Journal of Orthopaedic & Sports Physical Therapy
2000;30(8):473–483

Relationships Between Plantar Flexor Muscle Stiffness, Strength, and Range of Motion in Subjects With Diabetes-Peripheral Neuropathy Compared to Age-Matched Controls

Gretchen B. Salsich, PhD, PT[1]
Marybeth Brown, PhD, PT[2]
Michael J. Mueller, PhD, PT[3]

RESEARCH REPORT

Study Design: Descriptive study to compare relationships between muscle performance measures in 2 subject groups.
Objectives: To determine the relationships between plantar flexor (PF) muscle stiffness, strength (concentric peak torque), and dorsiflexion (DF) range of motion (ROM) in subjects with diabetes who have peripheral neuropathy (n = 17, 10 men, 7 women; age = 58 ± 11 years) and age-matched controls (n = 17, 10 men, 7 women; age = 62 ± 6 years).
Background: The relationships between muscle stiffness, strength, and joint ROM have not been clearly established. Furthermore, the effect of neuromuscular pathology on these relationships is unknown.
Methods and Measures: PF stiffness and strength measurements were obtained with an isokinetic dynamometer. DF ROM was measured with a goniometer. A Pearson correlation matrix was constructed for each subject group using stiffness, strength, and ROM variables. The percent contribution of passive torque to total torque was computed at 2 joint angles.
Results: In subjects with diabetes and peripheral neuropathy (DM-PN) peak concentric PF torque was positively correlated with passive torque at 5° DF (r = 0.77), Stiffness #1 (r = 0.58), and Stiffness #2 (r = 0.50). The percentage of passive PF torque at 5° DF was greater in subjects with DM-PN, compared to control subjects (29.3 ± 9.4% versus 12.6 ± 5.9%).
Conclusions: The positive correlation between PF stiffness and strength, and the greater percentage of passive PF torque in subjects with DM-PN suggest that patients with decreased strength may use passive torque to maximize total torque. Therefore, treatment methods designed to decrease stiffness should be used cautiously. *J Orthop Sports Phys Ther 2000;30:473–483.*

Key Words: *active and passive tension, ankle joint, biomechanics*

[1] *Post-doctoral fellow, Department of Biokinesiology & Physical Therapy, University of Southern California, Los Angeles, Calif.*
[2] *Associate professor, Program in Physical Therapy, Washington University School of Medicine, St Louis, Mo.*
[3] *Associate professor, Program in Physical Therapy, Washington University School of Medicine, St Louis, Mo.*
This study was supported by a post-professional doctoral scholarship from the American Physical Therapy Association and the National Center for Medical Rehabilitation Research (NCMRR) 2T32HD07434-04A1 and 1RO1HD36802-01 (Mueller).
This study was approved by the Washington University School of Medicine Human Studies Committee.
Send correspondence to Gretchen B. Salsich, University of Southern California, Department of Biokinesiology & Physical Therapy, 1540 E. Alcazar St. CHP-155, Los Angeles, CA 90033.
E-mail: salsich@hsc.usc.edu

Many factors may contribute to the production and control of movement, including muscle strength, passive muscle stiffness, and joint motion. Generally, muscle strength is an indication of the active tension producing capability of muscle, while muscle stiffness, defined as the change in passive tension per unit change in length,[5] is an indication of a muscle's passive resistance to elongation. Because both active and passive tension generation depend on the length of the muscle,[17,20,45] joint position may influence both muscle strength and passive muscle stiffness.

By definition, active tension is generated when the muscle is "activated" neurologically (ie, during a voluntary or reflexive contraction) and is attributed to structures within the contractile element of the muscle.[11,17,45] Passive tension develops when a passive (not neurologically stimulated) muscle is lengthened and is be-

TABLE 1. Subject characteristics.*

Group	Age (yrs)	Mass (kg)	BMI† (kg/m²)	Height (cm)	Duration of DM (yrs)	History of foot ulcers (number of subjects)
DM-PN‡						
$n = 17$	58.5 ± 11.7	89.7 ± 23.1	30.3 ± 6.6	171.4 ± 11.5	18.0 ± 9.9	13
Control						
$n = 17$	62.7 ± 6.2	83.7 ± 15.8	28.0 ± 3.5	172.5 ± 11.4	N/A	0

* 10 men, 7 women in each group. There were no significant differences in any variable. Numbers represent mean ± standard deviation.
† Body Mass Index = mass/(height)².
‡ Diabetes with peripheral neuropathy.

lieved to originate from the series elastic and parallel elastic elements of muscle (ie, the tendon, cross-bridge attachments, structural proteins within the myofibril, and connective tissue around the muscle fibers and fascicles).[11,45] Both passive and active tension contribute to the total tension produced by a muscle, a relationship often depicted by traditional muscle length-tension curves.[24]

All 3 variables (muscle strength, passive stiffness, and joint motion) may be altered under various conditions. For example, muscle strength, as indicated by peak concentric torque, has been shown to decrease with age,[12,13] immobilization,[34] and disease,[4,28] and joint motion has been shown to be affected by similar conditions.[3,8,25,27,39] Passive muscle stiffness, while not studied as extensively, has been shown to increase with age in rats,[1] with immobilization in cats,[38] mice,[41] and humans,[8] and to increase after neurological insult (ie, stroke).[36] However, there also is evidence that passive stiffness decreases with age in humans[14] and that it may be unaffected by neuromuscular pathology.[32]

While muscle strength, stiffness, and joint motion may be altered under various conditions, the relationships between these variables have not been clearly established. Furthermore, the effect of disease or disuse on these relationships is unknown. Understanding the relationships between strength, stiffness, and range of motion at the ankle joint would provide insight into mechanisms that cause specific impairments, as well as insight into the management of patients with pathological gait patterns. Studying subjects with known clinical deficits (decreased strength and range of motion) and nonimpaired control subjects would provide a wide range of strength, stiffness, and motion values, making relationships between these variables clearer. Patients with diabetes mellitus who have peripheral neuropathy (DM-PN) have decreased muscle strength (peak torque)[2,4,28] compared to control subjects, decreased ankle joint motion,[3,27,28] postural instability,[35] altered gait characteristics,[28] and in our experience, complain of "stiffness." In addition, research has provided evidence showing the ultrastructure of collagen, a component of the elastic elements of muscle (stiffness), is altered with long-term DM. Specifically, there is an increase in cross-linking of collagen molecules.[40]

