the representative hysteresis loop (plantarflexion direction movement) at -3Nm (the angle at s.PF and c.PF in fig 3).

**Quasistatic stiffness.** To investigate the changes in the static component of passive mechanical properties, the stiffness of ankle plantar- and dorsiflexors were assessed as  $K = \Delta T / \Delta \theta$ , where K is the quasistatic stiffness (spring-like property characterized by the elastic stiffness of the spring), and  $\Delta T$  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.<sup>33</sup> 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 and the area under the lower limb represents the energy during the release, the difference in the area under the 2 curves—the area enclosed within the hysteresis loop—represents the energy loss within the joint muscles involved.<sup>33</sup> The energy loss was calculated for both dorsiflexion and plantarflexion ROMs (fig 3). Because different subjects had different ROMs, the energy loss was normalized to the corresponding maximum dorsiflexion or plantarflexion ROM (eg, the dorsiflexion range in fig 3).

**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-

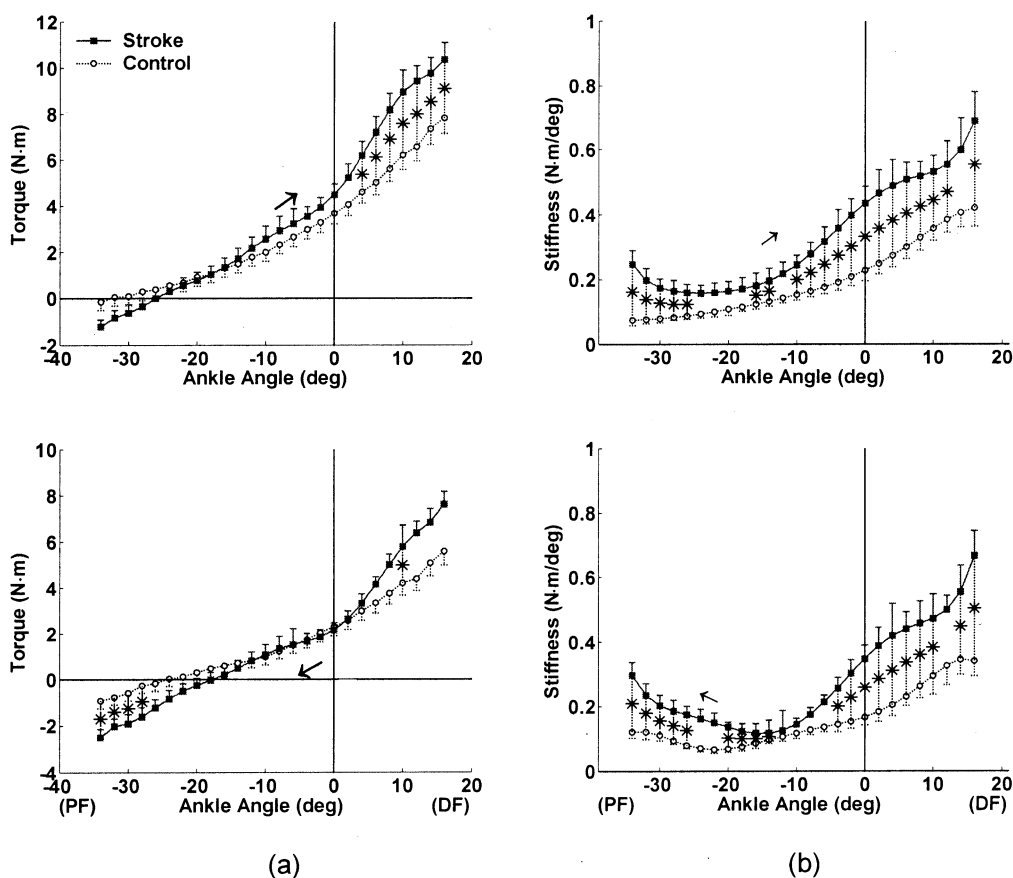


Fig 4. Continuous profiles of the passive resistance torque and quasistatic stiffness in the stroke and control groups. Torque-angle curves are averaged across subjects in each group at every 1° joint angle. Quasistatic stiffness at each ankle angle is calculated and also averaged across subjects in each group. Both (A) averaged torque and (B) stiffness curves are plotted for stroke and control groups during dorsiflexion direction and plantarflexion direction movement as indicated with arrows. The curves were polynomially fitted, and the symbols are shown at 2° intervals in the common ankle ROM. The vertical error bars (shown in 1 direction for simplicity) represent the standard error of mean. Significant differences are marked with asterisks.

