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Measurement of passive ankle stiffness in subjects with chronic hemiparesis using a novel ankle robot

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Roy A, Krebs HI, Bever CT, Forrester LW, Macko RF, Hogan N. Measurement of passive ankle stiffness in subjects with chronic hemiparesis using a novel ankle robot. *J Neurophysiol* 105: 2132–2149, 2011. First published February 23, 2011; doi:10.1152/jn.01014.2010.—Our objective in this study was to assess passive mechanical stiffness in the ankle of chronic hemiparetic stroke survivors and to compare it with those of healthy young and older (age-matched) individuals. Given the importance of the ankle during locomotion, an accurate estimate of passive ankle stiffness would be valuable for locomotor rehabilitation, potentially providing a measure of recovery and a quantitative basis to design treatment protocols. Using a novel ankle robot, we characterized passive ankle stiffness both in sagittal and in frontal planes by applying perturbations to the ankle joint over the entire range of motion with subjects in a relaxed state. We found that passive stiffness of the affected ankle joint was significantly higher in chronic stroke survivors than in healthy adults of a similar cohort, both in the sagittal as well as frontal plane of movement, in three out of four directions tested with indistinguishable stiffness values in plantarflexion direction. Our findings are comparable to the literature, thus indicating its plausibility, and, to our knowledge, report for the first time passive stiffness in the frontal plane for persons with chronic stroke and older healthy adults.

robotics; anklebot; ankle impedance

EACH YEAR ABOUT 790,000 AMERICANS suffer a stroke, making it the leading cause of adult disability in the country (American Heart Association 2009). Damage to descending pathways, as occurs in stroke, results in several forms of motor and/or sensory impairment (Katz and Rymer 1989; Young 1994). In the lower extremity, a common condition that afflicts mobility following a stroke is "drop foot" that refers to an inability to lift the foot owing to weakness in the dorsiflexor muscles. Both peripheral and central lesions are listed as possible reasons for foot drop; these include muscle disease, Peroneal nerve, Sciatic nerve, Lumbosacral plexus, or the L5 nerve root. The two major complications of drop foot are slapping of the foot after heel strike (foot slap) and improper clearing of the ground during the swing phase (toe drag). Furthermore, maintaining ankle stability during the stance phase is also essential for proper walking.

Ankle stability is influenced by passive mechanisms, e.g., ligamentous stiffness, as well as active mechanisms and neuromotor mechanisms such as reflex and voluntary control. Studies have shown that healthy subjects are able to accommodate surface changes (Ferris and Farley 1997; Ferris et al. 1998) and changes in gait speed by modulating their leg or ankle stiffness (Hansen et al. 2004). Adequate ankle-joint stiffness is also critical during the single support phase to control forward and downward body momentum (Lark et al. 2003), and ankle impedance (i.e., stiffness plus damping and other dynamic factors) is important for "shock absorption." It has been suggested that the impact force at floor contact is attenuated by the good cushioning during the supination and pronation of the ankle joint (Bahlsen and Nigg 1987).

In neurologically impaired individuals, however, spasticity (reflex hyperexcitability and hypertonus) might disrupt the remaining functional use of muscles (Chung et al. 2004). It may be accompanied by structural changes of muscle fibers and connective tissue, which may result in alterations of intrinsic mechanical properties of a joint. Studies have shown, for example, that those with spinal cord injury (Mirbagheri et al. 2001), spastic cerebral palsy (Lieber and Fridén 2002), multiple sclerosis (Zhang et al. 2000), or stroke (Chung et al. 2004) have abnormal passive ankle stiffness in addition to hypertonia[1] (caused by spasticity, dystonia, or rigidity, individually or in combination). Tracking passive ankle stiffness in neurologically impaired individuals over the course of an intervention program may lead to better characterization and assessment of a patient's improvement (Selles et al. 2005) and may even serve as signatures of ankle pathology.

Despite extensive literature on the subject, there appears to be little consensus within the biomechanics or motor-control communities about the accepted definition of terms such as stiffness. In the most general sense, dynamic impedance is a property of a system that maps the time history of displacement (or angle) onto the time history of force (or torque) and includes resistance to motion-related displacement, velocity, acceleration, and any other dynamic factors. In steady state, a linearized approximation relating steady displacement to steady force is characterized by a constant of proportionality known simply as stiffness. Stiffness can be categorized on the

[1] Hypertonia is defined simply as an abnormally increased resistance to externally imposed movement about a joint (Sanger et al. 2003). Spasticity, on the other hand, is defined as hypertonia in which one or both of the following signs are present: *1*) resistance to externally imposed movement increases with increasing speed of stretch and varies with the direction of joint movement, and/or *2*) resistance to externally imposed movement rises rapidly above a threshold speed or joint angle (Sanger et al. 2003).

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basis of whether it is measured under passive or active conditions. Passive stiffness can be defined as the resistance to elongation or shortening or, in physical terms, the change in tension per unit change in length. In the biological context, it refers to the mechanical stiffness provided by the combination of the joint, tendon, and connective tissue. Passive tension is generated when a passive (not contracting) muscle is lengthened, and it is believed to originate from the series elastic and parallel elastic elements of muscle, e.g., the tendon, structural proteins within the myofibril, connective tissue around the muscle fibers and fascicles (Salsich et al. 2000). Although the exact source of passive stiffness is debatable, several researchers (Magid and Law 1985; Wang et al. 1991) have suggested that structures containing collagen within the muscle tendon unit (e.g., perimysium) contribute to it mostly at end range (long sarcomere lengths) and that, within the physiological range of muscle length change, passive stiffness can be attributed to structures inside the myofibril (e.g., structural proteins such as titin). Active stiffness, on the other hand, is a function of muscle activation and the stretch-reflex-mediated contraction of the muscle fibers. Active tension is generated when the muscle receives input at the neuromuscular junction (e.g., during a voluntary or reflexive contraction) and has been attributed to structures within the contractile muscle element. Another property, the intrinsic joint stiffness, is one that provides an immediate torque response to any change in joint angle without any intervention required from the nervous system (Loram and Lakie 2002). It has been defined as the instantaneous mechanical stiffness provided by the combination of active muscle, tendon, and connective tissue (Loram and Lakie 2002). Whereas there is some evidence to suggest that the nervous system sets or modulates ankle stiffness (Carpenter et al. 1999; Gatev et al. 1999), others argue that intrinsic ankle stiffness is not under neural control but is a biomechanical constant (Loram and Lakie 2002).

Objective quantitative techniques to estimate passive ankle-joint stiffness include using perturbations of torque or angular displacement and measuring the resulting angular displacements or torques, respectively (Agarwal and Gottlieb 1977; Hunter and Kearney 1982; Kearney and Hunter 1982; Weiss et al. 1986a; 1986b; 1988). For instance, Zhang and colleagues developed a stretching device to treat the spastic and/or contractured ankle of neurologically impaired patients (Zhang et al. 2002). Their device stretches the ankle throughout the range of motion (ROM) and evaluates treatment outcome quantitatively, including joint stiffness. More recently, Lorentzen *et al.* (2010) used a computer-controlled robotic device to apply stretches to the ankle plantar flexor muscles at different velocities to distinguish the contribution of active reflex mechanisms from passive muscle properties in healthy participants and those with stroke, multiple sclerosis, and spinal cord injuries. They defined passive stiffness to be the applied torque recorded at the slowest stretch velocity (8°/s) at which no stretch-reflex response was elicited. Others have used passive perturbations generated by force plates (e.g., Rydahl and Brouwer 2004) or dynamometers (e.g., Lamontagne et al. 1997) to measure the resultant angular displacement. Using system-identification techniques and assuming linearity, ankle inertia, damping, stiffness (and other dynamic factors) can be approximated for various perturbations at different ankle angles and for different levels of muscular activation (Kearney and Hunter 1990).

Whereas the passive ankle stiffness has been estimated and reported extensively, both for healthy and neurologically impaired human subjects, nearly all those measurements have been made in the sagittal plane, i.e., the dorsi-plantarflexion degree-of-freedom (DOF) (Rydahl and Brouwer 2004; Lamontagne et al. 1997, Chung et al. 2004, Sinkjær et al. 1988; Harlaar et al. 2000; Singer et al. 2002; Lorentzen et al. 2010). In contrast, with the exception of only a few studies (e.g., Zinder et al. 2007), very little information is available on the ankle-joint stiffness in healthy individuals in the frontal plane, i.e., eversion-inversion DOF. In fact, to the best of our knowledge, no such information is available on subjects with hemiparetic stroke despite the fact that frontal-plane mechanics is important in the maintenance of balance and prevention of injury under a variety of conditions.

We have recently developed an ankle robot ("anklebot") with the dual purpose of providing a new therapeutic training modality and evaluation of the ankle impairment. The anklebot is a three-DOF wearable robot, backdriveable with low intrinsic mechanical impedance, and it provides actuation in two of these degrees of freedom, namely plantar-dorsiflexion and inversion-eversion (Roy et al. 2009). We are clinically testing a β-prototype of the anklebot in collaboration with the Baltimore Veterans Administration Medical Center. Here we estimate both sagittal- and frontal-plane passive ankle stiffness in chronic stroke subjects as well as young and age-matched healthy control subjects. We postulate that the increased tone observed in stroke patients would be reflected by an increased passive stiffness attributable to the changes in mechanical properties of muscle fibers, as observed in spastic patients (Fridén and Lieber 2003).

METHODS

Subjects

Ten chronic, hemiparetic stroke survivors (60 ± 8 yr) were enrolled in the stroke group (ST). Stroke subjects met the following inclusion criteria: *1*) older than 21 yr at the time of examination, *2*) duration of stroke before examination more than 6 mo for ischemic stroke or 12 mo for hemorrhagic stroke, *3*) not undergoing conventional physical therapy, *4*) adequate language and neurocognitive function to understand instructions, and *5*) residual hemiparetic gait deficits. The healthy population included two groups consisting of ten subjects in each group: an age-matched control group (AC) comprising of subjects (5 men, 5 women) between 55 and 71 yr of age (59 ± 6 yr), and a young healthy group (YH) comprised of subjects (6 men, 4 women) between 24 and 40 yr of age (32 ± 5 yr). Healthy subjects in both groups met the following inclusion criteria: *1*) no previous history of ankle injury, and 2) no history of neurological impairment. All subjects provided written informed consent before testing. Table 1 summarizes the demographics of the subjects who participated in this study.

Clinical Assessment

All stroke volunteers underwent routine medical and cardiovascular evaluations in the Baltimore VA Geriatric Research Education and Clinical Center (GRECC) Assessment Clinic. In addition, each stroke subject received standard neurological evaluations [NIH Stroke Scale, Modified Ashworth Scale (MAS)][2] to measure muscle tone (only in

[2] Performed bilaterally and the unaffected side tested first. Scores were graded on a subjective scale from 0 to 5, with 5 representing that patient can hold the position against maximum resistance and through complete ROM and 0 representing that no contractile activity can be felt in the gravity-eliminated condition.