In light of the clinical deficits of decreased plantar flexor muscle strength and ankle joint motion in subjects with DM-PN, and the physiologic changes in collagen associated with the disease that may contribute to stiffness, it is possible that the relationships between muscle stiffness, strength, and joint motion are different in subjects with DM-PN than in control subjects. The purpose of this study was to examine the relationships between passive plantar flexor muscle stiffness, strength (concentric peak torque), and ankle joint motion (dorsiflexion range of motion) in subjects with DM-PN and control subjects. Specifically, we hypothesized that, (1) plantar flexor stiffness and peak concentric torque would be negatively correlated in subjects with DM-PN and positively correlated in control subjects, (2) plantar flexor stiffness and dorsiflexion (DF) range of motion would be negatively correlated in both groups, and (3) subjects with DM-PN would have greater passive torque contribution to total (active plus passive) plantar flexor muscle torque than control subjects.

The rationales for our hypotheses are based on evidence of decreased strength[2,4,28] and our observed complaints of increased stiffness in subjects with DM-PN, and evidence of a positive correlation between muscle size and passive stiffness in nonimpaired subjects[9] (hypothesis #1), evidence of decreased muscle extensibility and increased passive tension in immobilized rat hindlimb muscles[38] (hypothesis #2), and evidence of greater passive torque contribution to total torque in rat hindlimb muscles that have undergone disuse[7] (hypothesis #3).

METHODS

Subjects

Two groups of subjects were examined: 17 subjects with DM-PN, and 17 age-matched control subjects. Subject groups were matched (mean values) on age, sex, height, weight, and body mass index. Complete subject characteristics are displayed in Table 1. Recruitment of subjects for the DM-PN group was through the Diabetic Foot Centers at Barnes/Jewish Hospital, Christian Hospital Northeast, and St John's Mercy Medical Center, St Louis, Mo. Subjects in the control group were recruited from family members of the DM-PN group, from employees of the medical

center, and from the community. Inclusionary criteria for both groups consisted of an ability to lie supine, ability to walk independently without an assistive device, and the presence (DM-PN) or absence (controls) of peripheral neuropathy. Peripheral neuropathy was characterized by the loss of protective sensation on the plantar surface of the foot, measured by using Semmes-Weinstein monofilaments.[6] Criteria for exclusion consisted of the presence of a severe orthopedic or neurologic problem, which noticeably compromised their gait.

Procedures

Each subject was tested in a single session. The procedures were explained thoroughly; all subjects read and signed an informed consent statement, approved by the Washington University School of Medicine Human Studies Committee, prior to testing. A brief medical history was obtained and demographic data recorded, including date of birth, sex, height, weight, body mass index, general health status, and activity level. Subjects with DM-PN were asked specific questions regarding the history, duration, and control of their diabetes.

Sensation Testing

Sensory testing, using Semmes-Weinstein monofilaments, was performed on each subject to assist in the confirmation of peripheral neuropathy in subjects with DM. This procedure involved using the 5.07 monofilament and the testing of several sites on the plantar surface of both feet as described by Mueller et al.[27] Subjects who were unable to detect the 5.07 monofilament lacked protective sensation, while subjects who could detect the monofilament were said to have intact sensation.[6]

Range of Motion Measurements

For each subject, dorsiflexion (DF) goniometric measurements were taken on one lower extremity, with the subject positioned prone, in full knee extension as previously described.[32] No attempt was made to randomize the selection of right versus left side, but there was no reason to believe there would be an inherent "side" difference in the measures used in this study. In order to determine intrarater reliability, the examiner repeated the DF measurement once during the testing session. Using the 2 obtained DF measurements, the intraclass correlation coefficient (ICC [3,1])[21] was 0.95.

An additional variable used to describe DF range of motion was plantar flexor muscle excursion. The definition and measurement method of this variable are described in the next section.

Stiffness Measurements

The Kinetic Communicator Exercise system, or Kin-Com (Chattecx Corporation, Hixon, Tex), with attached ankle apparatus, was used to measure passive plantar flexor torque and to compute stiffness. Although tissues other than the plantar flexor muscles may contribute to passive ankle torque, there is evidence suggesting that the major contributor to passive ankle torque throughout dorsiflexion is the plantar flexor muscle group.[15] Also, several researchers have studied passive ankle torque in humans using methods similar to ours, and have made this assumption.[13,14,36]

In order to verify that the stiffness measurements were truly passive (no obvious active muscle contractions elicited), electromyography (EMG) equipment was used throughout the procedure (CGS-67 Multichannel Electromyographic System, Therapeutics Unlimited, Iowa City, Iowa). The anterior and posterior surfaces of the shank (same lower extremity used in the DF procedure) were cleaned with alcohol to reduce skin impedance. Surface electrodes, with attached preamplifiers, were applied over the belly of the anterior tibialis, the gastrocnemius, and the soleus (distal to the gastrocnemius muscle belly and lateral to the Achilles tendon). The EMG amplifier gains were set to either 2000 or 5000, based on the EMG signal output viewed on an analog oscilloscope (ie, whichever setting that displayed a smooth resting baseline signal). The raw EMG signal was collected and high-pass filtered at 40 Hz, and given the upper frequency limit of the amplifier (4000 Hz). The frequency response of our EMG system was 40 Hz to 4000 Hz.

The Kin-Com was set in the isokinetic mode for ankle plantar flexion, and the gravity correction procedure was performed on the empty ankle apparatus. The limb was not included in the gravity correction because the weight of the foot was assumed to be negligible (approximately 1.5% body weight).[43] Each subject was positioned supine on the Kin-Com bench with their knee supported by a rolled towel and placed in 10° of flexion. Ten degrees of knee flexion was chosen because it approximates the average knee angle during the terminal stance phase of gait.[42,44] The foot was then placed in the ankle apparatus and positioned by visual approximation such that the point, midway between the lateral and medial malleolus in the sagittal plane, was aligned with the axis of rotation of the Kin-Com. The foot, ankle, and thigh were secured with straps. To check for a discrepancy between the subject's ankle angle and the Kin-Com angle, the height of the dynamometer was adjusted until the subject's shank was parallel to the floor, and the Kin-Com foot plate was placed in a vertical position (confirmed with a level), which produced a reading of 0. The subject's ankle angle was measured with a goniometer to verify a neutral

dorsiflexion position (defined as a goniometric reading of 0 ± 1°).

