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Ankle Mechanical Impedance During Walking in Chronic Stroke: Preliminary Results

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Abstract

Dynamic joint mechanics, collectively known as *mechanical impedance*, are often altered following upper motoneuron disease, which can hinder mobility for these individuals. Typically, assessments of altered limb mechanics are obtained while the patient is at rest, which differs from the dynamic conditions of mobility. The purpose of this study was to quantify ankle impedance during walking in individuals post-stroke, determine differences from the healthy population, and assess the relationship between impedance impairment and clinical outcome measures. Preliminary data were collected in four individuals post-stroke. Displacement perturbations were applied to the ankle during stance phase, and least-squares system identification was performed to estimate ankle impedance. In comparison to the healthy population, the paretic ankle showed reduced variation of stiffness during mid-stance of walking, and damping estimates during early and mid-stance were increased. Clinical measures obtained during dynamic tasks showed strong correlation with changes to the stiffness component of impedance, while clinical measures obtained passively were not correlated to stiffness. Impairment in ankle damping was not correlated with any of the measures tested. This work provides novel, preliminary insight into paretic ankle impedance during walking, differences from healthy data, and elucidates how current clinical metrics correspond to the true values of ankle stiffness and damping during gait.

I. Introduction

Our limited understanding of lower limb mechanics post-stroke hinders our ability to assess and treat the accompanying locomotor dysfunctions. There are 6.6 million stroke survivors in the United States, with nearly 800,000 people experiencing new or recurrent strokes annually [1]. Even after standard rehabilitation therapies, up to 80% of stroke survivors experience persisting difficulties with ambulation [2, 3]. Furthermore, limitations to mobility increase patients' risk for recurrent stroke and vascular death [4]. A key factor contributing

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to these locomotor impairments are changes in leg joint mechanics; manifesting as hemiparesis and/or spasticity and hypertonia (present in over 50% of individuals post-stroke within the first year [5, 6]). Paresis of the ankle causes drop-foot and reduced plantar flexion effort [7]. Moreover, spasticity and hypertonia create additional resistance that must be overcome during gait. These biomechanical changes that occur can be described quantitatively by modifications to joint stiffness and damping properties.

A number of clinical measures are used to assess impairment due to hemiparesis and spasticity, however, often these assessments are qualitative and their link to functional outcomes is tenuous. Sensorimotor impairment is commonly assessed using the Fugl-Meyer Assessment of Motor recovery (FMA), while measures such as the Six-Minute Walk Test (6MWT) are commonly used to assess activity and disability [8]. While the FMA scale is based on the well-defined stages of motor recovery, it is subject to ceiling effects and provides qualitative outcomes that can vary with the clinician administering the test [9]. The 6MWT provides a quantitative measure of submaximal exercise capacity, however is susceptible to learning and motivation effects, and does not directly assess biomechanical changes leading to reduced activity [10]. To evaluate spasticity and hypertonia, clinicians use coarse, qualitative assessments performed while the patient is at rest and the limb is not bearing weight (*e.g.* Modified Ashworth Scale (MAS)). The MAS is widely used, but is a highly subjective measure with lower levels of reliability, which limits the scale's usefulness for comparison across studies [10]. More sophisticated metrics exist, such as electromyography and postural dynamometry, but are not routinely used due to inconsistent findings [11]. Although treatment of spasticity has appeared to be successful using passive, categorical evaluations, this success has not translated to functional improvements during ambulation. Limited functional improvement may be attributed, in part, to an incomplete understanding of how stroke alters the mechanical properties of the limbs during dynamic tasks.

Although the importance of the regulation of joint mechanics—and changes due to pathology—have been recognized [12, 13], experimental and computational challenges have prevented the study of these properties during locomotion. Instead, research has focused on quantifying stiffness and damping—collectively with inertia known as *mechanical impedance*—in resting postures [14–16]. These studies have led to an improved understanding of the intrinsic and reflex components of ankle impedance post-stroke [17–19]. Wearable robotic technology have been developed with the purpose of estimating impedance during dynamic tasks, but have not yet quantified impedance during walking [20]. Our group has developed a perturbation-based approach to quantify ankle impedance dynamically during gait using a mechatronic platform. We have measured these properties in the healthy human ankle during walking [21, 22]; however, no studies have previously investigated ankle impedance in the presence of pathology during locomotion, and thus functional benefits of addressing these impairments remain unknown.

