Multivariable Static Ankle Mechanical Impedance With Active Muscles

Hyunglae Lee, *Student Member, IEEE*, Patrick Ho, Mohammad Rastgaar, Hermano Igo Krebs, *Senior Member, IEEE*, and Neville Hogan

***Abstract*—This paper reports quantification of multivariable static ankle mechanical impedance when muscles were active. Repetitive measurements using a highly backdrivable therapeutic robot combined with robust function approximation methods enabled reliable characterization of the nonlinear torque-angle relation at the ankle in two coupled degrees of freedom simultaneously, a combination of dorsiflexion–plantarflexion and inversion–eversion, and how it varied with muscle activation. Measurements on 10 young healthy seated subjects quantified the behavior of the human ankle when muscles were active at 10% of maximum voluntary contraction. Stiffness, a linear approximation to static ankle mechanical impedance, was estimated from the continuous vector field. As with previous measurements when muscles were maximally relaxed, we found that ankle stiffness was highly direction-dependent, being weakest in inversion/eversion. Predominantly activating a single muscle or co-contracting antagonistic muscles significantly increased ankle stiffness in all directions but it increased more in the sagittal plane than in the frontal plane, accentuating the relative weakness of the ankle in the inversion–eversion direction. Remarkably, the observed increase was not consistent with simple superposition of muscle-generated stiffness, which may be due to the contribution of unmonitored deep ankle muscles. Implications for the assessment of neuro-mechanical disorders are discussed.**

***Index Terms*—Ankle joint, ankle joint stiffness, ankle stiffness, human ankle, impedance structure, multivariable impedance, multivariable stiffness, stiffness anisotropy.**

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I. Introduction

CHARACTERIZATION of ankle mechanical impedance is essential to understand how the ankle supports lower-extremity function during physical interaction with the environment, including postural stabilization, propulsion, steering, and energy-absorption during locomotion, and also how it may deviate from the norm as a result of neurological or biomechanical disorders. The mechanical impedance of the human ankle and how it varies with muscle activation has been studied by several researchers. Sinkjaer *et al.* measured the stiffness of ankle dorsiflexors at different voluntary muscle contraction levels [1]. Hunter and Kearney [2] and Weiss *et al.* [3] used stochastic system identification techniques to examine elastic, viscous, and inertial properties of the active ankle joint. Rydahl and Brouwer measured ankle stiffness with active plantarflexors to compare unimpaired subjects and stroke survivors [4]. Other work measured active ankle stiffness during quiet standing [5] and during locomotion [6]. All of the above work identified ankle mechanical impedance in the sagittal plane (the dorsiflexion/plantarflexion (DP) direction). Considerably fewer studies measured active ankle stiffness in the frontal plane (the inversion/eversion (IE) direction) [7]–[9]. All of this prior work characterized ankle mechanical impedance in a single degree of freedom (DOF). Roy *et al.* measured passive ankle stiffness in both DP and IE but did not assess coupling between these degrees of freedom (DOFs) [10], [11].

Yet the ankle is a biomechanically complex joint with multiple DOFs. Although ankle motions are often described about a medial–lateral axis (perpendicular to the sagittal plane) and anterior–posterior axis (perpendicular to the frontal plane), respectively, the anatomical axes of the joint do not intersect, are far from orthogonal, and change with ankle movement [12], [13]. These complexities could introduce a biomechanical coupling between DP and IE. Furthermore, single DOF ankle movements are rare in normal lower extremity actions and the control of multiple ankle DOFs may present unique challenges. Consequently, quantitative characterization of multivariable ankle impedance promises better understanding of the functional role of the ankle and may afford unique insight about its special vulnerabilities.

In a previous work by the authors [14], the multivariable static ankle mechanical impedance of maximally relaxed young unimpaired subjects was measured and represented as a 2-D torque-angle vector field. Although that work provided a baseline for understanding ankle mechanical impedance, normal lower extremity actions typically involve active muscles, either singly, synergistically or antagonistically. In the study reported here the characterization of multivariable static ankle impedance was extended to active muscle conditions. We hypothesized that the

direction of lowest ankle stiffness, IE, when muscles were relaxed [14] would persist when major ankle muscles were active, because they contribute more in the sagittal than frontal plane. Measurements on 10 young healthy subjects verified our hypothesis. Ankle stiffness increased with muscle activation in all movement directions in the IE-DP space. However, stiffness increased least in IE, accentuating the relative weakness of the ankle in that direction. Moreover, the observed increase was less than would be expected from simple superposition of muscle-generated stiffness. This may have important implications for the consequences of neurological and biomechanical disorders.

II. Methods

A. Subjects

The participants in this study were 10 unimpaired young human subjects with no reported history of biomechanical or neuromuscular disorders (seven males, three females; age 19–31; height 1.55–1.80 m; weight 55.8–81.6 kg). The authors obtained approval for this study from MIT's Committee on the Use of Humans as Experimental Subjects (COUHES) and participants gave written informed consent to participate as approved by COUHES.