Once proper joint alignment was achieved, the subject's ankle was passively moved by the examiner into dorsiflexion and plantar flexion approximately 10 times to allow the calf muscles to relax. Four criteria were used to determine the maximal DF angle. The presence of a firm end-feel by the examiner (the point at which firm resistance to motion was detected) was the primary criterion; however, subject tolerance, heel movement in the apparatus, and the point at which EMG activity occurred were also considered. Intrarater reliability of maximal DF angle measurements was established in a group of 10 subjects of mixed age, sex, activity level, and disease status (DM-PN versus control). The resulting ICC (3,1) value was 0.98.

Once maximal DF angle was established, the subject's ankle was moved into plantar flexion (PF) as far as possible within the constraints of the apparatus, and this PF angle became the starting position for the passive test. No specific maximal PF angle was used, as all subjects were able to reach an angle sufficient to slacken the plantar flexor muscles. Next, the subject was asked to relax his or her leg while baseline EMG was observed on the oscilloscope. The Kin-Com ankle apparatus moved the ankle joint from the starting position of maximal PF into maximal DF (average total ankle range: 48.2 ± 9.8° for DM-PN group, 58.1 ± 5.9° for the Control group), while the subject's muscles remained passive. Three trials of passive torque and ankle angular displacement data were collected in both directions, lengthening and shortening of the plantar flexor muscles. The trials were collected at a speed of 60 per second to approximate the average ankle joint velocity during gait.[28,43] Also, pilot work indicated that passive torque curves at 60°/s did not appear different from curves generated at 5°/s. There was no evidence of a velocity effect on passive torque. Lamontagne et al[23] noted similar findings.

During the passive test, EMG signals were viewed on the oscilloscope to check for deviations from the subject's baseline signals, visually observed on the oscilloscope prior to ankle movement. In addition, the passive torque curves were viewed throughout the procedure to check the consistency of their shape (overlay). A coefficient of variation (CV) was calculated for each subject, using the 3 recorded passive torque curves in each direction. The CV represents the root mean square width of the standard deviation "band" of the ensemble averaged curve,[42] and it is expressed as a percentage of the mean curve. A coefficient close to zero indicates low variability, while a value close to 1.0 (100%) indicates high variability (Mean CV, *lengthening*: DM-PN = 0.09 ± 0.05, Control = 0.07 ± 0.03; Mean CV, *shortening*: DM-PN = 0.16 ± 0.12, Control = 0.13 ± 0.07). The consistency of the passive torque curves indicated that the subjects' muscles remained passive throughout the trials.

Concentric Peak Torque (Strength)

The concentric test procedure was identical to the passive procedure except that instead of remaining passive, the subject started in maximal dorsiflexion and pushed as hard as he or she could into plantar flexion, as cued by the investigator. The concentric test was performed at 60°/s to be consistent with the passive test; however, once each concentric contraction was completed, the Kin-Com passively moved the subject's ankle back to the maximal DF angle at 5°/s. Each subject was given several warm-up repetitions to accommodate to the machine, and 3 trials were collected once the torque versus angle curves appeared to overlay each other consistently.

Data Reduction

For each subject, the 3 PF torque trials during passive motion into PF and DF, and the three concentric PF torque trials were averaged. The resulting torque versus angle curves were plotted. From the averaged lengthening passive PF torque curve, the following variables were obtained: maximal DF angle, initial angle (defined as the greatest DF angle at which passive torque was 0 N m), plantar flexor muscle excursion (the difference between maximal DF angle and initial angle), passive torque at 5° DF, and stiffness. While 10 of dorsiflexion has been reported to be necessary for normal gait,[29] and, therefore, would have been a more "critical" angle at which to measure passive torque, 5° DF was used because it was found to be the mean maximal DF angle (goniometric measurement) in a group of subjects with DM-PN.[27] Stiffness (slope of passive torque curve) was calculated separately for the first and second halves of the passive torque curve (called stiffness #1 and stiffness #2 hereafter), using the initial angle and maximal DF angle to determine the total range (Figure 1). Calculating 2 slopes provided a better means of characterizing the passive torque curve, due to its curvilinear nature. This method was similar to that used by Chleboun et al[9] to characterize passive elbow stiffness.

For determination of the percent passive PF torque contribution, 2 variables from the averaged shortening passive PF torque curve were obtained: passive torque at 5° DF, and passive torque at peak concentric PF torque angle. The shortening passive PF torque curve was used to acquire these variables because it was more consistent with the concentric torque curve, in terms of direction of ankle motion (ie, from DF to PF). From the averaged concentric PF torque curve, the values of peak concentric torque and concentric torque at 5° DF were ob-

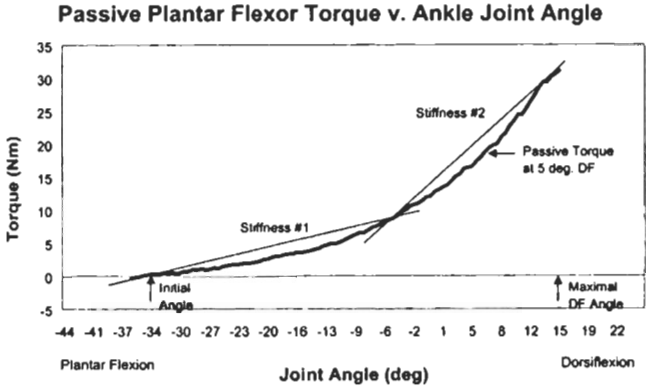

FIGURE 1. Representative of passive plantar flexor torque versus ankle joint angle curve. Stiffness variables are depicted using hypothetical data.

tained. Intrarater reliability of initial angle, stiffness, and concentric torque variables was determined from the 3 passive and 3 concentric PF torque trials. ICCs (3,1) were 0.97 (initial angle), 0.98 (passive torque at 5° dorsiflexion, lengthening curve), 0.87 (stiffness #1), 0.96 (stiffness #2), 0.97 (passive torque at 5° dorsiflexion, shortening curve), 0.95 (concentric torque at 5° DF), 0.98 (passive torque at peak concentric torque angle, shortening curve), and 0.97 (peak concentric torque).