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Table 1. *Subject characteristics for healthy and stroke groups*

Characteristic	ST	YH	AC
Age, yr	61 ± 8	32 ± 5	59 ± 6
Height, cm	173.4 ± 11.8	167.3 ± 13.1	164.4 ± 8.5
Weight, kg	80.2 ± 10.4	61.5 ± 16.3	67.0 ± 19.8
NIH Stroke Scale (0–42)	2–9	N/A	N/A
Ankle Tested	Paretic (5 right, 5 left)	Right	Right
Time Poststroke, yr	3.5 ± 1.9	N/A	N/A
Sex Distribution	6 males, 4 females	6 males, 4 females	5 males, 5 females
Overall PROM, degree	35.2 ± 7.2 (28–42)	79.0 ± 21.2 (64–94)	80.1 ± 16.2 (73–97)
Side Tested, number R/L	5 R, 5 L	Dominant	Dominant

	Number of stroke subjects		Number of healthy subjects (YH and AC)	
Modified Ashworth Score (0–5)	Dorsiflexion	Plantarflexion	Dorsiflexion	Plantarflexion
0	3	4	10	10
1	1	2	0	0
2	3	3	0	0
3	3	1	0	0
4	0	0	0	0
5	0	0	0	0

Values of passive range of motion are means ± SD, $n = 10$/group. NIH stroke scale scores range from 0 (normal) to 42; expressed as a range. Values in parenthesis are the range of a parameter in a group. The dominant leg is self-reported. Overall passive range of motion (PROM) is computed as the difference between PROM in plantarflexion and PROM in dorsiflexion. Modified Ashworth Scale is expressed as range from 0–5 with 0 representing no increase in muscle tone and 5 representing affected parts rigid in flexion and extension. YH, young healthy group; AC, age-matched controls; ST, stroke group; R, right side; L, left side. See METHODS for details.

dorsi-plantarflexion), 10-m timed walk, and manual muscle testing for strength (only in dorsi-plantarflexion). Passive and active ROMs were also measured for subjects in each group using a clinical goniometer (Jamar EZ-Read; Jamar, Clifton, NJ) with each subject seated on a plinth such that his/her ankle was freely suspended. Any offsets in the anatomical neutral measured in upright stance were accounted for in the ROM calculations.

Anklebot

The design, characterization, donning procedure, and safety features of the anklebot have been previously described (Roy et al. 2009). At this point, we briefly summarize its salient design features and measurement capabilities. It is a portable exoskeletal ankle robot that allows normal range of motion in all three DOFs but provides independent assistance or resistance in two of those DOFs (dorsi-plantarflexion and eversion/inversion). The anklebot can deliver a continuous net torque of ~23 Nm in dorsi-plantarflexion and 15 Nm in eversion-inversion. The robot can estimate ankle angles with an error less than 1° in both planes of movement (maximum 1.5°) over a wide range of movement (60° in dorsi-plantarflexion and 40° in eversion-inversion) and can measure ankle torques with an error less than 1 N·m. It has low friction (0.744 N·m) and inertia (0.8 kg per actuator for a total of 1.6 kg at the foot) to maximize the backdriveability. A previous study has shown that unilaterally loading the impaired leg with the additional mass of the anklebot had no detrimental effect on the gait pattern in subjects with chronic hemiparesis (Khanna et al. 2010).

Setup

All tests were performed with the subject in a seated position with the knee flexed at 45° and the ankle suspended from a specially constructed chair with adjustable height. Anatomical neutral was taken as the "zero" position and was determined by positioning the foot on the ground at 90° with respect to the long axis of the leg. Subjects experienced a series of passive displacements at their ankle joint (Fig. 1) while in this position such that any translational movement of their knee was physically constrained. This was to avoid any confounding effects of knee translation during dorsi-plantarflexion or eversion-inversion of the ankle and, therefore, "isolate" ankle movement. This postural constraint enables us to isolate any contribution of the mechanical properties of uniarticular knee musculature but at the same time include the effects of biarticular muscles, e.g., the gastrocnemius. In healthy subjects, the right (self-reported dominant) ankle was tested, whereas, in subjects who had endured stroke, the experiment was performed on the paretic ankle (5 right paretic and 5 left paretic). To minimize any contribution of voluntary "human" torque, subjects were instructed not to intervene when the perturbation occurred and to keep their foot relaxed throughout (Palazzolo et al.

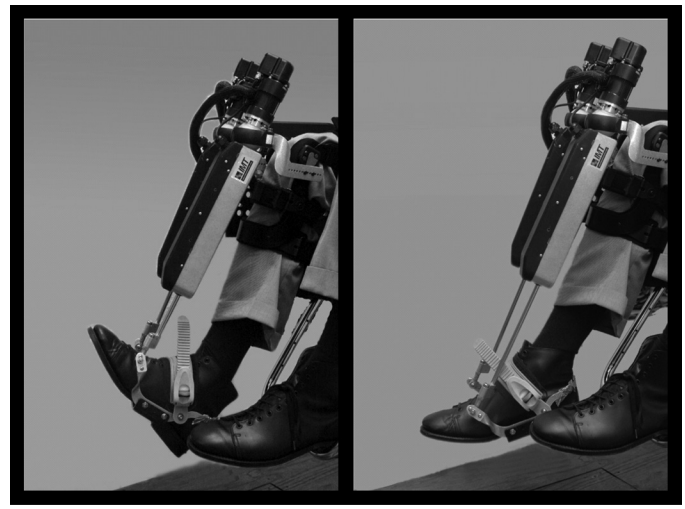


Fig. 1. Photograph of the anklebot applying torques to move the ankle joint in dorsiflexion (toe up) (*left*) and plantarflexion (toe down) (*right*). The subject (not shown here) is seated with the knee flexed at 45° and leg partly suspended from a custom-made barber chair.

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2007). The study protocol was approved by the Massachusetts Institute of Technology Committee on the Use of Humans as Experimental Subjects, the University of Maryland Institutional Review Board, and the Baltimore Veterans Affairs Research and Development Committee.

Stiffness Measurement

The ankle joint was continuously moved at a constant velocity according to ramp-and-hold displacement profile (Fig. 2). The steady-state movement amplitudes (which would have been achieved by the servo in the absence of the human ankle) were different for the stroke and healthy groups and also depended on the direction of movement; in the sagittal plane, the amplitude of each displacement ranged from 20° in plantarflexion to the passive ROM (PROM) in dorsiflexion for the stroke group[3] (7.58 ± 5.18°), and from 20° in plantarflexion to 30° in dorsiflexion for both healthy young and age-matched controls, i.e., each healthy subject experienced a total of 11 movements, and each stroke patient experienced between six and eight movements depending on his/her PROM. In the frontal plane, the amplitude of each movement ranged from 25° in inversion to 20° in eversion for each group, i.e., each subject experienced a total of 10 movements. Each one started from and returned back to neutral position. Movements were made in increments of 5° (e.g., at 0°, ±5° from and to neutral, ±10° from and to neutral, and so on), and this included the neutral position. Furthermore, when the ankle was perturbed in one plane of movement (e.g., dorsi-plantarflexion), no movement (voltage) was commanded in the other plane of movement (e.g., eversion-inversion). By convention, angles in dorsiflexion and eversion were considered positive, and those in plantarflexion and inversion were negative. Torque was assigned a polarity consistent with the direction of the movement that it would generate (e.g., dorsiflexion torque was taken as positive). Each perturbation was made at a constant velocity of 5°/s so as to not evoke stretch reflex (Saripalli and Wilson 2005; Hufschmidt and Mauritz, 1985; Lamontagne et al. 1997; Lorentzen et al. 2010). The passive movement of the ankle occurred with nominal controller settings (robot torsional stiffness and damping) of 100 N·m/rad and 2 N·m·s/rad. Note that, in the steady state, the estimate of passive stiffness is, in theory, invariant to the controller settings (see *Stiffness computation*). Each perturbation was followed by a hold period or interstretch interval in the steady-state position lasting 1 s to obtain a "clean" read-out, followed immediately by the subsequent perturbation, similar to procedures used by Mussa-Ivaldi and Hogan to measure arm postural stiffness (Mussa-Ivaldi et al. 1985) and Lorentzen et al. to measure ankle plantar flexor stiffness in stroke (Lorentzen et al. 2010). We repeated the trial three times at each amplitude to guarantee repeatability.

Electromyography

EMG activity was recorded from the gastrocnemius (GAS) and tibialis anterior (TA) muscles using surface electrodes (Patch electrode with snap connector; Cadwell Laboratories, Kennewick, WA) with encapsulated preamplifiers placed on both the paretic (or right) as well as the unaffected (or left) limbs. Electromyogram (EMG) data was recorded from the contralateral muscles to compare any differences in background activity with that from the paretic muscles. These signals were sampled at 1 kHz, and data collection commenced 5 s before the onset of each perturbation and continued until the completion of the hold phase of movement. The signals were then filtered by an eighth-order zero-lag high-pass Butterworth filter with a cutoff frequency[4] of 475 Hz, rectified, and detrended. A measure of the background EMG activity was then determined for each subject by computing the average EMG activity in an artifact-free epoch lasting from 5 s before the onset of each perturbation. During the movement phase, the mean EMG activity was computed within each trial and compared against a threshold defined as the background activity plus one standard deviation for that trial. This was repeated for each subject across all perturbations. Also, to determine the possibility of transient stretch-reflex activity, the amplitude of each EMG sample acquired through the entire movement phase within each trial was compared against its corresponding threshold.

[3] Dorsiflexion passive range of motion (PROM) was measured by an evaluator passively moving the ankle maximally in dorsiflexion. This was done with the orthopedic boots on and the reading taken using a clinical goniometer. If dorsiflexion PROM was less than 5° for a subject, the smallest (nonzero) perturbation in dorsiflexion was set at 5°.

[4] Per Nyquist sampling theorem, the cutoff frequency of 475 Hz was just under half of the sampling frequency.

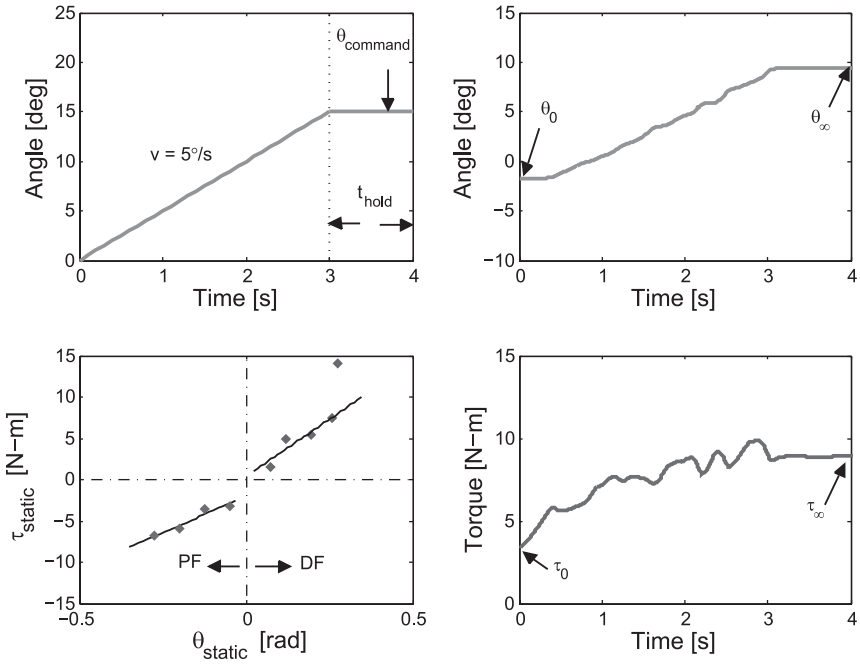


Fig. 2. Measurement of passive stiffness using the anklebot. *Top*, *left*: commanded ramp-and-hold displacement perturbation ($\theta_{command}$) of 15° in dorsiflexion (DF) with constant velocity (v) of 5°/s and hold time (t_{hold}) of 1 s. *Top*, *right*: raw traces of ankle angle and torque from a single representative control subject shown with initial (θ_0, τ_0) and final conditions (θ_∞, τ_∞). *Bottom*, *left*: steady-state torque (τ_{static}) and angle (θ_{static}) data obtained by perturbing the subject's ankle over the entire range of commanded perturbations in the sagittal plane (right positive: dorsiflexion, left negative: plantarflexion, PF). *Bottom*, *right*: each data point is obtained by perturbing the ankle to a commanded angle and measuring the resultant net torque ($\tau_\infty - \tau_0$) and angular displacement ($\theta_\infty - \theta_0$) under static conditions.