tonia in stroke may affect the 4 parameters simultaneously and the parameters may be interrelated, multivariate analysis of variance (MANOVA) procedures<sup>b</sup> were used to compare the 4 parameters as a whole between the 2 groups in dorsiflexion and plantarflexion directions. Normality and equality of covariances were tested by Kolmogorov-Smirnov and Levene tests, respectively. Statistical significance was accepted if the *P* value of the Pillai trace was less than .05.<sup>34</sup> If the MANOVA test for dorsiflexion or plantarflexion direction showed significant difference between the 2 groups, univariate analysis of variance (ANOVA) procedures were used to evaluate which parameters of the passive properties were significantly changed by spastic hypertonia.

In addition to alterations determined by the univariate ANOVA tests following MANOVA, the extent of the alterations were shown in continuous profiles of the passive resistance torque and quasistatic stiffness during dorsiflexion and plantarflexion direction movement throughout ankle ROMs. Passive resistance torque and quasistatic stiffness were compared between 2 groups at each ankle joint angle at a 1° interval by using independent-samples *t* test after determination of normality by Kolmogorov-Smirnov test.<sup>34,b</sup> Correlations between the MAS and the 4 quantitative parameters of the passive properties were investigated. Because the quantitative parameters were continuous while the MAS was ordinal, both the Pearson product-moment correlation coefficient (*r*) and the Kendall rank-correlation coefficient ( $\tau$ ) were calculated.<sup>34</sup> A Pearson *r* greater than .353 or a Kendall  $\tau$  greater than .346 with a *P* value less than .01 was considered significant.

## RESULTS

### 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 (*P* 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 ( $9.51 \pm 4.79\text{Nm}$  vs  $6.21 \pm 3.64\text{Nm}$ , *P*=.016), higher quasistatic stiffness ( $.54 \pm .19\text{Nm/deg}$  vs  $.35 \pm .20\text{Nm/deg}$ , *P*=.001) at 10° of dorsiflexion, larger normalized dorsiflexion energy loss ( $.06 \pm .04\text{J/deg}$  vs  $.04 \pm .02\text{J/deg}$ , *P*=.037), and decreased dorsiflexion ROM at a controlled 10Nm torque ( $10.77^\circ \pm 8.69^\circ$  vs  $20.02^\circ \pm 11.67^\circ$ , *P*=.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, *P* pertaining to Pillai trace=.041). Subsequent univariate ANOVA tests for each parameter revealed significantly higher passive resistance torque ( $-1.90 \pm 1.84\text{Nm}$  vs  $-.58 \pm 1.92\text{Nm}$ , *P*=.038), higher quasistatic stiffness ( $.20 \pm .14\text{Nm/deg}$  vs  $.11 \pm .09\text{Nm/deg}$ , *P*=.001) at 30° of plantarflexion, and decreased plantarflexion ROM at a -3Nm torque ( $36.23^\circ \pm 7.63^\circ$  vs  $-46.01^\circ \pm 9.65^\circ$ , *P*=.002) in the stroke group than in the control group. The