The contribution of passive PF torque to total PF torque was determined at 2 joint angles, 5° DF and the angle at which peak concentric PF torque occurred. Percent contributions were calculated by dividing the amount of passive torque (shortening curve) by the amount of concentric (total) torque at each of the 2 joint angles.

Data Analysis

To determine the relationships between plantar flexor muscle stiffness, strength, and DF range of motion in both groups, 2 Pearson correlation matrices were constructed using the following variables: maximal DF angle (goniometer measurement), plantar flexor muscle excursion, passive torque at 5° DF (lengthening curve), passive stiffness (slopes of first and second halves of passive torque curve), concentric peak torque, and body mass. Plantar flexor muscle excursion was included as a range of motion variable because it is an indication of the range of motion through which passive plantar flexor torque contributes to total torque, and, therefore, provides information that maximal DF angle does not. Body mass was included in the matrices because it was assumed to be related to muscle mass, and, therefore, would provide information about the relationships between clinical measures of muscle performance (strength and stiffness) and muscle size.

To determine the differences in passive torque contribution to total torque between the 2 groups, a Student's *t* test was performed comparing the following mean values at 2 joint angles, 5° DF and peak concentric torque angle: passive PF torque (shortening curve), concentric PF torque, and percent passive PF torque. The α level was set at 0.05 for all *a priori* hypotheses (percent passive PF torque at both joint angles), while Bonferroni correction was used for remaining comparisons to protect against a Type I error.[30]

RESULTS

The results of the Pearson correlation analyses for both subject groups are displayed in Table 2.

Subjects with Diabetes—Peripheral Neuropathy

Stiffness and peak concentric torque (strength) Contrary to our hypothesis, we observed a *positive* relationship between muscle stiffness and muscle strength in subjects with DM-PN. All passive torque variables (passive torque at 5° DF, stiffness #1, stiffness #2) were positively correlated with concentric peak torque (Table 2 and Figure 2). In addition, body mass was correlated with stiffness (passive torque at 5° DF, stiffness #1, stiffness #2) and strength (concentric PF peak torque) variables in subjects with DM-PN (Table 2).

Stiffness and range of motion Subjects with DM-PN did not demonstrate a relationship between passive plantar flexor stiffness and DF range of motion, as there were no significant correlations between stiffness and range of motion variables (Table 2).

Control Group

Stiffness and peak concentric torque (strength) In control subjects, we found no relationship between passive plantar flexor stiffness and plantar flexor strength. Body mass was correlated only with passive torque at 5° DF (Table 2).

Stiffness and range of motion As in subjects with DM-PN, there was no relationship between plantar flexor stiffness and DF range of motion in control subjects (Table 2).

Passive Torque Contribution to Total Plantar Flexor Muscle Torque

The results of the Student's *t* test comparing the mean passive and concentric PF torque values between groups are displayed in Table 3 and depicted in Figure 3. At 5° DF, subjects with DM-PN demonstrated a greater passive PF torque contribution to total PF torque than control subjects despite similar passive torque values between groups (Table 3). The amount of concentric torque at 5° DF was notably less in subjects with DM-PN (Table 3). A similar relationship (less concentric torque in subjects with DM-PN; passive torque simi-

TABLE 2. Association between ankle stiffness, strength, and range of motion.*

			Range of motion		Stiffness			Strength
		Mass	Max DF angle†	Plantar flexor excursion‡	Passive torque at 5° DF	Stiff #1§	Stiff #2‖	Conc. peak torque
	Mass	—	Ns	Ns	0.58 (0.02)	Ns	Ns	Ns
Range of motion	Max DF angle†	Ns	—	Ns	Ns	Ns	Ns	Ns
	Plantar flexor excursion‡	Ns	Ns	—	Ns	Ns	Ns	Ns
Stiffness	Passive torque at 5° DF (n = 16)	0.80 (<0.001)	Ns	Ns	—	0.73 (0.001)	0.84 (<0.001)	Ns
	Stiff #1§	0.56 (0.02)	Ns	Ns	0.7 (0.003)	—	0.67 (0.003)	Ns
	Stiff #2‖	0.58 (0.02)	Ns	Ns	0.85 (<0.001)	0.56 (0.02)	—	Ns
Strength	Conc. peak torque	0.61 (0.01)	Ns	Ns	0.77 (0.001)	0.58 (0.02)	0.5 (0.04)	—

*Cell values are Pearson r coefficients. DM-PN group is displayed below diagonal. Control group is displayed above diagonal. Values in parentheses are significant levels (P values). n = 17, except where noted. Ns indicates not significant.
† Maximal DF angle measured with a goniometer.
‡ Maximal DF angle minus initial angle (angle at onset of passive PF torque).
§ Slope of first half of passive torque v. angle curve.
‖ Slope of second half of passive torque v. angle curve.

lar to control subjects) was observed at the peak concentric PF torque angle (Table 3).

DISCUSSION

Subjects with Diabetes—Peripheral Neuropathy

Contrary to our hypothesis of a negative correlation between passive muscle stiffness and strength in subjects with DM-PN, our results showed a *positive* correlation between all passive PF torque variables and concentric PF peak torque. While this finding was surprising given subjective complaints of stiffness and documented collagen changes in patients with DM-PN, it may be explained by the fact that intramuscular structures that contribute to active tension generation (strength) also contribute to passive tension generation (stiffness). Magid et al[26] and Horowits et al[19] have attributed passive muscle tension to the large structural protein, titin, while Alnaqeeb et al[1] found a positive relationship between the amount of collagen and passive stiffness. Still others suggest that weak binding between actin and myosin contributes to passive stiffness.[18,22] If the amount of each of these physical structures, titin, collagen, and actin-myosin binding sites increases with muscle size, a positive correlation between strength and stiffness makes sense, as muscle strength (torque output) is also directly proportional to muscle size (cross-sectional area).[24,43] In fact, Chleboun et al[9] found a similar relationship in their study investigating factors contributing to elbow stiffness in young adults. The authors reported a strong positive correlation between elbow flexor muscle volume, an indication of muscle size, and passive elbow stiffness (r = 0.92).[9] Our finding of a positive correlation between body mass and passive stiffness in subjects with DM-PN further supports the idea that muscle size may influence passive muscle stiffness.