B. Experimental Setup

In general, ankle mechanical impedance is a dynamic operator that maps a time-history of angular displacement onto a corresponding time-history of torque. Its static component is a relation between angle and torque, possibly nonlinear, which was measured for IE and DP simultaneously using a wearable robot, Anklebot (Interactive Motion Technologies, Inc.), the device used in previous work [14]. The Anklebot provides actively controllable torques in two DOFs, both in DP and IE directions. The third DOF (axial rotation) is passive with extremely low friction, thereby avoiding imposing any inadvertent kinematic constraints on the motion of the ankle. The robot can provide torque at the ankle up to 23 Nm and 15 Nm in the DP and IE directions, respectively. The robot was mounted to a knee brace and end effectors of the robot were connected to a rigid U-shaped bracket attached to a custom designed shoe (Fig. 1). A proportional-plus-derivative controller for joint angle with proportional gain 200 Nm/rad and derivative gain 1 Nm-s/rad guaranteed safe and stable data capture even when muscles were highly activated. Since the robot is highly backdrivable (it has very low intrinsic mechanical impedance), this implementation enabled impedance control. Subjects were seated and the knee brace was securely fastened to a metal plate mounted on the side of the chair to support the weight of the robot and the leg and to ensure reliably repeatable measurements (Fig. 1). In addition, to prevent foot slippage inside the shoe, a proper shoe size was selected for each subject, the foot was tightly fastened with shoe laces, and a wide Velcro strap was secured over the laces.

Electromyographic (EMG) signals were recorded using differential surface electrodes with built-in pre-amplifiers (Delsys Inc.). They were placed on the bellies of major muscles related to ankle movement: tibialis anterior (TA), peroneus longus (PL), soleus (SOL), and medial gastrocnemius (GAS).

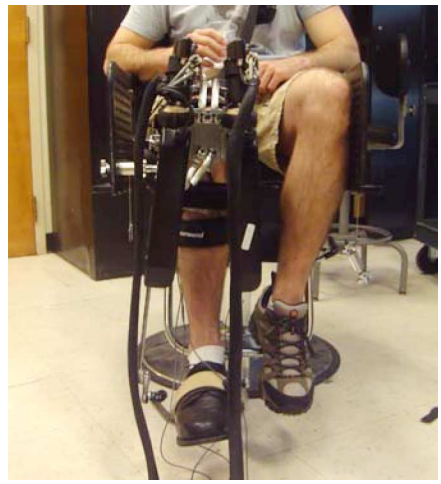

Fig. 1. Subject wearing Anklebot in a seated posture.

EMG signals, band pass filtered between 20 and 450 Hz, were sampled at 1 kHz and their amplitudes were estimated using a root mean square (rms) filter with a moving window of 200 ms after removing any dc component of the signal [15].

C. Experimental Protocol

Before measuring impedance, the maximum voluntary contraction (MVC) of each muscle was measured while subjects stood upright, and the target EMG level was set as 10% of MVC. We set the target to a constant level of muscle activity, rather than constant torque, to better understand the relative contributions of intrinsic muscle stiffness and nonlinear kinematic effects which may masquerade as stiffness [16], [17].

Subjects were seated with their ankle held by the robot in a neutral position with the sole at a right angle to the tibia. As a baseline for active studies, passive ankle impedance was first measured with muscles fully relaxed. For active studies, a graphical user interface was provided to subjects, showing real-time EMG amplitudes of TA and SOL along with the corresponding target levels (10% MVC) subjects were to maintain. EMG amplitudes were estimated as described above and normalized to the MVC level. Subjects were first instructed to activate a specific muscle and maintain it at the target level. When the activation level reached the target level, the robot applied terminated ramp perturbations to the ankle with a slow velocity $(10^\circ/\mathrm{s})$ selected to maintain quasi-static conditions (no contribution of inertia and minimal effect of viscosity) and minimize the involvement of spindle mediated stretch reflexes [18]. During measurements, subjects were instructed not to resist robot perturbations voluntarily. Three types of active study were performed: in two of them subjects were instructed to activate only a single muscle (TA or SOL) against a resisting torque exerted by the robot. In the third, subjects were instructed to co-contract both TA and SOL while maintaining each at a level comparable to that when nominally acting alone. To minimize any possible effects due to inconstant muscle activation, four repetitive measurements were performed for each study. Each measurement consisted of a total of 24 movements along 12 equally-spaced directions in IE-DP space, once