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Data Analysis

Stiffness computation. Passive ankle stiffness was estimated using a simple approach (Roy et al. 2009). The theory behind the approach is briefly repeated here. The human ankle is displaced by the application of the vector sum of anklebot torque and voluntary human torque, and, under steady-state (static) conditions, the relation between these measurements provides an estimate of the apparent stiffness of the ankle, i.e., at a given commanded perturbation,

$$K_{human} = (\tau_{robot,\infty} + \tau_{human,\infty})/(\theta_\infty - \theta_0) \quad (1)$$

where K_{human} denotes an estimate of passive ankle stiffness, $\tau_{robot,\infty}$ and $\tau_{human,\infty}$ represent the steady-state machine torque and human torque at the ankle, respectively, and θ_∞ represents the steady-state angular displacement of the ankle. In this approach, the human torque component is minimized by instructing subjects to not intervene. Assuming minimal voluntary contribution, i.e., $\tau_{human} \approx 0$, the total torque displacing the ankle can be considered to be approximately equal to the applied machine torque. Also, the bias torque, i.e., the torque output by the robot when no torque is commanded, is negligible (7×10^{-4} N·m). Under these conditions, the ratio of the net measured torque[5] to net angular displacement yields an estimate of the passive ankle stiffness. Stiffness estimates were thus obtained in each direction of movement within a DOF by fitting the pair-wise steady-state torque and angle data using least-squares linear regression, i.e.,

$$\hat{K}_{human} = \frac{\sum (\theta_i - \bar{\theta}) \cdot (\tau_i - \bar{\tau})}{\sum (\theta_i - \bar{\theta})^2} \quad (2)$$

where $\hat{K}_{human}$ is the least-squares estimate of passive stiffness, θ_i and τ_i are the angular displacement from neutral and applied robot torque for the i^{th} perturbation with corresponding means $\bar{\theta}$ and $\bar{\tau}$, respectively. Because the neutral point was neither in dorsiflexion (or eversion) nor in plantarflexion (or inversion), linear regressors did not include the neutral point. Moreover, given that the loading curve of the torque-angle relationship may be nonlinear with the different stiffness at different operating ranges, we excluded those trials *1*) which led to actuator saturation, i.e., when the physical hard-limit was reached for a given perturbation, or *2*) with "observable" nonlinearity, e.g., at extremities of movement, where the torque-angle relationship tended toward an apparent vertical asymptote. Linear regression was then performed in each direction over the remaining data set.

Statistical analysis. Values of variables or metrics are reported as their means ± SD unless otherwise specified. Standard *t*-test procedures were used to test for significant changes in passive stiffness attributable to subject group and direction of movement. Results with P values <0.05 were considered significant. Intragroup variability was characterized by the standard deviation as well as the coefficient of variation, C_v, i.e., the ratio of mean-to-standard deviation. Correlations between two sets of data were computed using the Pearson product-moment correlation (r^2), if parametric, and using Spearman's rank correlation coefficient (ρ), if nonparametric.

RESULTS

Typical Data

Figure 2 shows the position and torque signals recorded from a typical young healthy subject. The trial shows a recording of ramp-and-hold position perturbation in dorsiflexion with commanded amplitude of 15°, an angular velocity of 5°/s, and a hold period of 1 s. In both DOFs, the average angular velocity of the ankle during the movement phase across all perturbations was between 53–69% of the commanded velocity (e.g., in the sagittal plane, YH: 3.45 ± 0.47°/s; AC: 3.02 ± 0.66°/s; ST: 2.65 ± 0.54°/s). Furthermore, as commanded, the actual ankle velocity during the holding phase was nearly zero for all perturbations in both planes [e.g., in the sagittal plane, YH: 0.0015 ± 0.0002°/s; AC: $(1.5 \times 10^{-4}) \pm (2.9 \times 10^{-4})$°/s; ST: $(39 \times 10^{-4}) \pm (3 \times 10^{-4})$°/s, indicating near-perfect static conditions. Variations in the ankle torque during the hold period were negligible in both DOFs (e.g., in the sagittal plane, YH: $C_v = 0.013 \pm 0.007$; AC: $C_v = 0.011 \pm 0.007$; ST: $C_v = 0.015 \pm 0.011$). The magnitudes of ankle torque attributable to gravity were also relatively "small" in each group; absolute values averaged across the entire range of movements in dorsiflexion and plantar flexion were 1.42 ± 0.41 N·m and 1.30 ± 0.23 N·m for the YH group, 1.46 ± 0.42 N·m and 1.51 ± 0.31 N·m for the ST group, and 1.42 ± 0.41 N·m and 1.43 ± 0.41 N·m for the AC group, respectively. These values contributed, on an average, less that 1.4% and 2.5% to passive stiffness estimates in dorsiflexion and plantar flexion, respectively. Finally, variations in knee flexion-extension angle (measured from initial knee position of 45°) averaged across all perturbations in a given DOF in each group were generally less than ±1° or ~2% of initial angle (YH-Sagittal: 0.09 ± 0.55°; Frontal: 0.57 ± 1.15°; ST-Sagittal: 0.55 ± 1.27°; Frontal: 1.21 ± 1.01°; AC-Sagittal: 0.1 ± 0.15°; Frontal: −0.06 ± 0.15°).

Muscle Activity

Figure 3*A* shows the paretic TA and GAS muscle activity from a typical stroke subject when a perturbation was applied in eversion with commanded amplitude 15° and velocity 5°/s. In all subjects, we found that the mean EMG activity from both muscles was not significantly different from the average background activity (e.g., in the ST group, $P = 0.14$ for TA, $P = 0.46$ for GAS across all sagittal plane perturbations; $P = 0.53$ for TA, $P = 0.33$ for GAS across all frontal plane perturbations), confirming that both muscles were indeed "quiet" during all trials (Fig. 3*B*). Also, the mean background activity of both muscles were similar between the paretic and the contralateral limbs (e.g., $P = 0.29$ between GAS muscle of paretic vs. nonparetic limb, across all subjects in the ST group and for all trials). Finally, the fact that in each group no sample of EMG during movement exceeded its corresponding threshold in any trial provides evidence of absence of stretch-reflex activity.

Dependence on Direction of Movement

In both degrees of freedom, the estimates of passive ankle stiffness depended on the direction of ankle movement (Fig. 4). Mean passive ankle stiffness in each group was significantly higher in dorsiflexion than in plantarflexion [YH: 30.13 ± 18.13 N·m/rad (dorsiflexion), 18.14 ± 7.37 N·m/rad (plantarflexion), $P = 0.008$; ST: 47.92 ± 11.82 N·m/rad (dorsiflexion), 15.44 ± 3.41 N·m/rad (plantarflexion), $P = 0.0001$; AC: 37.74 ± 15.38 N·m/rad (dorsiflexion), 13.02 ± 0.87 N·m/rad (plantarflexion), $P = 0.02$]. Furthermore, within each group, the intersubject variability was higher in dorsiflexion (YH: $C_v = 0.60$; ST: $C_v = 0.24$; AC: 0.40) than in plantarflexion (YH: $C_v = 0.40$; ST: $C_v = 0.22$; AC: $C_v =$

[5] That is, with any nonzero (baseline) values subtracted.

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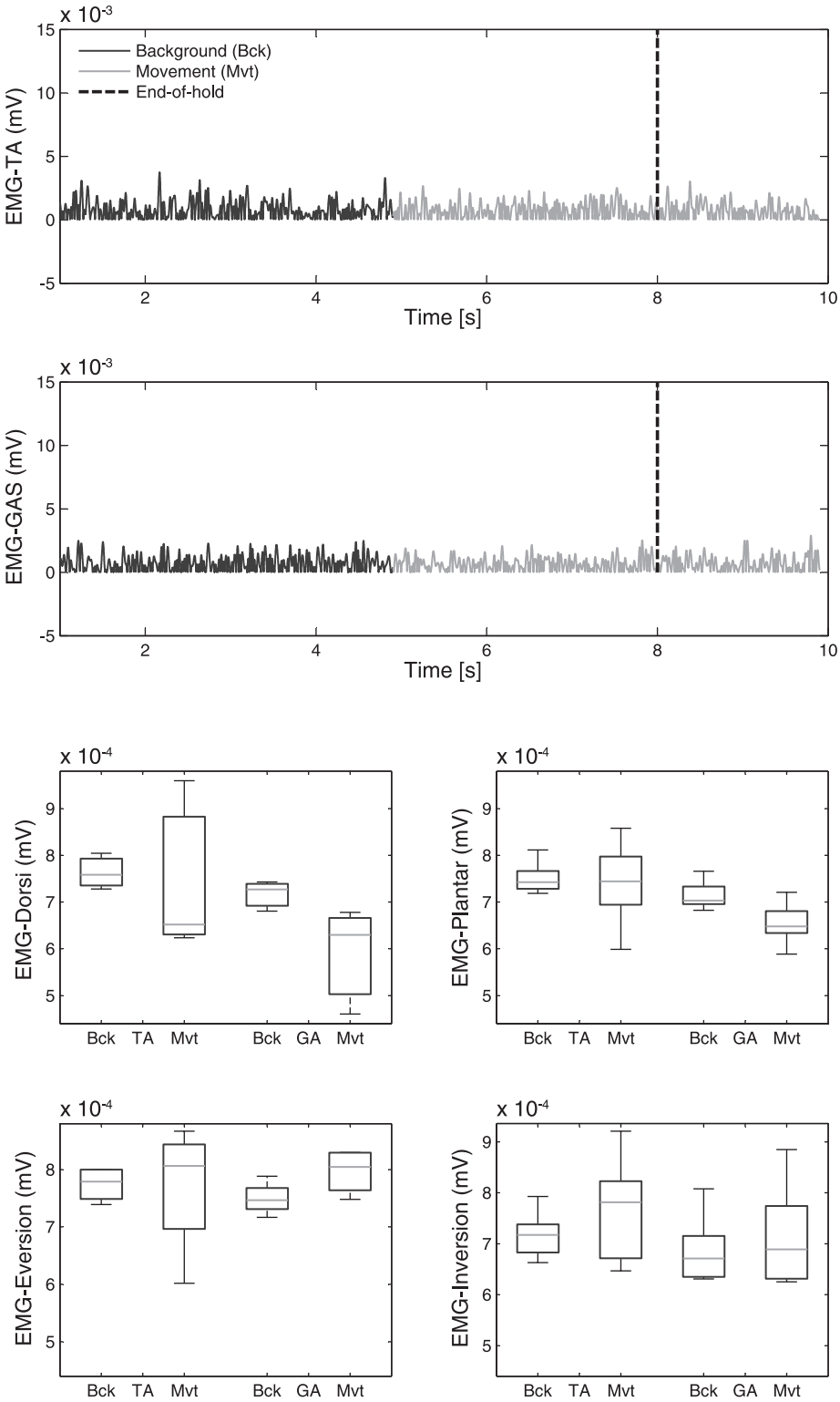


Fig. 3. *Top* and *top*, *middle*: sample electromyogram (EMG) data (in mV) from the tibialis anterior (TA) and gastrocnemius (GAS) muscles in a single stroke subject when undergoing a commanded ankle perturbation of 15° in eversion. The data show three distinct phases: background activity before onset of perturbation (gray), muscle activity during movement until the hold phase (black), and finally, the return of the ankle to its neutral position (to the right of the vertical dashed line); *Bottom* and *bottom*, *middle*: box and whisker plots of electromyographic activity (in mV) from TA and GAS muscles in stroke subjects, averaged over the entire range of commanded perturbations in the sagittal and frontal planes. In each direction of movement, the mean EMG from each muscle is shown separately for background (Bck) and movement (Mvt) phases. In each box plot, the upper and lower ends of each box correspond to the first and third quartiles of the data set, respectively, with the median value shown inside the box; the length of each whisker is 1.5 times the interquartile value; each whisker extends from the edge of the box to the nearest data points within the length of whisker.

