

**Background:**

Nearly 800 000 people experience new or recurrent stroke annually (Mozaffarian et al. 2016), with approximately 50-60% of stroke survivors experiencing motor impairment even after standard rehabilitation (Belda-Lois et al. 2011). Stroke survivors exhibit changes to joint kinetics, kinematics and mechanics during walking which manifests as gait deficits such as reduced gait speed, instability, and asymmetry. Changes to overall joint mechanics (stiffness and damping) during walking have not yet been addressed in the development of treatments and assistive technology. Joint stiffness, damping and inertia are collectively known as joint impedance, and are fundamental to our ability to interact with the world. The nervous system actively regulates these properties to facilitate weight acceptance, energy exchange between joints, and adaptation to unexpected changes in dynamic tasks such as walking (Burdet et al. 2001; Takahashi, Scheidt, and Reinkensmeyer 2001). Recent studies have quantified joint impedance of the human ankle throughout entire gait cycle for the young, healthy population (Rouse et al. 2014; Lee and Hogan 2015; Shorter and Rouse 2018), but it is still unknown how these properties are altered following stroke. Previous research has shown that upper motor neuron disorders impair joint stiffness in static postures when the patient is relaxed (Galiana, Fung, and Kearney 2005; Mirbagheri et al. 2008; Hyunglae Lee et al. 2011; Roy et al. 2013), however, it is unclear how relevant these results are to dynamic tasks such as walking. Our objective is to investigate how ankle joint stiffness and damping vary throughout the stance phase of walking in stroke survivors, and evaluate differences from healthy controls. Characterizing ankle impedance is critical to our understanding of how the nervous system regulates the mechanics of locomotion, and may provide novel, and fundamental information that can be used to better understand, assess and treat functional aspects of impaired joint mechanics.

**Methods:**

Apparatus: A single degree of freedom mechatronic platform, termed the Perturberator Robot, was used to elicit dorsiflexion perturbations necessary for estimation of ankle impedance during stance phase of walking. The robot was recessed into walkway such that the surfaces of the hinged platform and walkway align. A portable force platform rigidly attached to the hinged platform measured reaction forces. An AC gear motor (model: AKM42H-ANC2C-00, Kollmorgen, Radford, VA) controlled by a commercial servodrive (model: AKD-B00606, Kollmorgen, Radford, VA) was used to drive the device to the desired angle. The servodrive received position control instructions from a microcontroller (model: PIC32, Microchip Technology, Inc., Chandler, AZ, USA).

Prescreening Protocol: Stroke survivors (>2 years post stroke) recruited to participate were prescreened to evaluate level of impairment via standard clinical measures, and ensure capability and comfort in performing the experimental protocol. The 6 Minute Walk Test (6MWT) and 10 Meter Walk Test (10mWT) were used to evaluate ambulatory capabilities, and ensure participants are capable of meeting and maintaining the required walking speed during the experimental protocol. The degree of impairment of included participants was evaluated using the Lower Extremity Fugl-Meyer Assessment (FMA-LE), Modified Ashworth, and Spinal Cord Assessment Tool for Spastic Reflexes (SCATS) to assess motor and sensory impairment, as well as the degree of spasticity.

Experimental Protocol: Participants completed the following protocol for both their paretic and non-paretic ankle. The ankle was outfitted with an electrogoniometer, and subjects walked across the walkway such that the outfitted ankle aligns with the Perturberator's center of rotation upon stepping on the hinged platform. Ramp perturbations, 2° in magnitude, were triggered randomly with 50% probability when subjects stepped on the force platform. Four perturbation time points following heel strike were examined, occurring at 30, 50, 70, and 85% of stance phase.

Analysis: Ankle torque was determined by resolving ground reaction force (GRF) to equivalent torque and force at the ankle's center of rotation. Natural walking torque and angle profiles were removed by subtracting the average non-perturbed torque and angle profiles from the average perturbed trials. The resultant perturbation response was input to a second-order parametric model comprised of stiffness, damping and inertia. Mechanical impedance of the ankle was estimated using least squares system identification.

**Preliminary Results and Discussion:**

Preliminary results from a single stroke subject show stiffness in the spastic ankle to be increased relative to the healthy population reported in literature (Fig. 1). Additionally, the level of stiffness modulation throughout stance phase is decreased in the stroke subject, with the range of stiffness throughout stance limited to 9.36 – 12.85 Nm/rad/kg. Ankle damping in the stroke subject was similar to the healthy population during early and mid-stance phase, at approximately 0.001 Nms/rad/kg. However, rather than damping increasing in preparation for toe-off, as has been seen in healthy subjects (Lee and Hogan 2015; Shorter and Rouse 2018), the stroke subject showed a marked decrease in damping during late stance to approximately 0 Nms/rad/kg. Our preliminary results suggest that the spastic stroke population experience both an increase in joint stiffness, as well as a reduction in neuromodulatory drive; additional data collection is required to confirm and generalize these results.