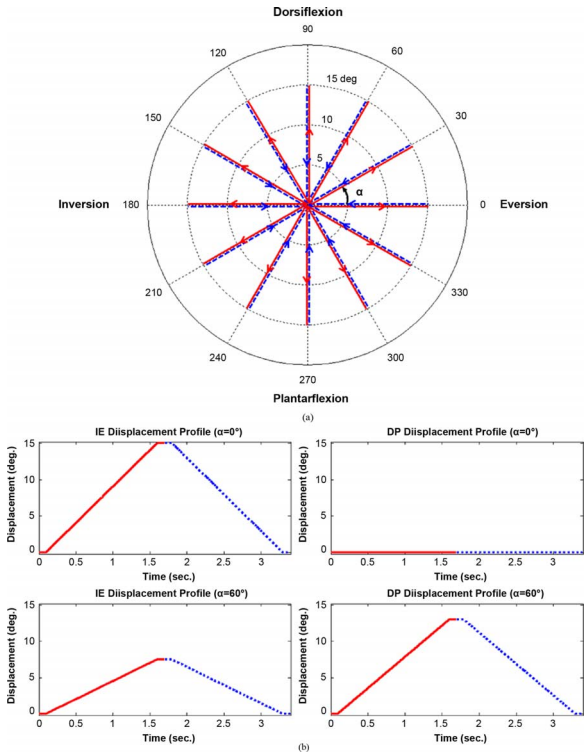

Fig. 2. (a) Displacement profile in IE-DP space: Perturbations began with the eversion direction (0°), rotated by 30° on each subsequent perturbation in the IE–DP space and ended at 330°. For example, $\alpha = 60°$ corresponded to a combination of dorsiflexion and eversion with more dorsiflexion than eversion, and $\alpha = 90°$ corresponded to pure dorsiflexion. Solid red lines denote outbound movements, and dotted blue lines represent inbound movements. (b) IE and DP displacement profiles: Example displacement profiles when $\alpha = 0°$ and $\alpha = 60°$ were presented. The x-axis and y-axis denote time in seconds and angular displacement in degrees, respectively. Color codes are the same as (a).

outbound and once inbound per direction [Fig. 2(a)]. Ramp perturbations were applied, beginning at 0° in polar coordinates, increasing by 30° and ending at 330° with 0°, 90°, 180°, and 270° corresponding to eversion, dorsiflexion, inversion, and plantarflexion, respectively. The robot moved the ankle along a commanded trajectory with a nominal displacement amplitude of 15° to cover the normal range of motion of the ankle, and held the foot for 0.1 s at the starting and ending positions. Examples of IE and DP displacement profiles are presented in [Fig. 2(b)]. The applied torque and actual angular displacement in both DOFs (IE and DP) were recorded at 200 Hz. To avoid fatigue, a 3 min rest period was given between measurements.

D. Analysis Methods

The measured multivariable torque-angle relation, possibly nonlinear, can be represented as a vector field (V)

$$(\tau_{IE}, \tau_{DP}) = V(\theta_{IE}, \theta_{DP}) \tag{1}$$

where θ_{IE} and θ_{DP} are angular displacements in the IE and DP directions, respectively, and τ_{IE} and τ_{DP} are the corresponding applied torques. We decomposed the vector field approximation (V) problem into two scalar function estimation problems (2), and each of them (ϕ_1 and ϕ_2) was approximated as a surface using a method based on thin plate spline (TPS) smoothing [19] with generalized cross validation (GCV) [20]

$$\begin{aligned} \tau_{IE} &= \phi_1(\theta_{IE}, \theta_{DP}) \\ \tau_{DP} &= \phi_2(\theta_{IE}, \theta_{DP}). \end{aligned} \tag{2}$$

This method enables determination of an optimal compromise between fidelity to the data and smoothness of the surface. The method is sufficiently accurate to eliminate the effect of noise and produce reliable measurements. Details of the method are in [14].

Any torque components required to overcome the friction of the actuators were identified by repetitive measurements as above but without a human subject, and subtracted from the measured torque prior to further analysis. Furthermore, data points around the neutral and target positions were discarded to avoid possible errors due to initial lengthening and shortening of muscle fibers [21], and the remaining data points were used for all data analysis. Four repetitions of measurements in the 24 directions (a total of 96 movements) were approximated as eight separate fields (four outbound and four inbound), and averaged into estimates of two continuous vector fields for outbound and inbound movements, respectively.

To quantify the anisotropy or directional variation of ankle mechanical impedance, the effective ankle stiffness was evaluated from the averaged single vector field for each direction of movement by computing the slope of a least squares linear fit to the displacement and torque data in that direction (data between nominal displacements of 1° to 14° were used for fitting), and the variation of ankle stiffness with direction in IE-DP space was constructed accordingly.

A significance level of 0.05 was used for all statistical analyses. EMG and stiffness data were tested for normality using a Jarque–Bera test (MATLAB's *jbtest* function). Paired t-tests (MATLAB's *ttest2* function) were used to compare normalized EMG levels of single-muscle-active and co-contraction studies, and to investigate changes of the stiffness increase ratio in active studies. In addition, Wilcoxon signed-rank tests were performed to identify changes of directional variation of stiffness with muscle activation, and to compare total muscle activation increase and corresponding stiffness increase in single-muscle-active and co-contraction studies.