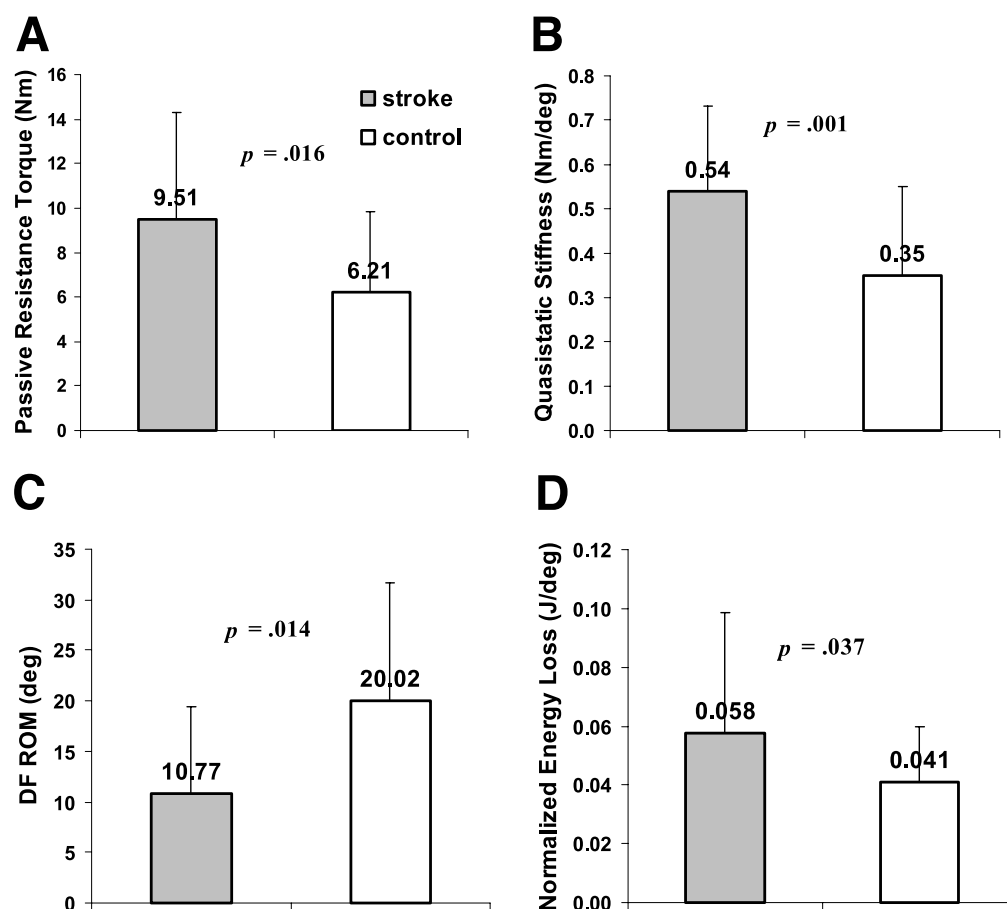


Fig 5. Comparisons of passive properties in dorsiflexion direction movement between groups. Mean and SDs of the parameters are shown. The stroke group shows higher (A) passive resistance torque, (B) quasistatic stiffness at 10° of dorsiflexion, (C) larger normalized dorsiflexion energy, loss and (D) decreased dorsiflexion ROM at 10Nm torque level. Statistical significances are shown with *P* values by univariate ANOVAs following a MANOVA testing 4 parameters together (*P* of Pillai trace=.016).

normalized plantarflexion energy loss was not statistically significant ( $.03 \pm .01$  J/deg in stroke vs  $.02 \pm .01$  J/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  $\tau$  between the MAS and the passive resistance torque at 10° of dorsiflexion ( $\tau = .255$ ), dorsiflexion ROM at 10Nm

( $\tau = -.323$ ), and quasistatic stiffness at 10° of dorsiflexion ( $\tau = .312$ ) 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-