We predicted a negative relationship between passive PF muscle stiffness and DF range of motion in subjects with DM-PN; however, there were no significant correlations between any of the stiffness and DF range of motion variables. One reason for this finding may be that factors other than passive stiffness determine range of motion in this population. We chose to study a group of subjects (DM-PN) with limited DF range of motion, assuming these subjects would fall towards the greater end of the passive stiffness spectrum, as a result of their disease. With the evidence of decreased DF range of motion in this population,[3,27,28] a negative correlation between these variables was logical to expect. However, a concurrent study has shown that there is no difference in passive PF stiffness (stiffness #1, stiffness #2, passive torque at 5° DF) between subjects with DM-PN and controls, despite a notable difference in DF range of motion.[32] It is possible, therefore, that in this population, factors such as muscle strength (ie, anterior tibialis) and sensation are more related to DF range of motion than stiffness.

Nonimpaired Subjects

Contrary to our hypotheses of a positive relationship between passive stiffness and strength, and a negative relationship between stiffness and range of motion in control subjects, we found no relation-

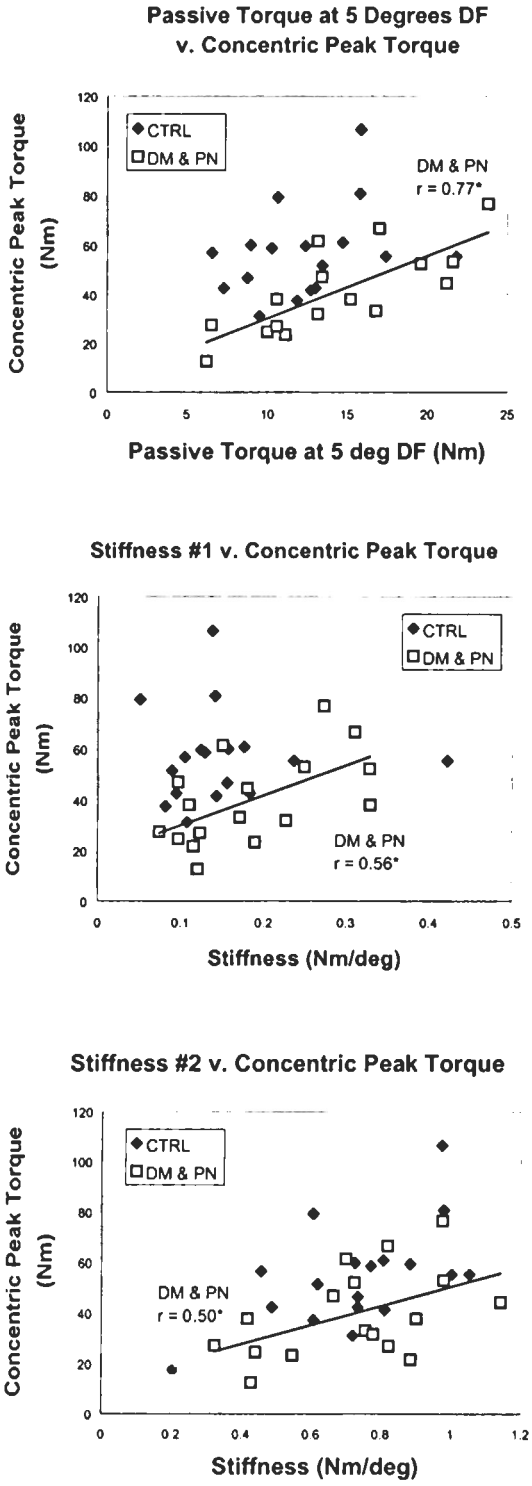

FIGURE 2. Stiffness and Strength: Correlations between passive torque variables and concentric peak torque. (A) Passive torque at 5° dorsiflexion (DF) versus Concentric peak torque, (B) Stiffness #1 (slope of 1st half of passive torque curve) v. Concentric peak torque, (C) Stiffness #2 (slope of 2nd half of passive torque curve) versus Concentric peak torque. *$P < .05$.

ships between stiffness, strength, and range of motion within the control group. We believe one factor contributing to this result was a reduction in the range of values for stiffness #1. As shown in Figure 3B, the range of values for stiffness #1 in the control group (excluding one outlier) was 0.19 N m/deg, while the range for stiffness #1 in the DM-PN group was 0.26 N m/deg. While the addition of more subjects may have resulted in significant correlations, a post-hoc power analysis revealed that nearly 800 subjects would be required to achieve significant correlations at a power level of 0.80.[10] This finding suggests that in nonimpaired individuals in true population correlations between stiffness, strength, and range of motion, as measured in our study, are unlikely.

The lack of correlation between stiffness, strength, and range of motion variables in the control group, while contrary to our hypotheses, provides important information regarding muscle performance. The absence of relationships between these variables suggests that the maximal torque producing capability of a healthy muscle may be more related to factors other than passive characteristics. For example, the contractile element of muscle employs neural activation parameters,[45] calcium dynamics,[24] and cross-bridge coupling,[20] all of which are likely to be functioning optimally in a healthy muscle. The passive elastic structures, therefore, may play a less important role in total tension development.