III. Results

A. EMG Analysis

The ratio of EMG amplitudes with muscles active to their corresponding MVC levels satisfied the normality condition ($p \gg 0.05$) justifying comparison using paired t-tests. They demonstrated that all 10 subjects could follow instructions (Fig. 3). In general, subjects were able to maintain TA activation around the target level, 10% MVC, quite well: the mean of all subjects was 8.63% (0.17%) for the TA active study, and 9.56% (0.43%) for the co-contraction study. The value in parentheses is the standard error (SE) over all subjects. Activation levels of the SOL were slightly lower than the target level: the mean of all subjects was 8.05% (0.42%) for the SOL active study, and 8.33% (0.28%) for the co-contraction study. In both studies, activation

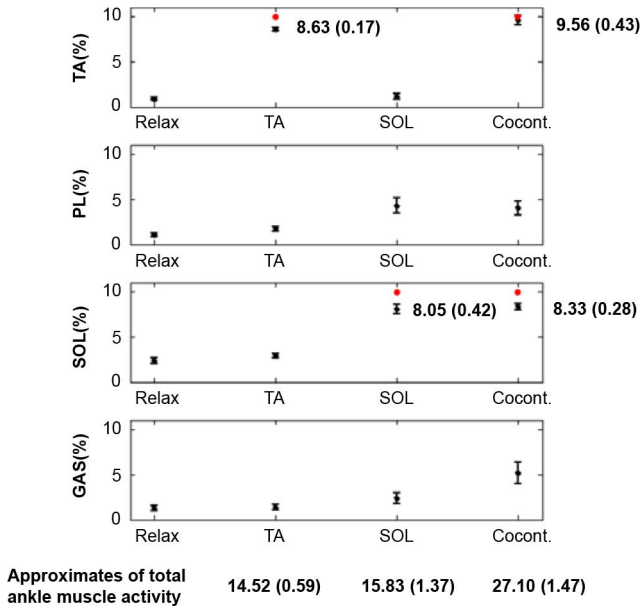

Fig. 3. Ratio of EMG amplitudes with muscles active: The x-axis represents the type of study and the y-axis presents each muscle's activation level as a percentage of the corresponding MVC level. Target muscle activation levels (10% MVC) are depicted as red dots. The mean ± SE of all analyzed subjects are illustrated as an asterisk and bars, respectively. For each study, total ankle muscle activity was approximated by summing normalized EMG amplitude of all measured muscles, and represented at the bottom of the figure.

levels of TA and SOL muscles in the co-contraction study were comparable to those when nominally acting alone ($p > 0.05$).

In addition, total ankle muscle activity was approximated by summing normalized EMG amplitudes of all measured muscles. For the TA and SOL active studies, estimates were 14.52 (0.59) and 15.83 (1.37), respectively. The estimate for the co-contraction study was 27.10 (1.47) (Fig. 3).

B. Reliability of Repetitive Measurements and Field Approximation

One subject (#1) from the SOL active study and another (#6) from the co-contraction study exceeded the torque limit of the hardware. Data exceeding the limit were excluded from subsequent analysis. The mean error between the friction-compensated measurements and surface approximates obtained by the TPS smoothing with GCV was less than 0.02 Nm, which is substantially smaller than the measurement error range, ± 1 Nm [10]. This validates the accuracy of the field approximation.

To investigate the variability of repetitive measurements, the mean and standard deviation (SD) of absolute error between the approximated field from each of four repeated measurements and their average as a single field were calculated for each subject separately, and averaged across all subjects (Table I). The mean and SD for the scalar function ϕ_1 were greater than for the scalar function ϕ_2. In addition, the mean and SD with muscles active were greater than when relaxed. In all study conditions both for outbound and inbound data, the mean error was less than 0.28 and 0.64 Nm for ϕ_1 and ϕ_2, respectively. The SD was even smaller, less than 0.08 and 0.18 Nm for ϕ_1 and ϕ_2, respectively.

TABLE I
VARIABILITY OF REPEATED MEASUREMENTS

Dir.	Study Type	Scalar Function (ϕ_1) Mean of Error	Scalar Function (ϕ_1) SD of Error	Scalar Function (ϕ_2) Mean of Error	Scalar Function (ϕ_2) SD of Error
Out	Relaxed	0.10 (<0.01)	0.02 (<0.01)	0.15 (0.01)	0.03 (0.01)
	TA Active	0.20 (0.03)	0.05 (0.01)	0.51 (0.06)	0.14 (0.03)
	SOL Active	0.25 (0.08)	0.08 (0.04)	0.52 (0.08)	0.17 (0.07)
	Co-cont.	0.25 (0.01)	0.06 (0.01)	0.64 (0.05)	0.18 (0.04)
In	Relaxed	0.11 (<0.01)	0.02 (<0.01)	0.15 (0.01)	0.03 (0.01)
	TA Active	0.20 (0.02)	0.05 (0.01)	0.54 (0.04)	0.12 (0.02)
	SOL Active	0.23 (0.04)	0.06 (0.01)	0.59 (0.07)	0.13 (0.02)
	Co-cont.	0.28 (0.03)	0.06 (0.01)	0.63 (0.04)	0.17 (0.02)

The mean and SD of absolute error in Nm between the field derived from each measurement and the single field averaged from four measurements were calculated for each subject separately. The mean and SE (in parentheses) across all analyzed subjects are presented in the table.