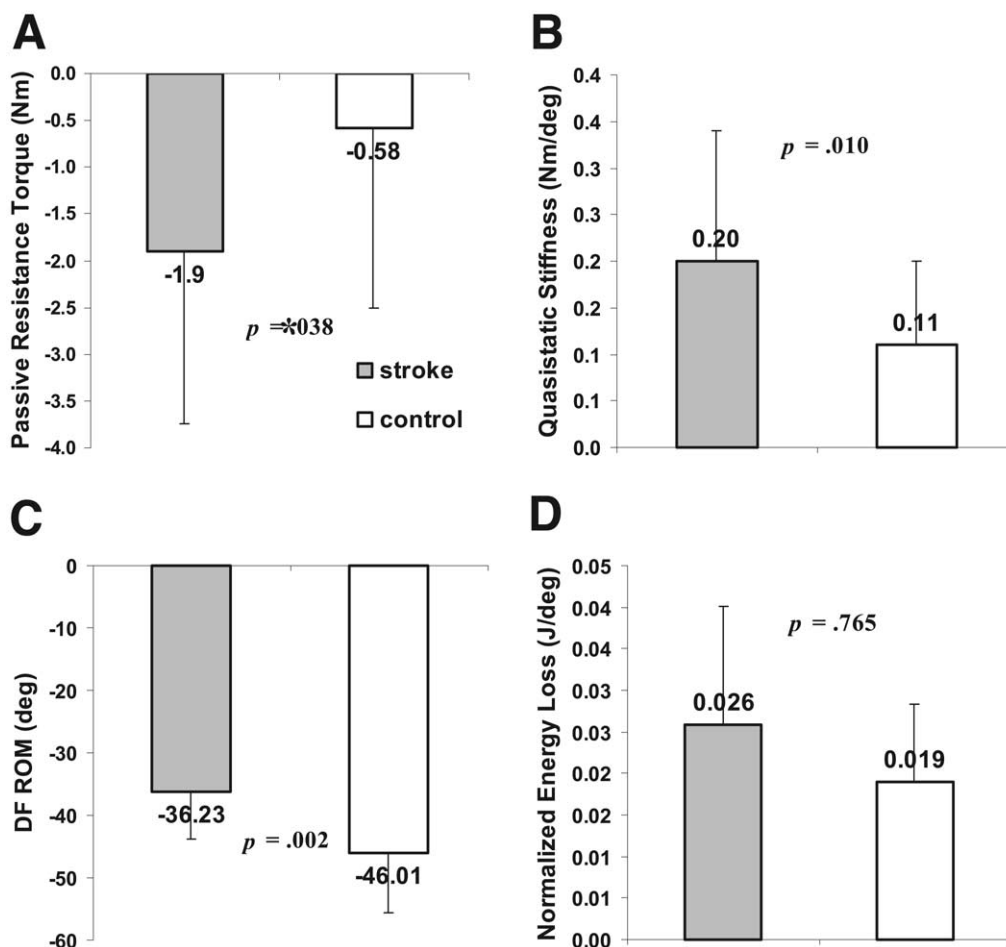


Fig 6. Comparisons of passive properties in plantarflexion direction movement between groups. The stroke group shows significantly higher (A) passive resistance torque, (B) quasistatic stiffness at 10° of plantarflexion, (C) decreased plantarflexion ROM at a -3Nm torque level. However, (D) normalized plantarflexion energy loss was slightly higher in the stroke group but was not statistically significant. Statistical significances are shown with P values by univariate ANOVAs protected by a MANOVA with 4 parameters together (P of Pillai trace=.041).

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 reflex<sup>5-10</sup> and/or nonreflex changes.<sup>11-13</sup> The former is sup-

ported by the exaggerated tendon jerks and the increased H-reflex response.<sup>5-10</sup> and the latter is associated with mechanical changes.<sup>11-13</sup> Several reports supported the predominant contribution of nonreflex component. Dietz et al<sup>11,12</sup> have suggested that changes in mechanical muscle properties were mainly responsible for muscle hypertonia. Lee et al<sup>13</sup> 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 al<sup>14,15</sup> 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 al<sup>3,16,17</sup> reported that spastic muscles in stroke patients had an increased nonreflex

**Table 1: Correlations Between the Biomechanic and Clinical Measures**

Measures	MAS	Resistance Torque at 10° of DF	DF ROM at 10Nm	Stiffness at 10° of DF	DF Energy Loss
MAS	1.00	.294*	-.380†	.297*	.230*
Resistance torque at 10° of DF	.255†	1.00	-.895†	.687†	.487†
DF ROM at 10Nm	-.323†	-.826†	1.00	-.721†	-.464†
Stiffness at 10° of DF	.312†	.517†	-.597†	1.00	.377†
DF energy loss	.139	.438†	-.407†	.223*	1.00

NOTE. The Kendall  $\tau$  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  $\tau > .346$  with  $P < .01$  was considered significant.

\* $P < .05$ .

† $P < .01$ .