Passive Torque Contribution to Total Plantar Flexor Muscle Torque

As hypothesized, subjects with DM-PN demonstrated a greater passive PF torque contribution to total torque at 5° DF than control subjects; however, the actual amount of passive PF torque at 5° DF was no different in the 2 groups. The greater passive PF torque percentage, therefore, was due to a significantly smaller amount of concentric PF torque in subjects with DM-PN. It appears that passive muscle properties were not significantly affected, despite a loss of active contractile tension generation. This finding is similar to those of Brown et al[7] who studied the effects of age and reduced muscle use on passive and active tension in rats. The authors reported a decrease in peak active tension with age and hindlimb suspension, a model of simulated bed rest. However, passive tension remained unchanged. In addition, hindlimb suspension in older rats resulted in a profound loss of muscle excursion (length change from rest length).[7]

Our finding of greater percent passive torque contribution suggests that, in the presence of decreased contractile capability (strength), passive muscle structures may play an important role in total tension production. For example, several authors[33,37] have suggested that passive PF torque contributes to the peak plantar flexor moment during gait in subjects with neuromuscular pathology. In addition, there is evidence that in subjects with DM-PN, a significant portion of variance in peak ankle moment and walking speed is due to passive plantar flexor stiffness.[31]

TABLE 3. Passive torque contribution to total (concentric) plantar flexor torque in subjects with diabetes, who have peripheral neuropathy and controls.*

	At 5° dorsiflexion (DF)			At peak concentric torque (CT) angle		
Group	Percent passive torque (%)‡	Passive torque (N m)	CT (N m)	Percent passive torque (%)§	Passive torque (N m)	CT (N m)
DM-PN‖						
n = 17†	25.3 ± 9.4	8.2 ± 3.1	36.1 ± 16.2	17.6 ± 18.0	5.9 ± 4.9	40.0 ± 17.6
Control						
n = 17	12.6 ± 5.9	6.4 ± 2.5	53.6 ± 18.8	9.2 ± 3.7	5.0 ± 2.0	56.8 ± 18.4
t ratio	−4.6	−1.9	2.9	−1.9	−0.7	2.7
df	31	31	31	32	32	32
P	<.001	.06	.008	.07	.49	.01

* Numbers represent mean ± standard deviation.
† *n* = 16 in comparisons at 5° DF.
‡ Percent passive torque = (passive torque at 5° DF/CT at 5° DF) × 100.
§ Percent passive torque = (passive torque at peak CT angle/peak CT) × 100.
‖ Diabetes and Peripheral Neuropathy.

Implications

The results of our study provide information about relationships between clinical measures of muscle performance (muscle stiffness, strength, and joint motion). The positive relationship between PF stiffness and concentric peak torque in subjects with DM-PN suggests that patients with decreased active muscle function may use passive torque to generate total torque output. In other words, a greater part of their "strength" may come from passive tension production than would be the case for nonimpaired individuals.

Because there was no correlation between stiffness and range of motion, clinicians should be cautious when using the terms, "stiffness" and "decreased range of motion" interchangeably. Limited joint motion can occur without an increase in passive stiffness, defined as the change in torque per unit change in joint angle;[32] however, clinicians often use the term "stiffness" to describe limited joint motion. What they might actually be perceiving is a fully lengthened muscle exerting tension at its end-range. Furthermore, patients often complain of stiffness, but they might be experiencing decreased stretch tolerance[16] or decreased range of motion.[32]

The finding of greater passive PF torque contribution in subjects with DM-PN suggests that in the presence of diminished strength, patients may use passive torque to assist with support and propulsion during a functional activity such as gait. Therefore, treatment methods designed to decrease "stiffness" (ie, stretching, surgical techniques to increase length, etc) should be used cautiously. While increased contributions from passive torque might be useful, clinicians should be aware of potential negative consequences. Patients with greater passive contributions to total torque, who also have decreased range of motion, may be more susceptible to tissue injuries, as they appear to function closer to the end-ranges of muscle length without as much active muscle "reserve" to protect the passive structures.

Limitations

While the results of our study have useful clinical implications, there are several limitations. Pearson correlation coefficients do not provide predictive or causal information about relationships. The subjects with DM-PN had less strength (Table 3) and range of motion[32] than control subjects; however, they demonstrated similar stiffness values.[32] This may be partially due to the multiple clinical and physical limitations of subjects with DM-PN. It is likely that they have muscle atrophy, a documented complication of DM-PN,[2] and increased collagen cross-linking associated with long-term hyperglycemia.[40] If stiffness is positively related to muscle cross-sectional area,[9] and positively related to collagen content,[1] having muscle atrophy and excessive collagen cross-linking may have resulted in no net change in passive muscle stiffness in subjects with DM-PN, making them appear similar to control subjects.

Because the strength measure involved a concentric (shortening) muscle contraction, we used the shortening passive PF torque curve in the percentage calculation to assess the PF torque versus angle relationship (Figure 3). While the use of the lengthening passive torque curve would have been possible with an eccentric (lengthening) strength curve, we were unable to obtain reliable and valid eccentric plantar flexor strength measurements, due to the configuration of the ankle testing apparatus. While it is possible that there was a hysteresis effect in the plantar flexor muscles, such that the shortening passive PF torque curve was not identical to the lengthening passive torque curve, we believe the effect would have been similar in both groups, and that our results would have been the same regardless of which passive curve was used. Previous work indicat-