0.06). Remarkably, in the eversion-inversion DOF, we found the change of passive stiffness with direction of movement for the stroke group to be opposite that of the healthy groups. Stroke survivors had higher passive stiffness ($P = 0.008$) in inversion (32.15 ± 7.43 N·m/rad) than eversion (20.17 ± 5.09 N·m/rad). On the other hand, healthy subjects in both groups had lower passive stiffness (YH: $P = 0.003$; AC: $P = 0.01$) in inversion (YH: 19.7 ± 3.26 N·m/rad; AC: 19.54 ± 3.19 N·m/rad) than in eversion (YH: 28.19 ± 7.46 N·m/rad; AC: 26.87 ± 1.33 N·m/rad). Unlike the sagittal plane, the intersubject variability of passive stiffness in the frontal plane with respect to direction of movement was group dependent; in the YH and ST groups, it varied more in eversion (YH: $C_v = 0.26$; ST: $C_v = 0.25$) than in inversion (YH: $C_v = 0.16$; ST: $C_v = 0.23$) but vice versa for the AC group (eversion: $C_v = 0.04$; inversion: $C_v = 0.16$).

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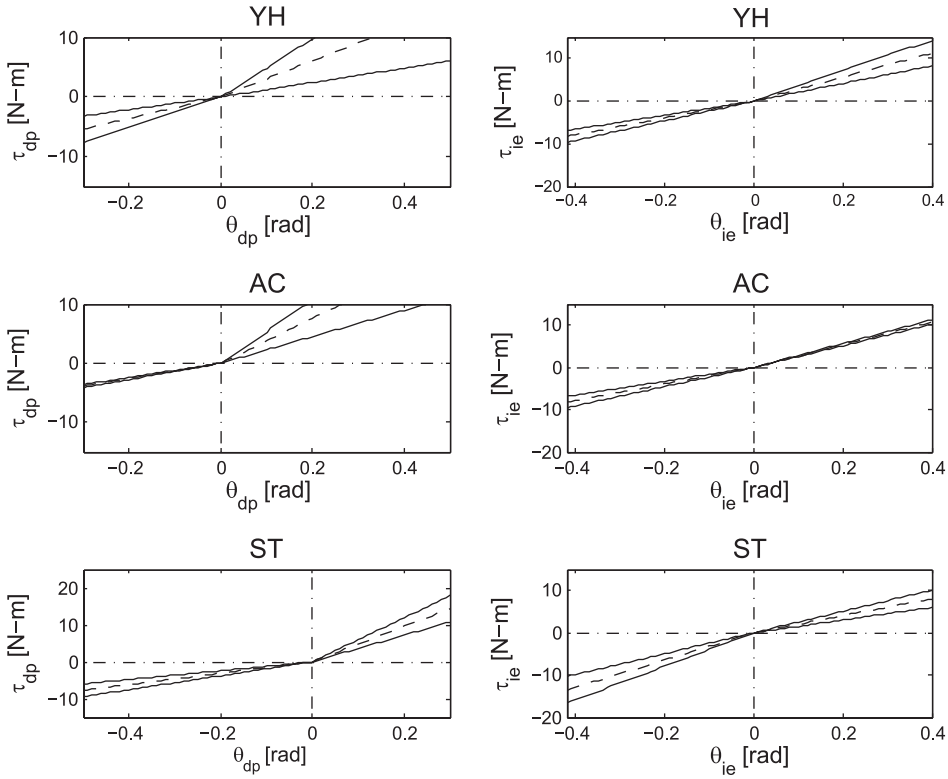


Fig. 4. Linear regressors showing the mean ± SD of passive stiffness estimates across the entire sample of subjects within all three groups (YH: young healthy, AC: age-matched controls, ST: stroke) in both the sagittal (*left*) and frontal (*right*) planes. Passive stiffness is estimated as the slope of the least-squares linear regressor between applied torque τ (sagittal: τ_{dp}, frontal: τ_{ie}) and angular displacement θ (sagittal: θ_{dp}, frontal: θ_{ie}) for each subject. Each regressor is drawn as a mean (dashed line) with 1 SD above and below (solid lines). Separate regressors are computed for estimating ankle stiffness in each direction within a degree of freedom: dorsiflexion (toe up, positive angle) and plantarflexion (toe down, negative angle) in the sagittal plane, and eversion (heel away from midline, positive angle) and inversion (heel toward midline, negative angle) in the frontal plane. The linear regressors are shown with the offset values of robot torque subtracted and are thus forced through the origin. The estimates are shown only for the range of commanded perturbations in each group.

Passive Ankle Stiffness in Stroke Survivors

Intergroup comparisons of passive stiffness in each direction of movement are shown in Fig. 5. Our results showed that, in dorsiflexion, stroke survivors had significantly higher passive stiffness (47.92 ± 11.82 N·m/rad) than both age-matched (37.74 ± 15.38 N·m/rad) as well as young healthy (30.13 ± 18.13 N·m/rad) controls by 27% ($P = 0.05$) and 59% ($P = 0.05$), respectively. However, the passive stiffness of stroke survivors in plantarflexion was similar to those of age-matched controls (ST: 15.44 ± 3.41 N·m/rad, AC: 13.02 ± 0.87 N·m/rad, $P = 0.15$) and young healthy individuals (YH: 18.14 ± 7.37 N·m/rad, $P = 0.37$). In the frontal plane, the disabled ankle was stiffer in inversion (ST: 32.15 ± 7.43 N·m/rad, AC: 19.54 ± 3.19 N·m/rad, $P = 0.01$) but more compliant in eversion (ST: 20.17 ± 5.09 N·m/rad, AC: 26.87 ± 1.33 N·m/rad, $P = 0.006$). This finding was also true compared with the frontal plane stiffness of young healthy subjects (eversion: 28.19 ± 7.46 N·m/rad, $P = 0.03$, inversion: 19.7 ± 3.26 N·m/rad, $P < 0.0005$).

Dependence on Anthropometric Measures

We tested whether simple anthropometric measures can be used as qualitative intrapopulation predictors of passive ankle stiffness. Studies, for example, have shown that the stiffness of the knee joint is significantly correlated to body height and weight (Cammarata et al. 2007). There is little in the literature, however, that explores dependence of passive ankle-joint stiffness on whole body measures. We used regression analysis to

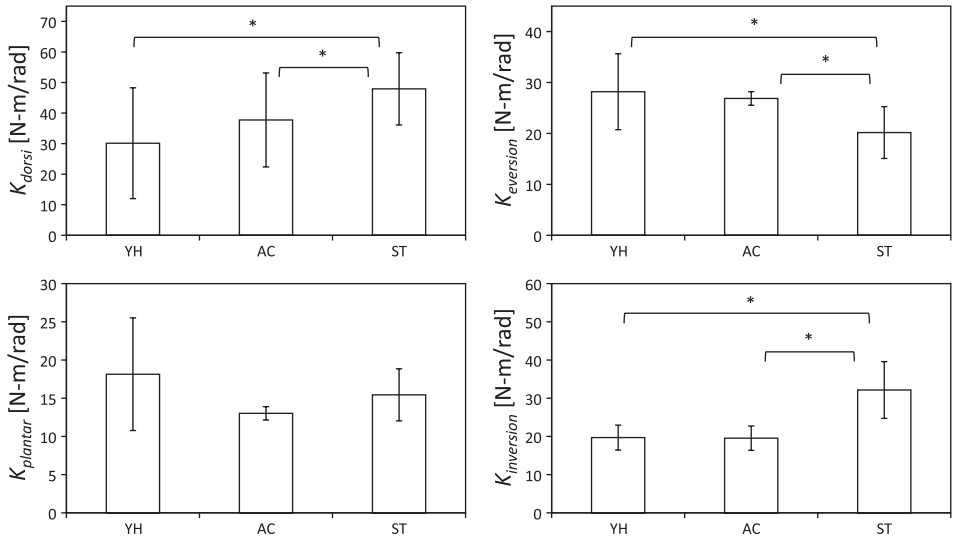


Fig. 5. Comparison of passive stiffness between healthy (YH and AC) and stroke (ST) groups in (counterclockwise from top left) dorsiflexion (K_{dorsi}), plantarflexion ($K_{plantar}$), inversion ($K_{inversion}$), and eversion ($K_{eversion}$). The error bars represent variability across all subjects tested within a group. Statistical significance between YH or AC vs. ST groups is $^*P < 0.05$.

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determine whether there were any significant correlations between passive stiffness and whole body height, weight, and the product of body height and weight, for each group and direction of movement (Table 2). We found that *1*) in both healthy groups, passive stiffness strongly correlated with body height (YH: $r^2 = 0.74$, $P = 0.01$; AC: $r^2 = 0.95$, $P = 0.02$) as well as with the product of body height and weight in dorsiflexion (YH: $r^2 = 0.53$, $P = 0.05$; AC: $r^2 = 0.91$, $P = 0.04$) but not significantly in plantarflexion. No significant correlations emerged in the frontal plane for both groups ($P > 0.5$). On the other hand, however, we found that *2*) in the stroke group, none of the anthropometric metrics correlated significantly with passive ankle stiffness in either plane of movement.

Correlation with MAS Scores

Despite the inherent limitations of the MAS, it is widely reported as an indicator of altered physiological function, e.g., abnormal muscle tone. Therefore, in the subjects with stroke we tested whether estimates of passive stiffness in the paretic ankle were correlated to it. We performed Spearman's rank-correlation analysis to determine any dependencies of passive ankle stiffness in the sagittal plane, specifically dorsiflexion, to the MAS. We found only a very weak correlation between these measurements ($\rho = -0.12$, $P = 0.63$).

Relationship to Walking Speed

Studies have shown that active ankle stiffness varies strongly with measures of mobility function, e.g., gait velocity (Palmer et al. 2002). Whether a similar relationship is true for passive ankle stiffness, especially in neurologically impaired individuals, is debatable and has been the subject of very few studies (e.g., Lamontagne et al. 2000). As an example, there is some evidence that passive stiffness added a unique contribution to walking speed in subjects with diabetes and peripheral neuropathy (Salsich and Mueller 2000; Salsich et al. 2000), but no such relationship was found in subjects with hemiparesis early after stroke (Lamontagne et al. 2000). In our study, no significant correlations emerged between passive ankle stiffness in both planes of movement and gait speed ($P > 0.05$) for any of the groups, a finding consistent with Lamontagne's study.

DISCUSSION

Methodological Considerations

Our paradigm evaluates the linear mapping between steady-state applied torque and output angle to estimate passive stiffness, a key system output. We believe that such an approach may have distinct advantages over anatomical/physiological approaches, which depend on the selected system structure and are valid only if the underlying structure corresponds to that assumed. In contrast, our method is free of any structural assumption and directly measures angular displacement of the ankle in response to applied torque at the ankle. In particular, our method does not assume the underlying system structure to consist of elastic springs alone; rather, the contribution of velocity-dependent components (e.g., damping) are, in theory, zero because input-output measurements are made at steady state. In fact, even if this were not the case, an ongoing study shows that conservative components of the elastic force fields at the ankle are much larger than its nonconservative components, providing evidence that the behavior of the neuromuscular system of the ankle is predominantly elastic or spring-like (Hyunglae et al. 2009; Ho et al. 2009). Furthermore, our paradigm is easy to implement in the clinical setting, takes little time to complete (order of minutes), and, most importantly, enables estimation of ankle stiffness in multiple DOFs.

Confounding Factors

Despite the inherent simplicity of the methodology, a number of factors had the potential to confound the fidelity of the data. These were taken into account and were controlled as best as possible without compromising the simplicity of the paradigm. They included *1*) in-shoe slippage: movement or slippage of the foot within the orthopedic shoe could confound the accuracy of kinematic and kinetic measurements. This was addressed by including only those subjects in the study whose shoe size was available[6]. *2*) Movements were not entirely passive: any voluntary torque contribution could result in overestimation of passive stiffness. The do-not-intervene paradigm, in which the subject is asked to not react to the experimental changes, has been used in previous studies in-

[6] The following shoe sizes could be accommodated: 4, 7, 10 1/2, and 11 1/2 (men) and 5 1/2, 8 1/2, 12, and 13 (women).