The purpose of this study was to quantify the mechanical impedance (*e.g.* stiffness and damping) of the ankle joint during walking for subjects with impairments post-stroke, while obtaining comparisons for standard clinical metrics. Ankle impedance was estimated at four discrete time points that span the stance phase of walking. We hypothesized that impedance estimates for the paretic ankle post-stroke would differ from the healthy population

previously studied in both magnitude and variation across stance phase. The intention of this work is to provide direct quantitative measures which characterize how paresis, spasticity, and other changes to joint impedance manifest during locomotion.

II. Methods

A. Experimental

1) Clinical Measures: Prior to completing the experiment, clinical outcome measures were performed for all participants using a consistent physical therapist. The Six Minute Walk Test (6MWT) and Ten Meter Walk Test (10MWT) were conducted to ensure all participants had sufficient endurance and speed to complete the experiment. To further assess impairment level, participants meeting the endurance and speed requirements performed the Spinal Cord Assessment Tool for Spastic Reflexes (SCATS), the Modified Ashworth Scale (MAS), and the Lower Extremity Fugl-Meyer (LE-FM). The 6MWT provides assessment of aerobic capacity and gait endurance. The 10MWT evaluates participants' functional mobility and walking speed. The SCATS assesses spastic motor behaviors and each item is scored from 0 (no impairment) to 2 (severe impairment). The MAS measures spasticity and is scored from 0 (no impairment) to 4 (severe impairment). Finally, the LE-FM evaluates sensorimotor impairment, and is scored from 0 (severe impairment) to 2 (no impairment).

2) Apparatus: A single degree of freedom mechatronic platform was used to perturb the ankle throughout stance phase of walking. This device, termed Perturberator Robot, has been previously described and validated in [23], and has been shown to yield stiffness estimates within approximately 5% error when compared to an independent measure. The Perturberator Robot was recessed into a 5.25 m walkway, and a portable force platform (model: 9260AA3, Kistler Inc, Amherst, NY) was rigidly attached to measure ground reaction forces (GRFs) during perturbations.

3) Protocol: Four chronic stroke participants (> 2 years post-stroke) were included in this preliminary study (1 male, 3 female, age 42 ± 7 years, weight 86.1 ± 10.9 kg). Participants were excluded if they had any history of major ankle injury or Botox treatment for ankle spasticity. Participants were also excluded if they were unable to complete the 6MWT or if their self-selected 10MWT speed was less than 0.45 m/s. This study was approved by the Northwestern University Institutional Review Board and all subjects provided written, informed consent. A description of the experimental protocol was previously detailed in [21], and summarized here.

Participants were secured to an overhead gantry system using a safety harness and wore treaded hospital socks (Medichoice, Mechanicsville, VA, USA) to prevent slippage. They were instructed to walk across the walkway at a pace between 55–60 steps/min, matching the frequency of a metronome. Subjects were given time to familiarize with that task, however error may arise from incorrect foot placement. Sensitivity of stiffness estimates to misalignment was previously quantified, showing a reduction in stiffness by 6% per cm offset [24]. Care was taken throughout the data collection to adjust subject's starting location such that their ankle aligned with the center of rotation of the robot, and trials with more

than 3 cm misalignment were excluded from analysis. Upon interaction with the force platform, 2° ramp perturbations were triggered with 50% probability. Perturbations were triggered at 30%, 50%, 70%, and 85% of stance phase (Fig.1) in a randomized order.

B. Analytical

Collected data were low-pass filtered using a bidirectional third-order Butterworth filter with a 20 Hz cutoff frequency, and were subsequently segmented into 100 ms windows beginning with the onset of perturbation. Machine forces arising from the robot's intrinsic inertia were removed using linear filters previously estimated in [23]. Ankle torque was determined by resolving GRFs to the equivalent torque at the ankle's center of rotation:

$$T = F_z \delta_x + F_x \delta_z \quad (1)$$

where F_x and F_z are the anterior-posterior and vertical components of the GRF respectively. The moment arms δ_x and δ_z were determined by transforming center of pressure (COP) data to the ankle's coordinate system, and represent the perpendicular distance to the ankle's center of rotation. During early-mid stance—when the foot is flat on the ground surface—the anterior-posterior distance between the point of heel contact and the ankle's center of rotation (determined in software using high definition video) was subtracted from COP data. As the heel rises during terminal stance, motion and deformation of the midfoot result in inaccurate results when using the static transformation. Therefore, a biomechanical model of the foot, previously shown to be robust to inter-subject variation was used to transform COP data to the ankle's center of rotation [22].