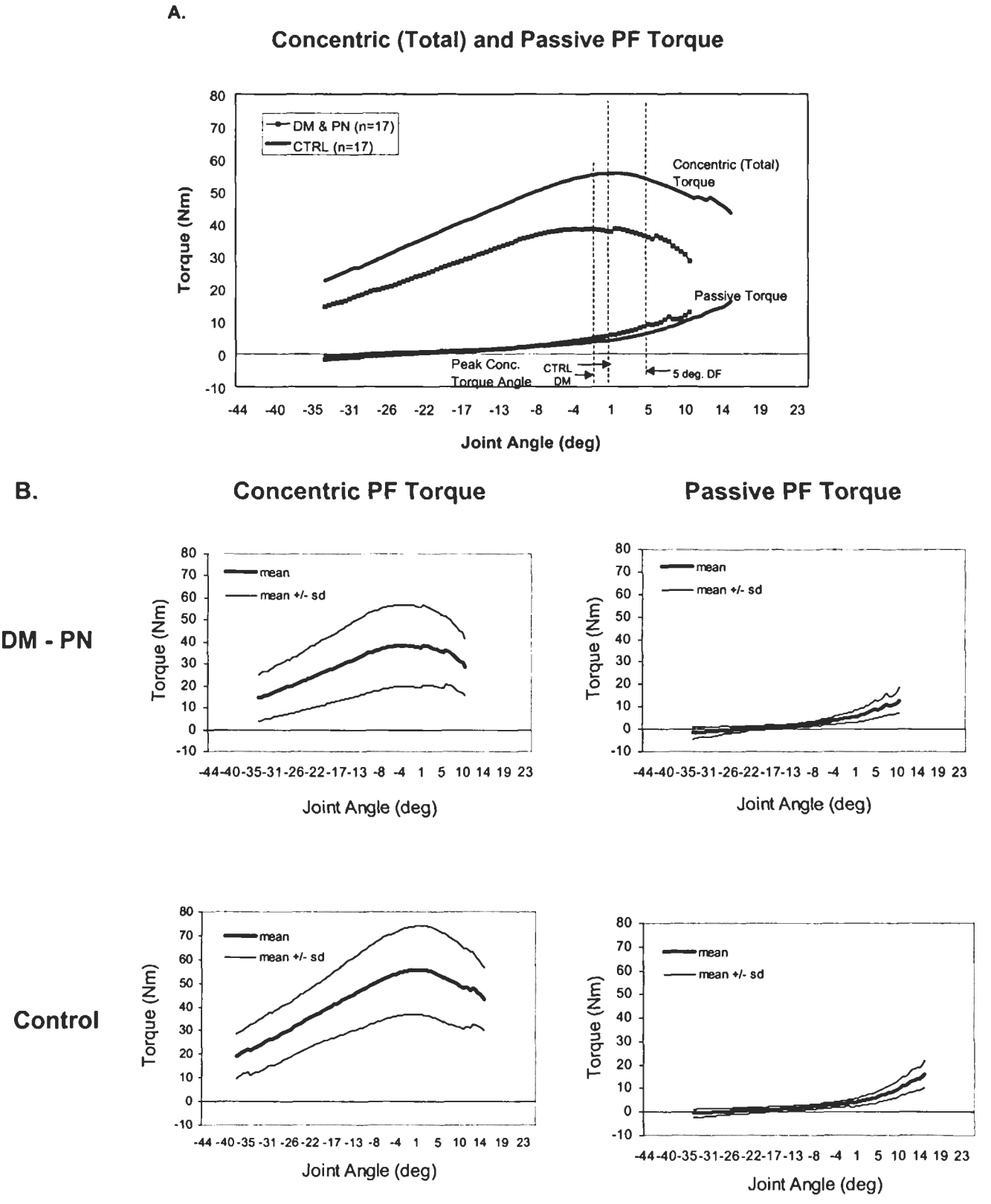

FIGURE 3. (A) Mean Concentric and Passive (unloading) torque curves for both groups. Curves represent ensemble averages (n = 17 for each curve). Note lower peak concentric torque value in subjects with DM-PN despite similar passive torque values. (B) Concentric and Passive torque curves for both groups, separated to illustrate mean (n = 17) ± 1 standard deviation.

ed that the amount of passive PF torque at 5° DF (lengthening curve) was not statistically different in subjects with DM-PN compared to controls,[32] a result that is consistent with the findings of our study.

Our method of determining the contribution of passive torque to total torque (ie, computing a ratio from a passive trial and an active trial) may have overestimated the actual passive component that would be present during an active contraction, as the parallel elastic component would effectively be shortened. However, we do not believe this occurrence would have made a great impact on our data because there is evidence that myofibrillar structures such as titin, rather than parallel elastic structures, bear the majority of passive muscle tension within a physiological range of movement.[26] Because titin also has been found to keep myosin filaments centered within the sarcomere during active contraction,[19] effectively transmitting tension, we believe that shortening of the parallel elastic component may not have caused a reduction in passive tension significant enough to impact our calculations.

CONCLUSION

Passive plantar flexor muscle stiffness and strength were positively correlated in subjects with DM-PN. In addition, subjects with DM-PN had greater percent contribution of passive torque to total plantar flexor muscle torque, compared to controls. These findings suggest that patients with decreased active torque production may use passive torque to maximize total torque output.

ACKNOWLEDGMENTS

The authors thank Richard Gajdosik, PhD, PT, for his assistance with the development of the plantar flexor stiffness measurement methods; Scott D. Minor, PhD, PT, for his contributions to study design and methods; and Mary Hastings, MHS, PT, for her assistance with data collection and analysis.