Table 2. *Correlation between passive ankle stiffness and whole body anthropometric measures*

Group	DOF	Direction	Height	Weight	Height × Weight
ST	Sagittal	Dorsiflexion	2.92 (0.68)	2.73 (0.69)	2.56 (0.70)
		Plantarflexion	8.60 (0.48)	1.97 (0.74)	3.15 (0.67)
	Frontal	Eversion	18.89 (0.38)	2.86 (0.74)	6.99 (0.61)
		Inversion	13.04 (0.48)	~0 (0.99)	0.94 (0.85)
YH	Sagittal	Dorsiflexion	**74.1* (0.01)**	45.5 (0.09)	**53.7* (0.05)**
		Plantarflexion	42.8 (0.11)	32.9 (0.17)	36.2 (0.15)
	Frontal	Eversion	1.8 (0.72)	3.2 (0.64)	0.9 (0.80)
		Inversion	0.2 (0.89)	1.1 (0.78)	0.9 (0.80)
AC	Sagittal	Dorsiflexion	**95.3* (0.02)**	0.14 (0.96)	**91.5* (0.04)**
		Plantarflexion	10.72 (0.67)	10.53 (0.10)	9.74 (0.68)
	Frontal	Eversion	29.34 (0.45)	82.73 (0.09)	77.1 (0.12)
		Inversion	6.69 (0.74)	87.59 (0.06)	0.69 (0.91)

Values are means of Pearson's correlation coefficient (r^2) expressed as percentage, $n = 10$/group. Values in parenthesis are the P value. *Values with $P \leq 0.05$ are considered significant and are printed in bold. DOF, degree of freedom.

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volving both the upper extremity, e.g., arm (Palazzolo et al. 2007) and wrist (Hicks et al. 2006) as well as lower extremity, e.g., lower leg (Moritomo et al. 2006), and has been found to be effective. We do, however, acknowledge that mechanical responses elicited from human subjects during protocols that use "do not intervene" as an instruction have the potential to be highly variable (Walshe et al. 1996; Crago et al. 1976). Analysis of our EMG signals confirmed, however, that the muscle activity for both the TA and GAS muscles were well below the variability of background activity for all subjects in each group and across all perturbations, indicating that subjects indeed remained passive throughout all perturbations (see Fig. 3*B*). This affirms that our ankle position and torque data yielded a "near-true" estimate of passive ankle stiffness. *3*) We also considered elicitation of stretch reflex: we used a slow-perturbation velocity to prevent evoking stretch-reflex activity (Saripalli and Wilson 2005; Lamontagne et al. 1997; Lorentzen et al. 2010). The decision to set 5°/s as the perturbation velocity in this protocol was, in part, based on previous work that has shown that stretch velocities of this magnitude do not evoke stretch reflex in both healthy (Saripalli and Wilson 2005; Lorentzen et al. 2010) and stroke (Hufschmidt and Mauritz 1985; Lorentzen et al. 2010) subjects under similar experimental conditions. In a recent study, Lorentzen et al. (2010) measured the evoked ankle torque at 17 different stretch velocities (8°/s to 200°/s) to compute the threshold for elicitation of the stretch reflex and found that the average lowest velocity at which a reflex response was evoked in stroke or healthy subjects was between 70.3°/s and 77.1°/s. EMG analysis also confirmed the absence of any myotatic reflex activity in all three groups. *4*) We considered effects of fatigue on passive joint stiffness: the duration of our experiments were short (≤5 min per subject excluding pre- and posttrial times). Also, verbal feedback was taken from each subject to assess whether they felt fatigued, and, if so, a 30-s rest period was provided. Despite these measures, there is always a possibility of fatigue having set in during one or more perturbation trials. Fatigue has the potential to influence passive stiffness of a joint. Studies have shown, for instance, that intrinsic stiffness decreases with fatigue in the elbow (Zhang and Rymer 2001). At the ankle, however, the effective stiffness has been shown to be invariant with fatigue (Hunter and Kearney 1983). This leads us to believe that fatigue in our subjects, if any, would have little or no influence on the passive stiffness of the ankle joint. *5*) We considered effects of gravity: the magnitudes of gravitational torques showed that gravity had a negligible effect on the estimates of passive ankle stiffness. *6*) We considered effects of knee movement: although translational knee movement was constrained, it was free to move in flexion-extension. Initial knee angle was set at 45° flexion for all subjects; however, during perturbations, if changes in knee movement during perturbation were large enough, it could influence components of stiffness related to gravitational or inertial components. Our analysis showed that variations in the mean flexion-extension as measured from initial knee position of 45° less than ±1° in any group were likely because of the subject's in-seat postural adjustments in between perturbation trials.

Direction-Dependence of Passive Ankle Stiffness: A Physiological Model

One of our main findings was that passive ankle stiffness was strongly dependent on the direction of movement in both the healthy and the disabled ankle. What could be a physiological basis for this direction-dependence? A dependence of passive stiffness of the ankle on the direction of movement has also been reported by Chung et al. (2004). A similar dependence has been found in other joints as well, e.g., for shoulder and elbow (Mussa-Ivaldi et al. 1985). There is evidence to suggest that this dependence could be attributable to the summed physiological cross-sectional area (SPCA) of the antagonist group of muscles undergoing passive stretch (Lamontagne et al. 2000). Furthermore, passive joint stiffness should also be proportional to the square of the mean moment arm of the antagonist muscle group. Assuming that the passive resistance to stretching is a main contributor to joint impedance (Rijnveld and Krebs 2007) and that agonist muscles go slack during passive stretch, we hypothesize that passive stiffness of the ankle joint is directly related to the SPCA of the antagonist muscle group lengthened during passive stretch. Accordingly, we propose a physiological model that relates a mechanical property of a joint, i.e., passive stiffness, to the intrinsic muscle properties of individual muscles or muscle groups:

$$\frac{k(\theta_+)}{k(\theta_-)} = \frac{\sum_i A_i^{ant}}{\sum_i A_i^{ag}} \cdot \left(\frac{r_{ant}}{r_{ag}}\right)^2 \qquad (3)$$

where $k(\theta_+)$ and $k(\theta_-)$ represent the passive ankle stiffness in a direction of movement taken by convention to be positive (e.g., dorsiflexion) and negative (e.g., inversion), respectively, A_i^{ant} and A_i^{ag} represent the physiological cross-sectional area (PCA) of the i^{th} muscle belonging to the antagonist and agonist muscle groups undergoing passive stretch to cause a corresponding movement in the θ_+ and θ_- directions, respectively, and r_{ant}, and r_{ag} represent the mean muscle-moment arm of the antagonist and agonist muscle groups, respectively.

If our hypothesis is true, then we may explain the qualitative trends seen between torque and angle and, in particular, the direction dependence of passive ankle stiffness. We expect to find *1*) the SPCA of plantarflexors and invertors to be higher than the SPCA of dorsiflexors and evertors, respectively, and *2*) the ratio of SPCA of plantar-to-dorsiflexors and that of invertors-to-evertors to be of the order of the ratio of passive stiffness in dorsi-to-plantarflexion and eversion-to-inversion, respectively.

Using cadaver data, we computed the SPCA of each group of physiologically intact muscles that are dorsiflexors, plantarflexors, evertors, and invertors (Yamaguchi et al. 1990), and scaled those values with the square of mean moment arms of each group (Table 3). Note that the cadaver age (young or old), where available, was taken into account in computing the SPCA of the YH and AC groups (Yamaguchi et al. 1990), affording us the ability to accurately test our hypothesis for young healthy subjects and age-matched controls separately. Nevertheless, it should be pointed out that muscle data were generally available for only a single cadaver per group (Friedrich and Brand 1990). Additionally, given that the muscles involved in each group have quite different PCAs, we also computed the ratio of stiffness in one direction vs. the other for

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Table 3. *Physiological data of ankle muscles/muscle groups*

Group	Muscle	YH†			AC†			Moment Arm, cm‡
		PCA, cm^2	SPCA, cm^2	W-SPCA, cm^2	PCA, cm^2	SPCA, cm^2	W-SPCA, cm^2	
Dorsiflexor	TA*	16.88	30.83	22.66	12.7	21.31	16.30	5.15
	EHL	6.49			2.21			
	EDL	7.46			6.4			
Plantarflexor	GAS-L*	14.3	89.3	66.5	8.6	83.36	64.03	4.12
	GAS-M*	17.0			17.04			
	SOL*	58.0			57.72			
Invertor	TA	16.88	68.16	51.77	12.7	42.05	31.79	1.92
	TP*	26.27			18.89			
	FHL	18.52			8.96			
	EHL	6.49			1.5			
Evertor	PL*	11.0	34.75	26.65	17.3	23.82	19.0	2.11
	PB*	19.61			5.29			
	PT	4.14			1.23			

*Primary muscles within a muscle group. Data are taken from physiologically intact muscles of cadavers (Yamaguchi et al. 1990). †Cadaver data used for the YH and AC group were from a 37-yr-old male and a 63-yr-old female, respectively (Friedrich and Brand, 1990). PCA, physiological cross-sectional area; SPCA, summed physiological cross-sectional area; W-SPCA, weighted summed physiological cross-sectional area; TA, tibialis anterior; EHL, extensor hallucis longus; EDL, extensor digitorum longus; GAS-L, gastrocnemius (lateral); GAS-M, gastrocnemius (medial); SOL, soleus; TP, tibialis posterior; FHL, flexor hallucis longus; PL, peroneus longus; PB, peroneus brevis; PT, peroneus tertius. ‡Value expressed as mean; not available separately for the 2 groups. See DISCUSSION for details on model.

both planes using weighted PCAs according to the individual muscles parameters[7]. Our findings were *1*) as predicted, the SPCA of plantarflexors in a group (YH: 89.3 cm^2, weighted: 66.5 cm^2, AC: 83.36 cm^2, weighted: 64.03 cm^2) was greater than that of dorsiflexors in the corresponding group (YH: 30.83 cm^2, weighted: 22.66 cm^2, AC: 21.31 cm^2, weighted: 16.30 cm^2). Similarly, the SPCA of invertors (YH: 68.16 cm^2, weighted: 51.77 cm^2, AC: 42.05 cm^2, weighted: 31.8 cm^2) was greater than that of evertors (YH: 34.75 cm^2, weighted: 26.65 cm^2, AC: 23.82 cm^2, weighted: 19.0 cm^2). *2*) The mean moment arm of plantarflexors (4.12 cm) is 79% of that of dorsiflexors (5.15 cm), and the mean moment arm of invertors (1.92 cm) is 91% of that of evertors (2.11 cm). *3*) In healthy young subjects, the ratio of plantar-to-dorsiflexor and invertor-to-evertor SPCA with moment arm correction was 1.85 (weighted: 1.87) and 1.62 (weighted: 1.60), respectively. In age-matched controls, these values were 2.5 (weighted: 2.5) and 1.46 (weighted: 1.38), respectively. As predicted by our model, for the YH group these values are of the order of the mean value of dorsi-to-plantarflexion and eversion-to-inversion passive stiffness ratios of 1.66 and 1.43, respectively. Similarly, for the AC group, the muscle SPCA ratios compared favorably to the stiffness ratios: the mean dorsi-to-plantarflexion and eversion-to-inversion passive stiffness ratios were 2.89 and 1.37, respectively. In these calculations, we assumed that the muscle-moment arm was invariant to passive stretch (Hicks et al. 2006). These calculations lend credence to the idea that antagonist muscle SPCA, lengthened during passive stretch in a given direction, as well as the mean moment arm of the muscle group as a whole may be directly related to passive stiffness in that direction and can, therefore, explain the direction dependence of passive stiffness.

[7] Weight for the physiological cross-sectional area (PCA) of the i^{th} muscle in a group was obtained by normalizing to the largest PCA in that group i.e., $w_i = A_i/A_{max}$.