Fig. 4 shows raw data and the resultant single vector field of a representative subject (subject #1) and 2-D slices in four major directions (inversion, eversion, dorsiflexion, and plantarflexion) to demonstrate how well the field fit the measurements and how small the variability of repetitive measurements was.

C. Anisotropy of Static Ankle Mechanical Impedance

For each subject, the directional variation of ankle stiffness was identified. The effective ankle stiffness was calculated in 36 directions in IE-DP space from the averaged vector field. To evaluate the effective resolution of stiffness estimation, we calculated the minimum difference of stiffness values between two adjacent directions among 36 directions for each study and for each subject separately. When averaged across all subjects, the resolution was substantially smaller than the stiffness values in all study conditions, verifying that our approach was sensitive enough to detect small stiffness changes in different movement directions (Table II). Results were represented in a polar plot (Fig. 5), where directions of 0°, 90°, 180°, and 270° represent eversion, dorsiflexion, inversion, and plantarflexion, respectively. Outbound and inbound results were averaged, and the means and SE for all analyzed subjects in all conditions, three active and one relaxed, are presented. The maximally-relaxed stiffness exhibited a "peanut" shape, similar to that previously reported [14]. Absolute stiffness values did not satisfy the normality condition, so comparisons were performed using Wilcoxon signed-rank tests. Predominantly activating a single muscle or co-contracting antagonistic muscles significantly increased ankle stiffness in all directions ($p < 0.05$). However, stiffness increased more in the sagittal plane (DP direction) than in the frontal plane (IE direction) in all active studies, substantially accentuating the "peanut" shape, pinched in the IE direction. The ratio of active stiffness to maximally-relaxed stiffness satisfied the normality condition ($p > 0.05$). Its values in the

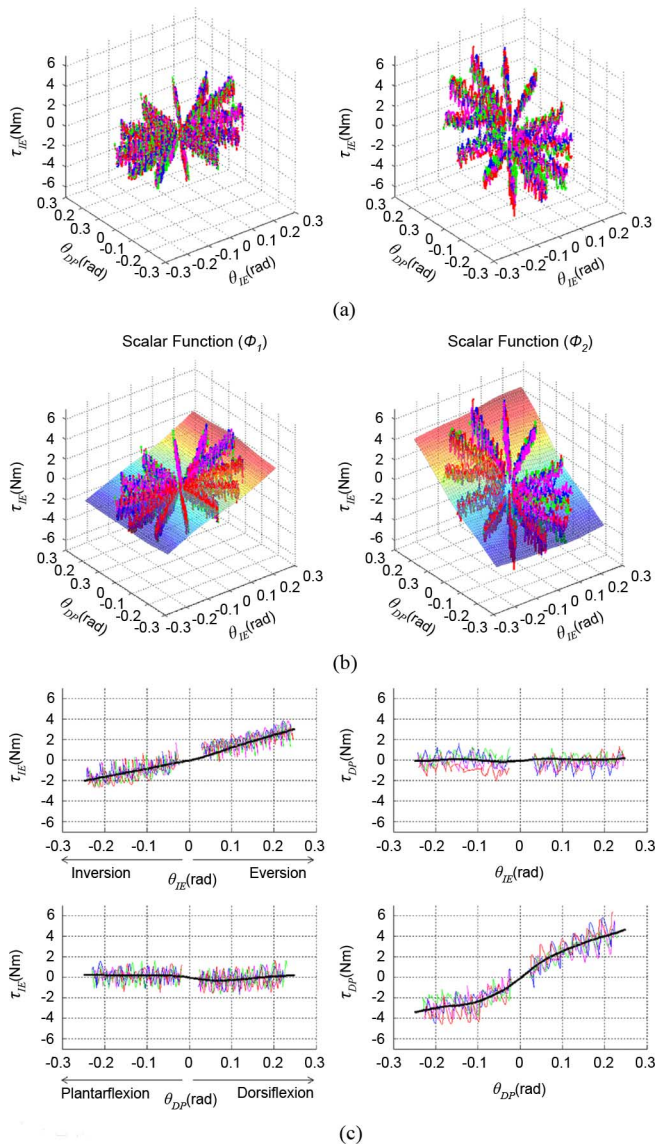

Fig. 4. Representative measurements and the resultant vector field. (a) Friction compensated measurements of a representative subject (subject #1 outbound data). Each color corresponds to each of four repeated measurements. (b) Four measurements and the fitted field (ϕ_1 and ϕ_2). (c) 2-D slices of the vector field in four major directions [IE (top) and DP (bottom)].

TABLE II
EFFECTIVE RESOLUTION FOR STIFFNESS ESTIMATION

Direction \ Study	Relaxed	TA Active	SOL Active	Co-contraction
Outbound	0.04 (0.01)	0.06 (0.02)	0.11 (0.03)	0.13 (0.04)
Inbound	0.05 (0.01)	0.06 (0.02)	0.13 (0.05)	0.13 (0.04)

Parentheses denote SE. The unit is Nm/rad.

four principal directions (inversion, eversion, dorsiflexion, plantarflexion) and the average for all directions are presented in (Table III). All subjects except one (#4) showed the greatest DP stiffness increase when co-contracting antagonistic muscles.