REFERENCES

1. Alnaqeeb MA, Al Zaid NS, Goldspink G. Connective tissue changes and physical properties of developing and ageing skeletal muscle. *J Anat.* 1984;139:677–689.
2. Andersen H, Gadeberg PC, Brock B, Jakobsen J. Muscular atrophy in diabetic neuropathy: a stereological magnetic resonance imaging study. *Diabetologia.* 1997;40:1062–1069.
3. Andersen H, Mogensen PH. Disordered mobility of large joints in association with neuropathy in patients with long-standing insulin-dependent diabetes mellitus. *Diabetic Medicine.* 1997;14:221–227.
4. Andersen H, Poulsen PL, Mogensen CE, Jakobsen J. Isokinetic muscle strength in long-term IDDM patients in relation to diabetic complications. *Diabetes.* 1996;45:440–445.
5. Beer FP, Johnston ER. *Vector Mechanics for Engineers—Statics.* 5th ed. New York, NY: McGraw-Hill Book Company; 1988.
6. Birke JA, Sims DS, Buford WL. Walking casts: effect on plantar foot pressures. *J Rehabil Res Dev.* 1985;22:18–22.
7. Brown M, Fisher JS, Salsich GB. Stiffness and muscle function with age and reduced muscle use. *J Orthop Res.* 1999;17:409–414.
8. Chesworth BM, Vandervoort AA. Comparison of passive stiffness variables and range of motion in uninvolved and involved ankle joints of patients following ankle fractures. *Phys Ther.* 1995;75:253–261.
9. Chleboun GS, Howell JN, Conaster RR, Giesey JJ. The relationship between elbow flexor volume and angular stiffness at the elbow. *Clin Biomech.* 1997;12:383–392.
10. Cohen J, Cohen P. *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences.* 2nd ed. London, England: Lawrence Erlbaum Associates; 1983.
11. Cole GK, van den Bogert AJ, Herzog W, Gerritsen KGM. Modelling of force production in skeletal muscle undergoing stretch. *J Biomech.* 1996;29:1091–1104.
12. Fugl-Meyer AR, Gustafsson L, Burstedt Y. Isokinetic and static plantar flexion characteristics. *Eur J Appl Physiol.* 1980;45:221–234.
13. Gajdosik RL, Vander Linden DW, Williams AK. Influence of age on concentric isokinetic torque and passive extensibility variables of the calf muscles of women. *Eur J Appl Physiol.* 1996;74:279–286.
14. Gajdosik RL, Vander Linden DW, Williams AK. Influence of age on the length and passive elastic stiffness characteristics of the calf muscle-tendon unit of women. *Phys Ther.* 1999;79:827–838.
15. Gillette PD, Fell RD. Passive tension in rat hindlimb during suspension unloading and recovery: muscle/joint contributions. *J Appl Physiol.* 1996;81:724–730.
16. Halbertsma JP, van Bolhuis AI, Goeken LN. Sport stretching: effect on passive muscle stiffness of short hamstrings. *Arch Phys Med Rehabil.* 1996;77:688–692.
17. Hill AV. The heat of shortening and the dynamic constants of muscle. *Proc R Soc Lond B.* 1938;126:136–195.
18. Hill AV. The series elastic component of muscle. *Proc R Soc Lond B.* 1950;137:273–280.
19. Horowits R, Kempner ES, Bisher ME, Podolsky RJ. A physiological role for titin and nebulin in skeletal muscle. *Nature.* 1986;323:160–164.
20. Huxley AF. Muscle structure and theories of contraction. *Prog Biophys Biophys Chem.* 1957;7:255–318.
21. Lahey MA, Downey RG, Saal FE. Intraclass correlations: there's more there than meets the eye. *Psych Bull.* 1996;93:586–595.
22. Lakie M, Robson LG. Thixotropy: the effect of stretch size in relaxed frog muscle. *Q J Exp Physiol.* 1988;73:127–129.
23. Lamontagne A, Malouin F, Richards CL. Viscoelastic behavior of plantar flexor muscle-tendon unit at rest. *J Orthop Sports Phys Ther.* 1997;26:244–252.
24. Lieber RL. *Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine.* Baltimore, Md: Williams & Wilkins; 1992.
25. Locke M, Perry J, Campbell J, Thomas L. Ankle and subtalar motion during gait in arthritic patients. *Phys Ther.* 1984;64:504–509.
26. Magid A, Law DJ. Myofibrils bear most of the resting tension in frog skeletal muscle. *Science.* 1985;230:1280–1282.
27. Mueller MJ, Diamond JE, Delitto A, Sinacore DR. Insensitivity, limited joint mobility, and plantar ulcers in patients with diabetes mellitus. *Phys Ther.* 1989;69:453–462.

28. Mueller MJ, Minor SD, Sahrmann SA, Schaff JA, Strube MJ. Differences in the gait characteristics of patients with diabetes and peripheral neuropathy compared with age-matched controls. *Phys Ther.* 1994;74:299–313.
29. Perry J. *Gait Analysis: Normal and Pathological Function.* Thorofare, NJ: SLACK, Inc; 1992.
30. Rosenthal R, Rosnow RL. *Contrast Analysis: Focused Comparisons in the Analysis of Variance.* New York, NY: Cambridge University Press; 1985.
31. Salsich GB, Mueller MJ. Effect of plantar flexor muscle stiffness on selected gait characteristics. *Gait & Posture.* 2000;11:207–216.
32. Salsich GB, Mueller MJ, Sahrmann SA. Passive ankle stiffness in subjects with diabetes and peripheral neuropathy versus an age-matched comparison group. *Phys Ther.* 2000;80:352–362.
33. Siegler S, Moskowitz D. Passive and active components of the internal moment developed about the ankle joint during human ambulation. *J Biomech.* 1984;17:647–652.
34. Simard CP, Spector SA, Edgerton VR. Contractile properties of rat hind limb muscles immobilized at different lengths. *Exp Neurol.* 1982;77:467–482.
35. Simoneau GG, Ulbrecht JS, Derr JA, Becker MB, Cavanagh PR. Postural instability in patients with diabetic sensory neuropathy. *Diabetes Care.* 1994;17:1411–1421.
36. Sinkjaer T, Magnussen I. Passive, intrinsic and reflex-mediated stiffness in the ankle extensors of hemiparetic patients. *Brain.* 1994;117:355–363.
37. 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;69:656–662.
38. Tardieu C, Tabary JC, Tabary C, Tardieu G. Adaptation of connective tissue length to immobilization in the lengthened and shortened positions in cat soleus muscle. *J Physiol.* 1982;78:214–220.
39. Vandervoort AA, Chesworth BM, Cunningham DA, Paterson DH, Rechnitzer PA, Koval JJ. Age and sex effects on mobility of the human ankle. *J Gerontol.* 1992;47:M17–21.
40. Vlassara H. Advanced nonenzymatic tissue glycosylation: cell-mediated interactions implicated in the complications associated with diabetes and aging. *Blood Purif.* 1990;8:223–232.
41. Williams PE, Goldspink G. Changes in sarcomere length and physiological properties in immobilized muscle. *J Anat.* 1978;127:459–468.
42. Winter DA. Kinematic and kinetic patterns in human gait: variability and compensating effects. *Human Movement Science.* 1984;3:51–76.
43. Winter DA. *Biomechanics and Motor Control of Human Movement.* 2nd ed. New York, NY: John Wiley & Sons, Inc; 1990.
44. Winter DA, Patla AE, Frank JS, Walt SE. Biomechanical walking pattern changes in the fit and healthy elderly. *Phys Ther.* 1990;70:340–347.
45. Zajac FE. Muscle and tendon: properties, models, scaling, and application to biomechanics and motor control. *Critical Reviews in Biomedical Engineering.* 1989;17:359–411.