Mechanical impedance was determined at each time point using least-squares system identification with a second order parametric model [21, 22]. Second order models have been shown to provide high quality estimates of ankle impedance in both postural and dynamic impedance studies [21, 22, 25–27]. To provide an estimate of variability, a bootstrapping technique was used [21]. In every iteration of this technique, an initial trial was selected at random. To account for slight differences in timing of perturbations at each time point, a probability algorithm selected additional trials such that temporally-similar trials were selected with higher probability until 60% of trials for a specific time were included. Selected trials were averaged, and offset was removed such that both torque and angle begin with zero. The perturbation torque and angle responses were isolated by subtracting the torque and angle variation that occurred naturally during walking, which were obtained from nonperturbed trials. Ankle impedance was estimated during walking using a second order parametric model

$$T_p = I_{tot} \ddot{\theta}_p + b_a \dot{\theta}_p + k_a \theta_p \quad (2)$$

where T_p is the torque response to perturbation, I_{tot} is the total inertia of the foot and other coupled body segments, k_a and b_a are the stiffness and damping components of ankle impedance, respectively, while θ_p is the angular displacement of the perturbation. The first and second derivatives of θ_p were calculated numerically, quantified by the coefficients of a second-order polynomial fit locally to the data [28]. Impedance parameters were estimated over the 100 ms window in each bootstrapped trial.

B. Comparisons

In this preliminary study, impedance estimates for the chronic stroke population are compared to healthy results, but statistical comparisons were not obtained due to the limited number of subjects.

To investigate the relationship between clinical outcome measures and impedance impairment, individual *impedance* scores were linearly regressed on 6MWT distance, total MAS for the ankle, and LE-FM scores for the ankle. A *impedance* score was calculated by first defining impedance parameters (stiffness and damping) as a function of stance phase for both the healthy and chronic stroke populations via interpolation. Change in each impedance parameter was determined by subtracting the average healthy impedance functions [21, 22] from each subject's impedance functions. The *impedance* score was defined as the average absolute change in impedance from healthy across stance phase, and was assessed separately for ankle stiffness and damping components. Impedance impairment post-stroke varied throughout stance phase such that in some portions of stance stroke survivors exhibited increased impedance, while in others impedance was reduced. The absolute change was selected to capture overall deviation from healthy, while reducing the data to a single number. Total scores for the MAS and LE-FM were determined as the summation of sub-item scores pertaining to the ankle (highlighted rows in Table 1).

III. Results

Clinical outcome measures for each participant are summarized in Table 1.

Ankle impedance estimates were highly variable across the four subjects. Comparing stiffness estimates across stance phase, values were relatively constant, and did not display the typical increase in stiffness during mid-stance seen in the healthy population (Fig. 2A). Damping estimates in early and mid-stance were increased post-stroke, decreasing to healthy values in terminal stance (Fig. 2B).

Comparison of *impedance* scores to clinical outcome measures showed that few clinical measures relate to ankle impedance impairment during walking. A larger change in stiffness from healthy values was strongly correlated with reduced 6MWT distance ($R^2 = 0.82$, Fig. 3A) and LE-FM score ($R^2 = 0.88$, Fig. 3E); a lower score in these measures indicates greater impairment. Weaker correlation is seen between increased change in stiffness and increased MAS score ($R^2 = 0.6$, Fig. 3C); where higher MAS indicates greater impairment. Damping impairment was not correlated with 6MWT Distance ($R^2 = 0.45$, Fig. 3B), MAS score ($R^2 = 0.03$, Fig. 3D), or LE-FM score ($R^2 = 0.14$, Fig. 3F).

IV. Discussion

This study provides preliminary investigation into how paretic ankle mechanical impedance varies throughout the stance phase of walking for individuals with chronic stroke.

Preliminary results show reduced variation in ankle stiffness across stance phase in individuals post-stroke. Damping is increased during early and mid-stance phase. Few clinical outcome measures correlate with ankle impedance.

A. Comparison with Healthy Population

On average, stiffness estimates during early stance phase and terminal stance phase were similar to the healthy population (Fig. 2A). However, the individuals post-stroke trended towards a reduced stiffness variation throughout mid-stance for the paretic ankle. These results contrast previous results from passive impedance studies, which showed the paretic ankle to have higher stiffness when compared to healthy controls [29]. These differences may be associated with changes to neuromodulatory drive occurring following stroke [30]. During mid and terminal stance phase, stroke survivors exhibit lower peak muscle activation in comparison to the healthy population [31]. Joint stiffness arises, in part, due to activation and co-activation of muscles spanning the joint [26, 32–34]; lower stiffness that stems from this reduced muscle activity may be expected.