Abnormal Joint Stiffness May Be a Result of Altered Muscle Physiology

Our measurements show that passive ankle stiffness is altered in stroke survivors but occurs only in three out of the four directions of movement tested. Specifically, it is higher in dorsiflexion and inversion, lower in eversion, and unchanged in plantarflexion. Many studies have reported changes in the apparent mechanical properties of the ankle in stroke survivors, e.g., an increase in passive dorsiflexion stiffness (Rydahl and Brouwer 2004; Lamontagne et al. 2000). Sinkjær and Magnussen also reported enhancement in passive stiffness of the ankle joint in stroke subjects (Sinkjær and Magnussen 1994). Galiana and colleagues, however, found no significant differences in the ankle passive stiffness in stroke subjects (Galiana et al. 2005). This could have been due to the selected population of subacute strokes and not chronic strokes as in our study. In fact, changes in passive stiffness stroke patients have also been observed in other joints, e.g., wrist (Formica et al. 2008), as well in individuals afflicted with neurological impairments beside hemiparetic stroke, e.g., spinal cord injury (Mirbagheri et al. 2001), cerebral palsy (Lieber and Fridén 2002), and peripheral neuropathy (Salsich et al. 2000). The apparent consensus among these studies appears to be that of cause and effect in that a neurological impairment leads to abnormal alterations of muscle properties.

The causes for hypertonus, i.e., an abnormal resistance to passive movement, at the disabled ankle are debatable. Some studies have indicated that hypertonus is attributable to hyperactivity of the segmental stretch reflex arc (Mirbagheri et al. 2001; Meinders et al. 1996). Other studies have attributed the abnormal muscle tone to changes in the nonreflex mechanics, e.g., passive stiffness for stroke patients (Sinkjær and Magnussen 1994; Lorentzen et al. 2010), for multiple sclerosis patients (Sinkjær et al. 1993; Toft et al. 1993), for cerebral palsy patients (Toft et al. 1993), and for spinal cord injury and multiple sclerosis patients (Lehmann et al. 1989; Lorentzen et al. 2010). The absence of voluntary muscle contraction and reflex activity during

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stretch as confirmed here suggests that hypertonus is directly reflected in the altered passive stiffness.

What could be causing the abnormal passive dorsiflexion stiffness in the paretic ankle? We believe that one possible reason for this could be the hypertonicity of plantarflexors, a frequent complication of stroke (Rydahl and Brouwer 2004; Bazarian et al. 2005). If so, this would result in higher resistance to passive muscle stretch in dorsiflexion, leading, in turn, to higher passive stiffness in that direction of movement. On the other hand, it is also possible that lesion-induced alterations in intrinsic muscle properties (Booth et al. 2001; Lieber et al. 2004; Marbini et al. 2002; Romanini et al. 1989; Tabary et al. 1976; Trotter and Purslow 1992) and/or architecture of the plantarflexors cause the abnormal increase in passive ankle stiffness in dorsiflexion. Mirbagheri and colleagues, who also report an increase of passive stiffness at the elbow joint, proposed that the increase in passive stiffness may be attributable to a shift of the passive length-tension curve to the left, caused, in turn, by alterations in structural cellular and mechanical properties of spastic muscles and of connective tissues (Mirbagheri et al. 2007).

On the basis of the above ideas, according to our physiological model, an increase in the SPCA of plantarflexors relative to that of dorsiflexors, for example, would result in higher dorsiflexion stiffness. Although we do not have data from muscle physiology to confirm whether this is indeed true, other studies provide some indications to support this view. For instance, studies have shown that the abnormally high passive ankle stiffness in stroke survivors and cerebral palsy patients can be likely attributed to alterations in connective tissue and the accumulation of collagen-increasing fibrosis within the high tone muscle (Booth et al. 2001; Dietz and Berger 1983; Jozsa et al. 1990). Mirbagheri and colleagues (Mirbagheri et al. 2007) also attributed joint immobility and prolonged muscle shortening to muscle atrophy causing a decrease in the number of muscle fibers as well as an increase in the ratio of collagen to muscle fiber tissue, reducing the muscle compliance (Lieber et al. 2004; Gracies et al. 1997; O'Dwyer et al. 1996). Changes in muscle (or muscle group) PCA can also arise from muscular abnormalities such as alterations in fiber type distribution. Case in point, Pontén and colleagues, who studied morphological properties of skeletal muscle in wrist flexors and extensors of children with cerebral palsy, found that there was a significantly greater percentage of type II-b fibers in flexors compared with extensors but no significant difference in fiber type percentages for the type I and type II-a fiber types (Pontén et al. 2005). This results in a higher PCA of flexors (vs. extensors), leading to increased passive stiffness in extension (Fridén and Lieber 2003). In fact, lesion-induced alterations in the constitutive properties are not limited to ankle muscles alone. A study by Macko and colleagues demonstrated lesion-induced changes in cellular structure, i.e., a significant increase in the proportion of fast heavy-chain fibers in the vastus lateralis, a primary extensor of the knee (Macko et al. 2005). This, in turn, results in an increase in the ratio of fast-to-slow twitch fibers, and, because the former type of fiber (type II-a or II-b) has a larger diameter, it may lead to an increase in the PCA of the muscle. Similarly, isolated single fiber segments taken from spastic wrist muscles (flexor carpi ulnaris) in patients with cerebral palsy were reported to have increased elastic modulus, possibly attributable to a dramatic remodeling of intracellular or extracellular muscle structural components such as titin and collagen (Fridén and Lieber 2003).

The stiffness trends of the paretic ankle in the inversion-eversion can be qualitatively explained by the synergistic role muscles have between dorsi-plantarflexion and inversion-eversion. The primary evertor muscles, peroneus brevis and peroneus longus, are also plantarflexors of the ankle. Because stroke survivors have hypertonic plantarflexors, we should expect increased resistance to passive stretch in inversion. Likewise, the majority of the ankle invertor muscles (tibialis anterior and extensor hallucis longus) have a synergistic role as ankle dorsiflexors (except for tibialis posterior, which plantarflexes the ankle). Weakness in the ankle dorsiflexors of stroke survivors can, therefore, lead to lower passive stiffness (i.e., higher joint compliance) in eversion. Another possibility is that stroke survivors develop ankle eversion at rest, a mechanism analogous to drop foot, and this behavior contributes to the increased inversion stiffness. However, the rest position of stroke subjects in the frontal plane was found to be negligible ($0.001 \pm 0.0024°$), indicating that the paretic ankle was at anatomical neutral when at rest.

Abnormal Joint Stiffness May Also be a Consequence Of Distorted Neurophysiology

An alternative explanation for our measurements showing that passive ankle stiffness is altered in stroke survivors in three out of the four directions of movement tested might the neural factors, specifically, abnormal muscle activation and/or abnormal neural feedback.

In healthy subjects when the muscles are relaxed, the force-length relation for each individual muscle resembles a spring (albeit possibly nonlinear), and any arbitrary connection of spring-like muscles to the skeleton will yield a spring-like joint behavior (again possibly nonlinear). However, neural feedback (e.g., from spindles and Golgi tendon organs) may also contribute to static ankle mechanical impedance. If it does, then intermuscular feedback between muscles that act on different DOFs may introduce a deviation from spring-like behavior (Hogan 1985). If we represent the measured multivariable torque-angle relation via a vector field, then we can assess the directional variation of ankle mechanical impedance, the extent to which the ankle behaves as a spring, and evidence of uniquely neural contributions. A representative decomposition of the vector field into a conservative field (with zero curl) and a rotational field (with zero divergence) allows us to determine whether the ankle behaves as a spring.

Experiments with healthy subjects quantified the behavior of the maximally relaxed human ankle, showing that ankle mechanical impedance is spring-like but strongly direction dependent. However, when cocontracted muscle activation is not spring-like, it includes nonzero rotational components (Lee et al. 2010). If the nonconservative curl is zero while intermuscular feedback is nonzero, then the feedback gains must be exactly balanced; a dorsiplantar flexion torque evoked by an inversion-eversion displacement must be identical to the inversion-eversion torque evoked by a comparable dorsiplantar flexion displacement. Conversely, with "constant" muscle activity, nonzero curl can only be due to unbalanced intermuscular feedback. Whether the observed nonzero curl was exclusively attributable to unbalanced feedback

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or to a failure to maintain constant muscle activity is presently under investigation.

Quantifying the nonspring-like (rotational) components of static ankle mechanical impedance is important because they are fundamentally nonpassive: unlike the conservative (spring-like) components, under certain conditions, the rotational components may function as an energy source. Consequently, they may affect the stability of the musculoskeletal system during posture and locomotion. This may be especially important in persons with neurological injury (e.g., stroke or spinal cord injury). The prevalence of abnormal muscle tone and spasticity indicates that the peripheral neural networks may be disordered following neurological injury. This will affect the static ankle mechanical impedance, and, if intermuscular feedback is unbalanced, it will manifest as a nonzero rotational component.

Even if the reason for the observed nonconservative curl is not attributable to unbalanced feedback, but the failure to maintain constant muscle activity, one can speculate that the abnormal joint stiffness may still be a result of altered neurophysiology. There is evidence that tibialis anterior normally has stronger or more direct cortical projections than gastrocnemius or soleus. We speculate that the stroke may damage or weaken the usual projections to these muscles, which, in turn, may cause abnormal stiffness at the paretic ankle stiffness (Capaday et al. 1999; Schubert et al. 1997; Christensen et al. 2001; Perez et al. 2004).

Clinical Measures Do Not Necessarily Reflect Changes in Passive Ankle Properties Poststroke

The MAS is the most widely used scale for assessing muscle tone in clinical practice and research. However, there exists controversy with regard to the properties being measured by the MAS. We did not find significant correlation between MAS scores and passive stiffness of the paretic ankle in either direction of sagittal movement. It is, therefore, likely that the clinical i.e., MAS, and quantitative tests are measuring somewhat different features of joint stiffness. This finding suggests that clinical measures such as the MAS, though widely accepted by the clinical community, are "context-specific," i.e., they may not necessarily reflect the alterations in the passive properties of (spastic or nonspastic) hypertonic ankles. Moreover, it has been reported that the psychometric properties of the MAS are poor (Rydahl and Brower 2004), and, given their ordinal nature, MAS scores tend to cluster in the lower ranges, limiting its ability to discriminate between individuals or groups (Allison et al. 1996; Blackburn et al. 2002). Studies have also found that its interrater reliability is good for the upper extremity, e.g., elbow flexors (Bohannon and Smith 1987), but poor for the lower extremity, e.g., ankle plantar flexors (Blackburn et al. 2002; Bierring-Sørensen et al. 2006). These arguments further underline the need to use objective measures such as passive stiffness, in addition to clinical measures before or during therapy.

Frontal Plane Stiffness May be a Stronger Signature Of Ankle Impairment or Altered Physiology

The stroke group had significantly higher stiffness in dorsiflexion compared with age-matched controls; however, there was some overlap when intersubject variability (± SD) of the two groups was taken into account. For example, there were three subjects in each group (AC and ST) whose passive dorsiflexion stiffness was within the variability of the other group. Furthermore, plantarflexion stiffness was similar between both groups. In the frontal plane, on the other hand, each subject's (from both groups) passive stiffness in both directions was outside the variability of the other group, indicating that frontal, rather than sagittal plane, stiffness is a stronger descriptor of ankle impairment (Fig. 5).

Correlation of Passive Stiffness to Whole Body Measures is Consistent With Inherent Human Anatomy

Our results showed that, in healthy individuals, passive stiffness in the sagittal (but not frontal) plane correlated strongly with whole body measures, specifically body height and the product of body height and mass. We believe this trend to be a result of the inherent human geometry: passive stiffness may partially offset the "negative stiffness"[8] about the ankle because of the inverted-pendulum mechanics of upright posture. That particular stiffness should be the product of body mass, acceleration due to gravity, and the center-of-mass height. Because acceleration attributable to gravity is a constant and the center-of-mass height is (roughly) a constant fraction of body height, we can expect the passive stiffness to strongly correlate with the product of body height and weight. In addition, human anatomy is such that our legs are side-to-side (not front-to-back) so we can expect that relationship to be more pronounced in the sagittal plane (dorsi-plantarflexion) but less so in the frontal plane (eversion-inversion).