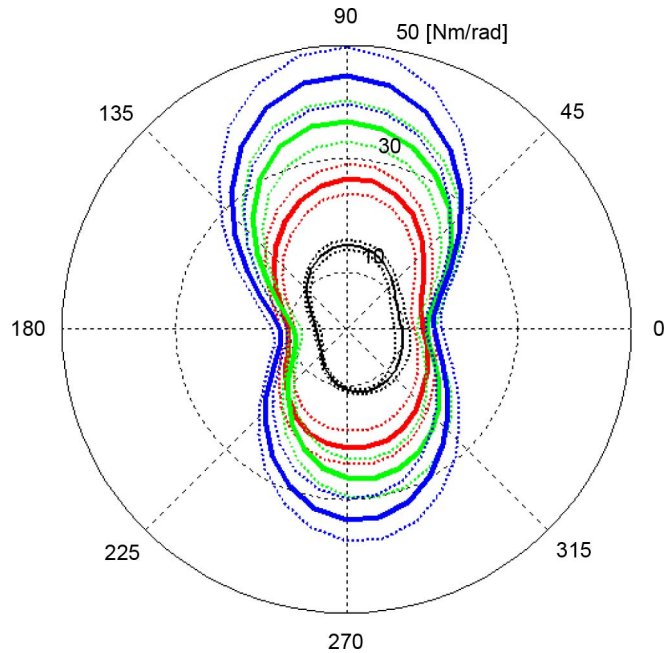

Fig. 5. Anisotropy of ankle stiffness: Stiffness increases significantly in all movement directions with active muscles ($p \ll 0.05$). Black: Relaxed. Red: TA active. Green: SOL active. Blue: Co-contraction. Solid line: Mean value of all analyzed subjects. Dashed: Mean $\pm$ SE.

TABLE III
RATIO OF ACTIVE STIFFNESS TO RELAXED STIFFNESS

Direction \ Study	TA Active	SOL Active	Co-contraction
Inversion	2.00 (0.24)	1.85 (0.20)	2.30 (0.22)
Eversion	1.45 (0.13)	1.55 (0.12)	1.69 (0.12)
Dorsiflexion	1.85 (0.23)	2.60 (0.40)	3.12 (0.42)
Plantarflexion	1.98 (0.26)	2.62 (0.29)	3.14 (0.35)
All Directions	1.87 (0.21)	2.22 (0.23)	2.65 (0.24)

Parentheses denote SE.

IV. DISCUSSION

An accurate characterization of ankle mechanical impedance with active muscles is important since most normal lower extremity functions require muscle activations either singly, synergistically, or antagonistically. Furthermore, abnormal muscle tone, a condition in which some muscle or group of muscles is hyper-active or hypo-active, is a common consequence of neurological disorder [4], [11], [22]. Mechanical impedance is not confined to elastic, viscous, and inertial behavior, but it is generally known that the static component predominates in the low frequency region (< 10 Hz) for single DOF ankle impedance [23]. In this study we identified the static component of active ankle impedance of unimpaired subjects in two coupled DOFs. Repetitive measurements using a wearable robot combined with robust function-approximation methods based on TPS with GCV enabled reliable characterization of the multivariable torque-angle vector field at the ankle in IE-DP space and how it varied with muscle activation. Although stiffness can

be calculated at any point of interest in the displacement field, to estimate the predominant behavior of the ankle, stiffness was calculated as the average slope of data points between the neutral and target positions. With these data we assessed the directional variation of ankle stiffness.

Measurements with a decreased number of movement directions from 48 [14] to 24 minimized muscle fatigue during the four repeated measurements, and EMG analysis showed that our young unimpaired human subjects could maintain substantially constant muscle contraction under the given experimental conditions. However, despite instructions, most subjects had difficulty activating only one muscle exclusively (Fig. 3). This is consistent with a growing body of evidence that the central nervous system addresses muscles in functional groups called synergies, and not individually [24], [25]. In addition, most subjects could voluntarily activate SOL more easily than GAS, and appeared to be especially incapable of focusing voluntary control on PL, at least in the context of this experiment. This was the reason we selected TA and SOL as targets for single muscle activation studies.

Under voluntary contraction of ankle muscles at 10% MVC either singly or antagonistically, the variability of repeated measurements was significantly smaller than the measurement error of our apparatus (Table I), although it was higher than when muscles were relaxed. This is consistent with previous studies showing increased variability of force with voluntary contraction [26].