Damping estimates throughout early and mid-stance phase are increased in comparison to the healthy population, returning to healthy levels in terminal stance (Fig. 2B). These results are in agreement with previous research in ankle impedance post-stroke obtained during passive conditions. Prior work found the affected ankle to have increased damping in comparison to the healthy ankle when in dorsiflexion, while damping values during plantar flexion were not significantly different from healthy controls [18]. Data obtained in this study are consistent with these previous findings; however, the increase in damping values during walking is greater than during static postural studies, indicating that there may be other factors contributing to impaired damping values during dynamic tasks.

B. Comparison with Clinical Outcome Measures

Impairment of mechanical impedance manifested differently for each subject. Both stiffness and damping coefficients of the paretic ankle were affected, but only the stiffness component correlated with functional outcome measures. Clinical measures obtained passively did not correlate with impedance during walking.

The 6MWT strongly correlated with stiffness impairment (Fig. 3A). Chronic stroke survivors have reduced stiffness during mid-stance potentially arising from weakness and paresis of the triceps surae muscles, which also leads to foot drop and plantarflexion failure [7]. Consequently, more impaired individuals may have difficulty with forward propulsion, as well as reduced gait endurance and speed [35].

The MAS scores weakly correlated with stiffness impairment (Fig. 3C). Participants primarily experienced spasticity of plantar flexors according to the MAS (Table 1), therefore, rapid stretch of the plantar flexors may elicit a spastic response. Throughout mid-stance phase, plantar flexors are stretched as the tibia progresses over the foot. Contrary to indications from the MAS, ankle stiffness during mid-stance (30 % to 40% stance) remained low (Fig. 2A). These results agree with previous works which found MAS did not correlate joint stiffness in passive conditions [36].

The LE-FM correlated strongly with ankle stiffness impairment during walking in the chronic stroke population (Fig. 3E). A sub-item of the LE-FM that was impaired across all four subjects was “dorsiflexion out of synergy” (Table 1). During this item participants stand and lift their foot, while keeping their knee extended and heel on the floor. Participants all

had difficulty activating the tibialis anterior while the limb was loaded. Joint stiffness arises in part due to activation and co-activation of muscles spanning the joint [37], therefore weakness of dorsiflexors and/or plantar flexors is expected to translate to reduced overall stiffness of the ankle. Our stiffness results align with these findings as all participants exhibit decreased stiffness during mid-stance (between 30% and 70% of stance) when the ankle is dorsiflexed.

Perhaps the most striking impedance impairment is the high damping values throughout early and mid-stance post-stroke. The results of the clinical measures performed do not correlate with increased damping in these subjects (Fig. 3B, D, F). This suggests that standard clinical measures tested may not be able to assess damping impairments that present in functional tasks following stroke. The MAS measures velocity-dependent increase in muscle tone—corresponding to the damping component of impedance—obtained during passive conditions, but MAS scores did not correlate with impaired ankle damping values during walking.

C. Limitations

The preliminary results of this study provide first insight into ankle mechanical impedance during walking for the chronic stroke population, assuming quasi-static second-order dynamics of the ankle. In reality, during the 100ms analysis window the kinetics, kinematics and muscle activation, that define the system's operating point, are nonstationary. This simplification was required for the analysis techniques, but may not accurately capture the time-varying nature of mechanical impedance during locomotion [21, 22, 38].

Our results are compared to previously reported impedance estimates for the young, healthy population walking at 85–90 steps/min. Previous work has shown impedance estimates to vary with changes in muscle activation [27, 39]. Gait speed effects overall muscle activity [40], therefore, differences in impedance seen between the chronic stroke and healthy populations cannot be entirely attributed to mechanical changes of the ankle post-stroke. Future work will conduct comparisons between the affected and non-affected ankles of the stroke population. Furthermore, data from age matched and gait speed matched controls are needed to further isolate the effect of chronic stroke on ankle impedance changes.

The impedance scores regressed against clinical measures represent average deviation from healthy impedance values across stance phase. However, impedance impairment varies based on the sub-phase of stance (*i.e.* early, mid or terminal stance). These differences throughout stance phase, and the sign of deviation from healthy data, are currently not addressed. Future analyses will provide more focused analyses at each time point.

Finally, this preliminary work includes results from only four chronic stroke participants. Prior to conducting this experiment, a power analysis was completed. A detectable difference of 0.75 Nm/rad/kg was chosen to capture a 15% change in stiffness across the range of values reported across stance phase of walking [21, 22]. For a statistical power of $(1-\beta) = 0.8$, nine subjects were required for the selected detectable difference. Future work will focus on a larger clinical study, which will enable statistical analyses.