Table 2: Comparison With Previous Studies

Parameters	Current Study		Singer et al <sup>27</sup>		Harlaar et al <sup>25</sup>	
	Subjects	Controls	Subjects	Controls	Subjects	Controls
Sample	24	32	13	18	8	8
Characteristics	Hemiplegia	Healthy	Brain injury	Healthy	Hemiplegia	Contralateral
PRT at 0° (Nm)	4.53±2.40	3.60±2.54	NA	NA	1.7 (.2–2.7)	1.1 (.6–1.4)
DF PROM (deg)	10.77±8.69	20.02±11.67	10.0±4.7	19.0±1.9	20.3 (13.6–31.8)	25.9 (18.4–28.0)
PF PROM (deg)	36.23±7.63	46.01±9.65	NA	NA	NA	NA
DF stiffness (Nm/deg)*	.54±.19	.35±.20	.53±.36	.44±.21	.44 (.31–.61)	.36 (.32–.47)
PF stiffness (Nm/deg)*	.29±.18	.13±.13	NA	NA	NA	NA
Energy loss (J/deg) <sup>†</sup>	.08±.05	.06±.03	NA	NA	NA	NA

NOTE. Values are expressed as mean ± SD or mean (range).

Abbreviations: NA, not available; PRT, passive resistance torque.

\*DF or PF stiffness is stiffness measured in dorsiflexion or plantarflexion passive movement. The methods used to measure the stiffness differed slightly among the articles.

<sup>†</sup>Normalized total energy loss.

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,<sup>14,28</sup> a conflicting result has been reported recently that the MAS measures muscle hypertonia rather than spasticity.<sup>35</sup> 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  $\tau$ ) 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  $\tau$  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,  $10.77^\circ \pm 8.69^\circ$  at a 10Nm resistance torque, was comparable to the results reported by Singer et al.<sup>27</sup> The PROM of the control group ( $n=18$ ;  $19.0^\circ \pm 1.9^\circ$ ) of Singer<sup>27</sup> was also similar to our result ( $n=32$ ;  $20.02^\circ \pm 11.67^\circ$ ). The quasistatic stiffness of ankle plantarflexor measured at  $10^\circ$  of dorsiflexion (.54Nm/deg for stroke, .35Nm/deg for control) was comparable to the 2 previous studies, which reported  $4.4\text{Nm}/10^{25}$  and  $.53\text{Nm}/\text{deg}^{27}$  in hemiplegic ankles and  $3.6\text{Nm}/10^{25}$  and  $.44\text{Nm}/\text{deg}^{27}$  in controls. Harlaar et al<sup>25</sup> reported lower stiffness in both hemiplegic and unaffected contralateral ankles than that reported by Singer<sup>27</sup> 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

- Dietz V. Spastic movement disorder. *Spinal Cord* 2000;38:389-93.
- Rymer WZ, Katz RT. Mechanisms of spastic hypertonia. *Phys Med Rehabil State Art Rev* 1994;8:441-54.
- Sinkjaer T, Magnussen I. Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. *Brain* 1994;117:355-63.
- Young RR. Spasticity: a review. *Neurology* 1994;44(11 Suppl 9):S12-20.
- Gottlieb GL, Agarwal GC, Penn R. Sinusoidal oscillation of the ankle as a means of evaluating the spastic patients. *J Neurol Neurosurg Psychiatry* 1978;41:32-9.
- Levin MF, Hui-Chan C. Are H and stretch reflexes in hemiparesis reproducible and correlated with spasticity? *J Neurol* 1993;240:63-71.
- Meinders M, Price R, Lehmann JF, Questad KA. The ankle reflex response in the normal and spastic ankle: effect of ankle position. *Arch Phys Med Rehabil* 1996;77:487-92.
- Pierrot-Deseilligny E, Mazieres L. Spinal mechanisms underlying spasticity. In: Delwaide PJ, Young RR, editors. *Clinical neurophysiology in spasticity*. Amsterdam: Elsevier Science; 1985. p 63-76.
- Rack PM, Ross HF, Thilmann AF. The ankle stretch reflexes in normal and spastic subjects. *Brain* 1984;107:637-54.
- Thilmann A, Fellows S, Garms E. The mechanism of spastic muscle hypertonus. Variation in reflex gain over the time course of spasticity. *Brain* 1991;114:233-44.
- Dietz V, Berger W. Normal and impaired regulation of muscle stiffness in gait: a new hypothesis about muscle hypertonia. *Exp Neurol* 1983;79:680-7.
- Dietz V, Trippel M, Berger W. Reflex activity and muscle tone during elbow movements in patients with spastic paresis. *Ann Neurol* 1991;30:767-79.