In general, ankle stiffness increased in all directions with activation of a single muscle or co-contraction of antagonistic muscles (Table III, Fig. 5). Ankle stiffness was highly direction-dependent whether muscles were maximally relaxed or active. In all cases, the directional variation of stiffness exhibited a characteristic "peanut" shape, pinched in the IE direction. This might be expected, as normal locomotor progression is predominantly in the sagittal plane. However, the effect of muscle activation was not simply to scale stiffness magnitude as observed in the upper extremity [27]. The shapes observed with co-contraction or predominant TA or SOL activation were not scaled-up copies of relaxed behavior. Instead, contraction or co-contraction of the major dorsi- and plantar-flexors contributed much less to increase IE stiffness (Table III), which means the ankle remains relatively more vulnerable to frontal plane perturbations even with voluntary contraction of these muscles. This result is consistent with the observation that most ankle-related injuries occur in the frontal plane rather than in the sagittal plane [28], [29].

What might account for these results? One possible explanation is a different contribution of "passive" and "active" stiffness to the different movement directions. Passive stiffness adds to active muscle stiffness as a bias or offset from zero which was greater in DP than IE. If active muscles contributed more to DP than IE and their ratio was greater than the ratio of passive contributions to DP and IE, the total stiffness would increase more in DP than IE, as we observed. To clarify with a hypothetical example, a muscle that was aligned to contribute exclusively to DP torque and stiffness, with zero contribution to IE torque and stiffness, would add to DP stiffness but not IE stiffness. This explanation is consistent with the preponderance of potentially synergistic muscles contributing to DP but not IE: only five out of 13 ankle muscles contribute to inversion–eversion, while 12 out of 13 muscles contribute to dorsiflexion–plantarflexion.

TABLE IV
EFFECT OF MUSCLE ACTIVATION WAS NOT SIMPLY TO SCALE STIFFNESS MAGNITUDE

Increase Ratio in Co-contraction Study \ Reference Study		TA Active	SOL Active
Total Muscle Activation		1.87	1.71
Stiffness	DP Direction	1.64	1.20
	IE Direction	1.16	1.17
	All Direction	1.42	1.19

The ratios of total muscle activation and stiffness in the co-contraction study to their values in the single-muscle-active studies were calculated. Mean values for all subjects analyzed are presented.

Interestingly, the joint stiffness when antagonistic muscles were co-contracted was less than the sum of what was observed in the TA and SOL active studies (passive stiffness was included just once in summing the stiffness of TA and SOL active studies): 2.00 versus 2.43 for the IE direction, 3.13 versus 3.53 for the DP direction, and 2.65 versus 3.09 for all directions (Table III). This may have been due, in part, to the fact that the TA and SOL active studies also evoked a degree of co-contraction due to synergies. However, estimated total muscle activity in the co-contraction study exceeded that of the TA active study by 87% while the co-contraction joint stiffness exceeded that of the TA active study by only 64% in the DP direction, 16% in the IE directions, and 42% averaged over all directions (Table IV). Comparing the co-contraction study to the SOL active study, the discrepancy was even more marked: estimated total muscle activity in the co-contraction study exceeded that of the SOL active study by 71% while the co-contraction joint stiffness exceeded that of the SOL active study by only 20% in the DP direction, 17% in the IE direction, and 19% averaged over all directions (Table IV). In both studies, the joint stiffness increase ratios in DP, IE, and all directions were significantly lower than the corresponding increase ratios of estimated total muscle activity ($p \ll 0.05$).

Several possibilities might explain these experimental observations. First, it is known that, with fatigue, a greater amplitude of EMG is required to maintain a constant force. To the extent that subjects maintained constant levels of EMG, if they fatigued, the force (and presumably stiffness) they generated would have declined. However, the experimental protocol was specifically designed to avoid fatigue. Compared with previous experiments on ankle muscle fatigue [30], contractions at 10% MVC for 40 s with intervening 3-min rest periods seem very unlikely to have induced fatigue.

A second possibility arises from nonlinear musculo–tendon kinematics. The derivatives of muscle length with respect to joint angle define muscle moment arms which, in general, vary with joint angle [31], [32]. The nominal ankle positions in the TA and SOL active studies were slightly dorsiflexed and plantarflexed from the neutral position, respectively, while the nominal ankle position in the co-contraction study was close to

TABLE V
STIFFNESS MAY NOT INCREASE WITH CO-CONTRACTION

Ratio of active stiffness to relaxed stiffness				EMG amplitude levels normalized to MVC					
Direction / Study	Dorsi-flexion	Plantar-flexion	DP	Muscle / Study	TA	SOL	PL	GAS	Approximates of total muscle activity
TA Active	1.86	2.74	2.30	TA Active	8.27	3.10	1.30	1.42	14.09
SOL Active	2.65	2.28	2.47	SOL Active	1.23	7.55	4.45	1.86	15.09
Co-contraction	2.23	2.20	2.22	Co-contraction	8.83	8.33	3.55	1.93	22.64

One subject (#4) exhibited lower DP stiffness during co-contraction than with single muscles active despite comparable or greater levels of activation.

the neutral position. This difference of nominal ankle position might have influenced the measured ankle stiffness. To explore this possibility, the stiffness in each study was evaluated based on shifted displacements. However, the effect of different initial ankle positions was negligible, not enough to explain our experimental observation.