V. Conclusion

This work provides preliminary estimates of ankle mechanical impedance during walking for the chronic stroke population, and enables insight into how these properties relate to clinical outcome measures and differ from ankle mechanical impedance observed in healthy populations. Passive clinical measures did not correlate to impedance impairment, and no clinical measures tested captured damping impairment. However, the 6MWT and LE-FM did correlate with changes to the stiffness component of impedance. In comparison to the healthy population, the paretic ankle shows a reduced stiffness variation throughout mid-stance phase of walking, and increased damping during early and mid-stance phase.

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Figure 1. Average torque-angle relationship for a representative subject. Time points analyzed are denoted by dots. Sub-phases of stance are indicated by shaded regions.

Figure 2.

Time varying ankle impedance during walking modified from [21]. Bodyweight normalized ankle stiffness (A) and damping (B) are reported for the stance phase of walking for two populations. Grey traces denotes previous results for ankle impedance in the healthy population [20, 21]. Blue traces indicate preliminary results for average stiffness and damping across 4 chronic stroke subjects.

Figure 3.

Absolute average change in impedance across stance phase are plotted against clinical outcome measures for each subject. Clinical measures indicated denote the sum of values for sub-measures related to the ankle for each clinical test (highlighted in blue in Table 1). A larger change in stiffness from healthy values is strongly correlated with reduced 6MWT distance (A) and LE-FM score (E); a lower score in these measures indicates greater impairment. A weaker correlation is seen between increased change in stiffness and increased MAS score (C); where higher MAS indicates greater impairment. Change in damping from healthy was not highly correlated with 6MWT Distance (B), MAS score (D), or LE-FM score (F).

TABLE I.

Clinical Measures

Clinical Measure	Subject			
	CVA01	CVA02	CVA03	CVA04
6MWT				
<i>Distance (m)</i>	496.8	566.9	199	375.5
10MWT				
<i>Self-selected velocity (m/s)</i>	1.48	1.34	0.53	0.95
<i>Fast velocity (m/s)</i>	2.73	1.95	0.95	1.24
SCATS (paretic/nonparetic) ^{a, b}				
<i>Plantar flexor clonus</i>	0/0	0/0	1/0	0/0
<i>Knee/Hip flexor spasms</i>	1/1	0/0	0/0	1/0
<i>Knee/Hip extensor spasms</i>	0/0	0/0	0/0	0/0
MAS ^{a, c}				
<i>Hip Flexors</i>	1	0	1	0
<i>Hip Extensors</i>	0	0	0	0
<i>Hip Adductors</i>	1	0	0	0
<i>Knee Flexors</i>	1	1	2	1
<i>Knee Extensors</i>	0	0	0	0
<i>Dorsiflexors</i>	0	0	0	0
<i>Plantarflexors</i>	0	1	3	1
LE-FM ^{a, d}				
<i>Motor Function (28)</i>	22	23	17	23
Seated knee flexion	2	2	2	2
Seated dorsiflexion	1	2	0	2
Tremor	2	1	2	2
Dysmetria	2	1	0	1
Speed	1	0	1	1
Hip flexor synergy	2	2	2	2
Knee flexor synergy	2	2	2	2
Dorsiflexion synergy	2	2	1	2
Hip extensor synergy	2	2	2	2
Adductor synergy	2	2	2	2
Knee extensor synergy	2	2	2	2
Plantar flexion synergy	1	2	1	2
Knee flexion out of synergy	1	2	0	1
Dorsiflexion out of synergy	0	1	0	0
<i>Reflex Activity (4)</i>	4	4	4	2
Achillies	2	2	2	0
Patellar	2	2	2	2
<i>Sensory Function (12)</i>	11	10	8	11

Clinical Measure	Subject			
	CVA01	CVA02	CVA03	CVA04
Thigh light touch	2	1	1	2
Sole of foot light touch	1	1	1	1
Hip proprioception	2	2	2	2
Knee proprioception	2	2	2	2
Ankle proprioception	2	2	2	2
Toe proprioception	2	2	0	2

^a Clinical measures pertaining to the ankle are highlighted in blue.

^b Scored from 0 to 2 where 0 indicates no impairment and 2 indicates severe impairment

^c Scored from 0 to 4 where 0 indicates no impairment and 4 indicates severe impairment

^d Scored from 0 to 2 where 2 indicates no impairment and 0 indicates severe impairment