13. Lee WA, Boughton A, Rymer WZ. Absence of stretch reflex gain enhancement in voluntarily activated spastic muscle. *Exp Neurol* 1987;98:317-35.
14. O'Dwyer NJ, Ada L. Reflex hyperexcitability and muscle contracture in relation to spastic hypertonia. *Curr Opin Neurol* 1996; 9:451-5.
15. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain* 1996;119:1737-49.
16. Sinkjaer T, Andersen JB, Nielsen JF. Impaired stretch reflex and joint torque modulation during spastic gait in multiple sclerosis patients. *J Neurol* 1996;243:566-74.
17. Sinkjaer T, Toft E, Larsen K, Andreassen S, Hansen H. Non-reflex and reflex mediated ankle joint stiffness in multiple sclerosis patients with spasticity. *Muscle Nerve* 1993;16:69-76.
18. Young RR. Hypertonia: diagnosis and management. In: Lazar RB, editor. *Principles of neurologic rehabilitation*. McGraw-Hill: New York; 1998. p 329-36.
19. Dietz V, Quintern J, Berger W. Electrophysiological studies of gait in spasticity and rigidity. Evidence that altered mechanical properties of muscle contribute to hypertonia. *Brain* 1981;104: 431-49.
20. Berger W, Horstmann G, Dietz V. Tension development and muscle activation in the leg during gait in spastic hemiparesis: independence of muscle hypertonia and exaggerated stretch reflexes. *J Neurol Neurosurg Psychiatry* 1984;47:1029-33.
21. Ada L, Vattanasilp W, O'Dwyer NJ, Crosbie J. Does spasticity contribute to walking dysfunction after stroke? *J Neurol Neurosurg Psychiatry* 1998;64:628-35.
22. Lamontagne A, Malouin F, Richards C. Contribution of passive stiffness to ankle plantarflexor moment during gait after stroke. *Arch Phys Med Rehabil* 2000;81:351-8.
23. Siegler S, Moskowitz G, Freedman W. Passive and active components of the internal moment developed about the ankle joint during human ambulation. *J Biomech* 1984;17:647-52.
24. Tardieu C, Lespargot A, Tabary C, Bret M. Toe-walking in children with cerebral palsy: contributions of contracture and excessive contraction of triceps surae muscle. *Phys Ther* 1989;69: 656-62.
25. Harlaar J, Becher J, Snijders C, Lankhorst G. Passive stiffness characteristics of ankle plantar flexors in hemiplegia. *Clin Biomech (Bristol, Avon)* 2000;15:261-70.
26. Vattanasilp W, Ada L, Crosbie J. Contribution of thixotropy, spasticity, and contracture to ankle stiffness after stroke. *J Neurol Neurosurg Psychiatry* 2000;69:34-9.
27. Singer B, Dunne J, Singer K, Allison G. Evaluation of triceps surae muscle length and resistance to passive lengthening in patients with acquired brain injury. *Clin Biomech (Bristol, Avon)* 2002;17:151-61.
28. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther* 1987;67:206-7.
29. Bates B. *A guide to physical examination and history taking*. 5th ed. Philadelphia: Lippincott; 1991.
30. Meythaler JM, DeVivo MJ, Hadley M. Prospective study on the use of bolus intrathecal baclofen for spastic hypotonia due to acquired brain injury. *Arch Phys Med Rehabil* 1996;77:461-6.
31. Zhang LQ, Chung SG, Bai Z, et al. Intelligent stretching of ankle joints with contracture/spasticity. *IEEE Trans Neural Syst Rehabil Eng* 2002;10:149-57.
32. Vaughan CL, Davis BL, O'Connor JC. *Dynamics of human gait*. 2nd ed. Western Cape: Kiboho; 1999.
33. Burstein AH, Wright TM. *Fundamentals of orthopaedic biomechanics*. Baltimore: Williams & Wilkins; 1994.
34. Field AP. *Discovering statistics using SPSS for Windows: advanced techniques for the beginner*. London: Sage; 2000.
35. Bakheit AM, Maynard VA, Curnow J, Hudson N, Kodapala S. The relation between Ashworth scale scores and the excitability of the alpha motor neurones in patients with post-stroke muscle spasticity. *J Neurol Neurosurg Psychiatry* 2003;74:646-8.

#### Suppliers

- a. JR3 Inc, 22 Harter Ave, Woodland, CA 95776.
- b. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.