Coupling Between the Sagittal and Frontal Plane Passive Stiffnesses is Relatively Weak

When a ramp-and-hold perturbation was imposed to one DOF (e.g., plantarflexion), the ankle was free to move in the other DOF (e.g., inversion). This could be due to slight difference in rest lengths of the actuators and/or the inherent biomechanical synergy between movements in the two planes. The resultant coupling, i.e., the linear mapping between torques in one DOF resulting in angular displacements in the other DOF, was computed between the DOFs for each group (Table 4). In the stiffness (or compliance) tensor, this coupling was represented by its off-diagonal terms. We found that the magnitude of coupling at rest, as characterized by the inverse of joint stiffness (or compliance), was relatively "weak" (a fraction of uncoupled stiffnesses on the tensor diagonal) for all three groups. Of particular importance, however, was the relatively "strong" coupling between dorsiflexion-inversion or vice versa (vs. dorsiflexion-eversion or vice versa) and plantarflexion-eversion or vice versa (vs. plantarflexion-inversion or vice versa), within the stroke group (see Table 4). This appears to be consistent with our qualitative explanation provided earlier for the frontal-plane passive-stiffness trends seen in these individuals. For example, given the synergistic role ankle evertors (or invertors) play as plantarflexors (or dorsiflexors), we expected an appreciable coupling between inversion (or eversion) and dorsiflexion (or plantarflexion), and this is indeed true at the disabled ankle. The interpretation of coupling and its potential relation to neurological impairment

[8] In the absence of damping, the equation of motion for an unforced inverted pendulum system with stiffness k and inertia I, is given by: $I \cdot d^2\theta/dt^2 = -(-k \cdot \theta)$, where θ is angle from vertical, and therefore $(-k)$ can be considered to be "negative stiffness".

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Table 4. *Magnitude of coupling between degrees of freedom*

Degree of Freedom		Sagittal		Frontal	
		Dorsiflexion	Plantarflexion	Eversion	Inversion
YH					
Sagittal	Dorsiflexion	**1.90***	n/a	0.13†	0.13
	Plantarflexion	n/a	**3.15***	0.44	0.35
Frontal	Eversion	0.46	0.34	**2.02***	n/a
	Inversion	0.52	5.6×10^{-4}	n/a	**2.9***
ST					
Sagittal	Dorsiflexion	**1.19***	n/a	0.01	0.18
	Plantarflexion	n/a	**3.71***	0.31	0.15
Frontal	Eversion	6.6×10^{-4}	0.3	**2.84***	n/a
	Inversion	0.56	6.2×10^{-4}	n/a	**1.78***
AC					
Sagittal	Dorsiflexion	**1.51***	n/a	0.08	0.24
	Plantarflexion	n/a	**4.39***	0.82	0.31
Frontal	Eversion	0.62	0.78	**2.12***	n/a
	Inversion	0.82	0.72	n/a	**2.92***

†Values are means of coupled passive ankle compliance (in °/N·m) for each group and direction of movement (off-diagonal), $n = 10$/group. *Uncoupled compliance values (diagonal), marked bold. See DISCUSSION for details.

requires further investigation. Future studies would fully characterize and interpret this property by perturbing the ankle in more directions.

Comparison with Other Studies

How do our stiffness estimates compare with those previously obtained by others under similar experimental conditions? Although many studies report passive ankle-sagittal-plane-stiffness measurements in healthy older adults to afford comparison against their neurologically impaired test subjects (e.g., Rydahl and Brouwer 2004; Lamontagne et al. 1997; Chung et al. 2004; Lorentzen et al. 2010), few studies have reported data on young healthy individuals. Moreover, estimates of passive ankle stiffness vary greatly between studies because of differences in experimental conditions: *1*) Posture, e.g., upright stance vs. supine, flexed vs. extended knee (Riemann et al. 2001; Saripalli and Wilson 2005; Morasso and Sanguineti 2002), *2*) perturbation type, e.g., active vs. passive, magnitude and waveform (Riemann et al. 2001; Morasso and Sanguineti 2002; Kearney et al. 1997; Casadio et al. 2005), *3*) physiological conditions, e.g., elicitation of maximal muscle contraction or joint ROM vs. submaximal activation conditions (Riemann et al. 2001; Morasso and Sanguineti 2002; Rao 2006), *4*) population characteristics, e.g., age (Lark et al. 2003), sex (Padua et al. 2005; Cammarata et al. 2007; Lephart et al. 2002; Granata et al. 2002), *5*) time postlesion (Mirbagheri et al. 2007), and *6*) location of lesion, e.g., cortical vs. subcortical, have all been shown to be important determinants of passive ankle stiffness. We sought to, therefore, compare our findings specifically to (Tables 5 and 6) the following: *1*) Sinkjær, who measured the intrinsic and reflex components of total stiffness in ankle dorsiflexors (Sinkjær et al. 1988) in young healthy individuals; *2*) Rydahl and Brouwer, who estimated the passive ankle stiffness in stroke survivors and age-matched controls using a series of displacement perturbations in dorsiflexion (Rydahl and Brouwer 2004); *3*) Lamontagne, who studied the contribution of passive stiffness to ankle plantarflexor moment during gait in young healthy subjects (Lamontagne et al. 1997); *4*) Chung, who investigated biomechanical changes in the passive properties (e.g., quasistatic stiffness in dorsiflexion and plantarflexion) in hemiplegic spastic ankles and age-matched controls (Chung et al. 2004); *5*) Harlaar, who measured passive stiffness characteristics of hemiplegic ankle plantarflexors in subjects with a wide age range (Harlaar et al. 2000); *6*) Singer, who evaluated passive lengthening of triceps surae muscles in patients with acquired brain injury (Singer et al. 2002); and *7*) Lorentzen, who measured active and passive components of ankle plantar flexor stiffness in subjects with stroke, multiple sclerosis, spinal cord injury as well as healthy individuals (Lorentzen et al. 2010).

Our ability to compare stiffness measurements in the frontal plane, however, is greatly limited due to very few studies available on the subject. To the best of our knowledge, only two studies have measured passive ankle stiffness in the frontal plane: *1*) Saripalli and Wilson, who examined dynamic ankle stiffness and dynamic inversion stabilization as a function of ankle inversion and eversion (Saripalli and Wilson 2005), and *2*) Zinder and colleagues, who tested the validity and reliability of a new measure of inversion-eversion ankle stiffness using a novel medial/lateral swaying cradle device (Zinder et al. 2007). Both those studies used healthy young subjects in an upright stance (Table 5). We did not find any published work that has examined inversion-eversion passive ankle stiffness in stroke survivors or in older healthy individuals.

Overall, our stiffness estimates compare favorably to published values. Specifically, in dorsiplantar flexion they are *1*) comparable to those obtained in Sinkjær's study ($<$0.06 N·m/° difference); *2*) comparable to those obtained for stroke and age-matched control groups in Rydahl's study ($<$0.09 and 0.05 N·m/° difference); *3*) higher than those obtained for young healthy and stroke subjects in Lamontagne's (1997, 2000) studies. Our stiffness estimates were nearly twice those reported by Lamontagne et al. (2000) and Lorentzen et al. (2010) for stroke survivors (Table 6), but these differences may be due to the fact that participants in Lamontagne's (0.2 ± 0.07 yr) and Lorentzen's (0.85 ± 0.72 yr) studies were in the subacute and acute stages of stroke as opposed to chronic stroke participants in our study (3.3 ± 2.2 yr); *4*) higher than those obtained for healthy individuals in Chung's study and those of others

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Table 5. *Comparison of Anklebot protocol with similar previous paradigms*

Work	Posture	Mechanism/Apparatus	Perturbation Characteristics: Sagittal (Groups tested)	Perturbation Characteristics: Frontal (Group)
Anklebot	Seated, knee flexed	**Passive robot forces**	**20° PF – 30° DF 30° PF – PROM° DF v = 5°/s (YH, AC, ST)**	**25° Inversion – 20° Eversion v = 5°/s**
Sinkjær et al. (1988)		Passive force plate rotations	2° DF v = 100°/s (YH)	N/A
Rydahl and Brower (2004)			5° DF v = 100°/s (ST, AC)	N/A
Lamontagne et al. (2000)		Kin-Com dynamometer	30° PF – PROM + 5° DF v = 12–37°/s (YH, ST)	N/A
Chung et al. (2004)		Custom-built stretching device	PROM in both DF and PF, Torque limit =10 N-m (DF, PF) v = variable (fixed 90-sec trials) (ST, AC/YH)	N/A
Lorentzen et al. (2010)		Motor-driven footplate	6° DF v = 100°/s (ST, AC/YH*, MS, SCI)	N/A
Harlaar et al. (2000)	Both seated (knee flexed) and supine (knee extended)	Customized dynamometer driven manually	PROM or < preset torque level (12 N-m plantar, 8 N-m dorsi) whichever occurs first v = variable (fixed 5-sec trial) (ST, AC/YH*)	N/A
Singer et al. (2002)	Supine (knee flexed at 90°)	Computer-controlled ankle dynamometer	20° PF – 5° DF v = 5°/s (ST, AC/YH*)	N/A
Zinder et al. (2007)	Upright stance, 100% WB	Custom-built swaying cradle device	N/A	5° Inversion – 5° Eversion (YH)
Saripalli and Wilson (2005)	Upright stance, Variable WB (30%–70%)	Cradle platform and force plate		10° Inversion – 10° Eversion (YH)

Age of healthy subjects in Chung's, Harlaar's, Singer's, and Lorentzen's studies were 42.1 ± 20.5 yr, 24 ± 66 yr, 22 ± 50 yr, and 46.3 ± 15.8 yr, respectively, significantly different or outside of the mean age or mean ± SD age of subjects in YH or AC groups in this study (see METHODS for details). DF, dorsiflexion; PF, plantarflexion; MS, multiple sclerosis; SCI, spinal cord injury; v, perturbation velocity; WB: weight bearing. N/A refers to where measurements were not made in that DOF.

(Singer et al. 2002; Harlaar et al. 2000); and *5*) comparable to those obtained for healthy subjects by Lorentzen (<0.08 N·m/° difference).

We found that any differences between stiffness estimates obtained in this study and those reported previously can be attributed to differences in sample age, time since onset of injury (chronic, acute, and subacute), characteristics of perturbations (e.g., stretch velocity, amplitude, and range/ROMs that may or may not include the neutral), and posture (e.g., seated or not, knee flexed or not, and at what angle). For instance, Chung reported lower passive quasistiffness in plantar flexion than that reported here, possibly because they measured stiffness in a much wider ROM (46.01 ± 9.65°) than used for our study (16.16 ± 1.08°). Another reason could be the difference in average sample age, e.g., 42.1 yr in Chung's study vs. 59 yr in this study for healthy age-matched control subjects. In Harlaar's study, the age of subjects was between 24 and 66 yr and thus included younger subjects compared with our study. Furthermore, "control" data in that study were taken from the nonparetic ankle, whereas we used healthy age-matched controls. We also observed differences in time postlesion, e.g., 0.27 yr, 0.2 yr, and 0.85 yr in Singer's, Lamontagne's, and Lorentzen's studies as opposed to 3.3 yr in this study, as well as testing conditions, e.g., supine posture with knee flexed at 90° in Singer's study as opposed to seated position with knee flexed at 45° in this study. Despite these differences, overall our sagittal-plane stiffness estimates for the hemiparetic ankle matched very favorably against those reported by Chung (<0.03 and 0.12 N·m/° difference in plantarflexion and at 10° dorsiflexion, respectively) and Singer (<0.13 N·m/° at 10° dorsiflexion). The comparisons were slightly less favorable to estimates obtained by Harlaar (0.44 N·m/° at 10° dorsiflexion) and Lorentzen (0.43 N·m/° at 6° dorsiflexion), possibly because of differences in mean sample age, time poststroke, knee flexion angle in the case of Lorentzen's study, and the large variability associated with manual stretch in the case of Harlaar's study.