A third possibility is also related to nonlinear musculo-tendon kinematics. Because humans have an endo-skeleton, if tendon tension is nonzero, that leads to a negative joint stiffness of purely kinematic origin [16], [17]. Any relation between intrinsic muscle stiffness and tension will vary between muscles; longer muscles of lower cross-sectional area may be expected to contribute more tension with less stiffness. Thus contraction (or co-contraction) of longer, slenderer muscles may, in principle, reduce joint stiffness. However, both the positive (intrinsic) and negative (kinematic) contributions of any muscle contribute linearly to net joint stiffness. Therefore, if muscle activity in the co-contraction study was the simple sum of muscle activities in the TA and SOL active studies, the stiffnesses should also have added; but they did not.

A fourth possibility may arise from possible contributions of unmonitored muscles. We monitored four superficial muscles but eight out of 13 ankle muscles are deep and were not monitored. Those muscles might have contributed positively or negatively to ankle stiffness. If they contributed positively, it might be that these deep muscles were comparably active in all three studies. As a result, their activity in the co-contraction study would not have been the sum of their activities in the TA and SOL active studies. That would result in our observation that co-contraction stiffness was not the sum of TA and SOL active stiffnesses. In contrast, deep muscles tend to be more slender than superficial muscles (such as gastrocnemius) and might contribute negatively to joint stiffness. If they were more active in the co-contraction study, they would result in our observation that co-contraction stiffness was not the sum of TA and SOL active stiffnesses.

Interestingly, one subject (# 4) exhibited lower DP stiffness when co-contracting antagonistic muscles than when activating single muscles, even though muscle activation levels were comparable in all cases and this cannot be dismissed as imprecision of our measurement (Table V). In detail, when all normalized EMG amplitudes were summed as an approximate estimate of total ankle muscle activity of this subject, the co-contraction study showed 50%–60% greater total activation: TA active sum was 14.09; SOL active sum was 15.09; co-contraction sum was 22.64 (Table V). Joint impedances add and we should expect an increase of impedance with co-contraction [33]. However, in the dorsiflexion direction, the stiffness increase in the co-contraction study was only 18.8% compared to TA active study and decreased compared to the SOL active study by -16.6%. In the plantarflexion direction, the difference was even more compelling: -24.6% compared to the TA active study and -3.6% compared to the SOL active study. The average of both directions was -3.6% and -11.3% compared to the TA active and SOL active studies, respectively, substantially smaller than the 50%–60% increase expected from the sum of muscle electrical activities.

Of the possible explanations we considered above, the only one that appears to be compatible with this observation is that unmonitored deep muscles contributed negative joint stiffness (consistent with their slenderness) and were more active in the co-contraction study. However, only one subject out of 10 clearly displayed this phenomenon and further study is required to understand this observation. Nevertheless, even this single subject emphasizes that there is no guarantee that joint stiffness will increase in proportion to muscle activation.

Consequently, our observation that, averaging over all subjects, net ankle stiffness increased with muscle activation-by as much as a factor of 2 to 3 with co-contraction (Table III)—is not *a priori* obvious. It suggests that impedance modulation by antagonist muscle co-contraction—despite its substantial cost in metabolic energy consumption—serves an important function; and that function is sufficiently important that muscle may have co-evolved with musculo-skeletal kinematics so that its stiffness increased sufficiently rapidly with force to ensure stability [34]. In fact, a recent study showed that the limits of upper-extremity force production were determined by the need to stabilize the joints [35] rather than by muscle strength.

The musculo-skeletal anatomy of the ankle is highly variable in humans [36] and that implies a comparable variability in the effect of muscle activity on joint stiffness; as we observed, increasing muscle activity may not always increase joint stiffness. Moreover, biomechanical injury to the passive tissues of the ankle may permanently change the relation between muscle activity and joint stiffness, and the full range of ankle stiffness (and in particular, joint stability) that was available pre-injury may no longer be accessible post-injury. Further experimental study is required to quantify these possibilities.

Our observation that, averaging over all subjects, ankle stiffness increased with muscle activation implies that hypertonus will generally result in elevated stiffness. In a recent study, Roy *et al.* reported that chronic stroke survivors exhibited greater passive ankle stiffness in dorsiflexion and inversion than age-matched unimpaired subjects [11]. These stroke survivors also

exhibited abnormal tone, an average in dorsiflexion of 1.6 on the Modified Ashworth Scale (MAS) which runs from 0 (normal) to 5 (rigid) [37]. Roy reported only a very weak correlation between the MAS and their stiffness measurements but that may be due to the widely-acknowledged inherent limitations of that scale, which tends to yield scores clustered near the low end of the range [37], [38]. Measurements such as those we report here may provide a more reliable assessment of altered physiological function [39].

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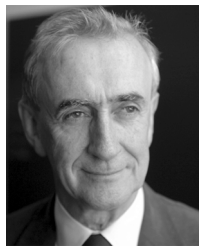

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