Our frontal-plane stiffness estimates for young healthy individuals were lower than those obtained by Saripalli or Zinder. We speculate that this might be attributable to the differences in postural and/or loading conditions under which passive stiffness was measured. To illustrate, in Zinder's study, passive stiffness was measured with subjects in upright stance with full weight bearing condition, which could have led to a

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Table 6. *Comparison of Anklebot stiffness estimates with published values*

Criterion for Comparison	Published Study	YH	ST	AC
		Sagittal Plane		
Torque (τ) <5 N-m (DF only)	Sinkjær et al. (1988)	0.6	N/A	N/A
	Anklebot	**0.54 ± 0.07**[a,b]	**0.84 ± 0.56**	**0.87 ± 0.24**
Perturbation = 5° (DF only)	Rydahl and Brower (2004)	N/A	1.1 ± 0.4	0.8 ± 0.4
	Lorentzen et al. (2010)*,[c]	0.38 ± 0.03	0.43 ± 0.03	N/A
	Anklebot	**0.46 ± 0.14**	**1.01 ± 0.72**	**0.85 ± 0.28**
Full DF range	Lamontange, 2000	0.3	0.43	N/A
	Anklebot	**0.52 ± 0.31**	**0.83 ± 0.20**	**0.65 ± 0.26**
At 10° DF	Chung, 2004*	N/A	0.54 ± 0.19	0.35 ± 0.20
	Singer, 2002*		0.53 ± 0.36	0.44 ± 0.21
	Harlaar, 2000*		0.44 ± 0.36 (0.31–0.61)	0.36 ± 0.22 (0.32–0.47)†
	Anklebot	**0.63 ± 0.15**	**0.66 ± 0.20**	**0.58 ± 0.17**
Full range of movement (PF only)	Chung, 2004	N/A	0.29 ± 0.18	0.13 ± 0.13
	Anklebot	**0.31 ± 0.12**	**0.26 ± 0.06**	**0.22 ± 0.01**
At 30° PF	Chung, 2004	N/A	0.20 ± 0.20	0.11 ± 0.08
	Anklebot	OUTP	**0.29 ± 0.03**	OUTP
		Frontal Plane		
INV perturbations	Saripalli, 2002	~0.35–0.43‡	N/A	N/A
	Anklebot*	**0.34 ± 0.05**	**0.56 ± 0.12**	**0.34 ± 0.05**
Full range of movement§	Zinder, 2007	0.62 ± 0.16	N/A	N/A
	Anklebot	**0.41 ± 0.09**	**0.34 ± 0.09**	**0.43 ± 0.04**

[a]Values are group means ± SD of passive ankle stiffness (in °/N·m) in each study, where available.[b]For comparison purposes, values obtained using the Anklebot are marked in bold.[c]Value reported for 6° DF perturbation at 8°/s stretch velocity. *Average age (or range) of healthy subjects in Chung's, Harlaar's, Singer's, and Lorentzen's studies were 42.1 ± 20.5 yr, 24 ± 66 yr, 22 ± 50 yr, and 46.3 ± 15.8 yr, respectively; stroke subjects in Harlaar's and Lorentzen's studies were 42.1 ± 20.5 yr and 55.2 ± 11 yr; significantly different or outside of the mean age or mean ± SD age of subjects in YH, AC, or ST groups in this study except one stroke participant in Lorentzen's study (36 yr) was within mean ± SD age of subjects in YH, AC, or ST groups in this study (see METHODS). †Ankle of the unaffected limb taken as control. ‡Approximate extrapolated range for 0% loading. §Single linear regressor was used for the entire range of frontal plane movement. N/A refers to where a group was not tested in that study. OUTP refers to outside the range of commanded perturbations in a given direction of movement.

much higher value. In Saripalli's study, it was measured under upright stance with varying levels of body weight loading (30%–70%). They showed that passive stiffness increases with loading of the joint, and, when extrapolated for 0% loading, their values are within the range of our inversion stiffness estimates (<0.01 N·m/° difference).

Clinical Implications

A potential signature of ankle pathology? Our findings demonstrate that passive ankle stiffness is significantly different between healthy (both young and older) and stroke populations, both in the sagittal as well as frontal planes. In fact, even with the intersubject variability included, the differences between the two populations are obvious (more so in the frontal plane). This motivated us to develop a biomechanical metric that is simple yet is able to differentiate between healthy and stroke populations, the logarithm of the ratio of passive stiffness in opposite directions in a given DOF.

$$\alpha = \log[k(\theta_+)/k(\theta_-)] \quad (4)$$

where α is the metric and $k(\theta_+)$ and $k(\theta_-)$ are as defined earlier (see *Eq. 3*). We found that in the sagittal plane this ratio is able to broadly separate stroke survivors (3.27 ± 1.12) from both young healthy (1.72 ± 0.37, $P = 0.05$) as well as age-matched controls (2.64 ± 0.34, $P = 0.05$). The differences in this metric were even more pronounced in the frontal plane in which a "magnitude reversal" occurred with respect to the direction of movement between the stroke (0.69 ± 0.24) and healthy groups (YH: 1.43 ± 0.35, $P = 0.006$; AC: 1.41 ± 0.32, $P = 0.001$). An added advantage is that, per our physiological model, this ratio is related to an intrinsic muscle property, i.e., the summed PCA. We believe, therefore, that this metric (or functions thereof) is a useful biomechanical (population) marker (Fig. 6), possesses physiological relevance, and can be computed in a

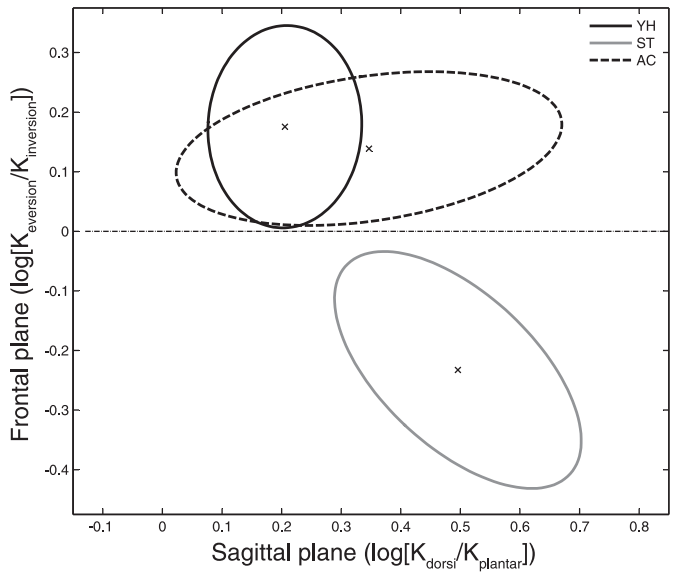


Fig. 6. Logarithm of the stiffness ratio in the frontal vs. sagittal plane for all 3 groups (YH, ST, and AC). The figure shows minimum-area, 99%-confidence ellipses (with corresponding centers of the data set ×) in the frontal-sagittal plane for each group. Notice the overlap between the 3 groups in the sagittal plane but a null intersection between healthy (YH, AC) and stroke groups (ST) in the frontal plane.

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clinical setting, and, most importantly, its individual components provide us with information about passive properties of the ankle joint. Because many studies have shown that passive properties of a joint are altered following an onset of neurological impairment (Chung et al. 2004), it is quite possible that this metric may be a signature of ankle pathology and may be able to differentiate between populations with neurological impairments besides stroke.

Assessment of motor recovery. In the present practice of therapy, motor performance and recovery following a stroke are generally graded using established clinical scales. For instance, muscle tone is typically assessed by the clinician passively moving a joint or limb and grading the outcome using the Ashworth scale (Ashworth 1964). Most such measures, however, suffer from a fundamental disadvantage in that they are generally subjective in nature. Studies have demonstrated the "limb-preferential" nature of these measures, i.e., they weigh one limb more than the other (e.g., the Fugl-Meyer weighs the arm more than the leg), thereby reducing their usefulness as a clinical assessment tool for the lower extremity. Others suffer from "limb-specificity", i.e., they are reliable only for certain limbs but not others. For example, the reliability of the MAS has been demonstrated only for use in the elbow and wrist (Bohannon and Smith 1987; Gregson et al. 1999) but not the ankle (Biering-Sørensen et al. 2006). Quantifiable measures (e.g., passive joint properties), on the other hand, possess the potential to provide us with objective unbiased information about motor recovery in all limbs. Although both reflex and nonreflex changes in ankles with spastic and nonspastic hypertonia can substantially affect the functional performance of stroke patients (Chung et al. 2004), there is evidence that nonreflex (e.g., passive) changes have more profound and consistent effects than did reflex changes (Dietz and Berger 1983; Dietz et al. 1981; Berger et al. 1984; Ada et al. 1998; Lamontagne et al. 2000). For example, changes in passive biomechanical properties at the paretic ankle have been shown to contribute to the internal ankle joint torque in functional movement (Lamontagne et al. 2000; Siegler et al. 1984; Tardieu et al. 1989). These, and many other studies, all reinforce the fact that there is a greater need to assess passive biomechanical changes in hemiplegic ankles with intervention protocols and their potential relationship to improvements in ankle function. For example, Zhang and colleagues, who estimated the reflex and intrinsic properties of muscles and other soft tissues crossing the ankle joint via small-amplitude random perturbations applied over a range of initial ankle flexion angles, found that stretching of the ankle in stroke patients as part of an intervention program resulted in considerable changes in both joint passive and active ROM and joint stiffness and reflex gain (Zhang et al. 2002). Selles and others found that intelligent stretching of the dorsi- and plantarflexors in stroke survivors led to significant improvements in passive ROM, maximum voluntary contraction, passive stiffness, and comfortable walking speed (Selles et al. 2005). There is also evidence to show that sustained muscle stretch (e.g., constant-angle, cyclic-stretching, or constant-torque protocols) in hemiplegic ankle joints reduced MAS grade, yielded an increase in ankle ROM, and reduced viscoelastic components of dorsiflexors (Yeh et al. 2007).

We have recently conducted a pilot training protocol at the Baltimore VA Medical Center to investigate potential benefits of robotic therapy on ankle motor control and impairment including passive ankle-joint stiffness, as well as gait function in eight distinct stroke survivors. Survivors, at least 6 mo poststroke, received visually evoked seated training in dorsi- and plantarflexion ranges for a period of 6 wk, three times per week for a total of 18 sessions (Forrester et al. 2010). We evaluated these volunteers using clinical instruments, as well as robot-based assessment metrics before intervention and at completion. Findings are very promising in that seated robotic therapy had positive impact on ankle motor control and gait function including increased unassisted floor walking speed and anterior-posterior propulsive impulses during single support stance duration (Forrester et al. 2010). We also observed positive changes on passive ankle-joint stiffness over the 6-wk period that were strongly related to changes in overground gait function reported by Forrester et al. (Forrester et al. 2010). These will be reported in a separate clinical manuscript.

Conclusions

To summarize, we presented findings on the passive ankle stiffness in stroke survivors using a novel ankle robot prototype. The results of this study indicate that passive stiffness is strongly direction-dependent in both planes of movement and that, compared with individuals without known pathology of similar age, individuals with stroke have increased passive ankle stiffness in dorsiflexion and inversion but are more compliant in eversion.

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DISCLOSURES

Drs. H. I. Krebs and N. Hogan are coinventors in the MIT and VA-held patent for the robotic device used in this work. They hold equity positions in Interactive Motion Technologies, the company that manufactures this type of technology under license to MIT and VA.

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