Estimation of Intrinsic Joint Impedance using Quasi-Static Passive and Dynamic Methods in Individuals with and without Cerebral Palsy

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Abstract— **Modeling the passive behavior of the knee in subjects with spasticity involves the applied external torques (e.g. gravitational torque), the intrinsic moments due to tissue properties, as well as active, neurally defined moments resulting from the hypersensitivity of reflexes introduced by disability. In order to provide estimates of the necessary intrinsic terms in the equation of motion, the push-pull and Wartenberg Pendulum Knee Drop (PKD) tests were administered. Four subjects without disability and two subjects with Cerebral Palsy (CP) were evaluated for their active and intrinsic knee stiffness parameters. Separation of these two terms requires an additional stiffness term be added to the traditional equation of motion. This holds true for subjects with and without neurological disability. Very interestingly, the optimized non-disabled PKD produced lumped stiffness (K) that is similar to the push-pull passive stiffness (K_I) for both populations. On the other hand the optimized K value in the PKD test for subjects with disability was approximately 19 times larger than the K_I value found graphically from the push-pull test. This leads us to the conclusion that we can partition our lumped K as the sum of a neurally generated stiffness (K_a) and K_I to complete the trajectory model. Therefore, this study shows that spasticity is a velocity dependent, that would not appear in disabled individuals unless the examined limb has a non-zero velocity.**

Keywords: Pendulum Knee Drop test, PKD, push-pull test, Intrinsic Stiffness, Neural Stiffness, Cerebral Palsy, Spasticity, Dystonia, Muscle Tone, Modeled Trajectory, Optimization Model.

I. Introduction

Spasticity is a manifestation of neuromotor disorders (including CP, stroke and spinal cord injury) and is generally believed to negatively impact functional motor skills. It is widely referred to as velocity-dependent hypersensitivity of the stretch reflexes [1]. This results in increased muscle tone that resists movement, and in turn higher joint impedance (stiffness and damping) that is of neural origin. In contrast, all biological joints also have intrinsic or passive stiffness and damping due to the properties of the materials surrounding the joint. Among individuals whose disability may include spasticity, there is a risk of increased passive joint impedance due to contracture and other tissue changes [2] Therapeutic interventions must be differently tailored to address these increases in joint stiffness and damping of neural (active) and mechanical (passive) origin. [3]

To assess the resulting changes in joint motion due to spasticity, the PKD is often administered [4]. Initially, PKD trajectories of disabled and non-disabled individuals were assessed subjectively based on appearance. Later work has extracted quantitative measures from the test by modeling the pendular behavior of the resulting trajectories. Among non-disabled subjects these appear as damped pendulum oscillations that can be represented by the following equation of motion that includes the shank's length to the center of mass (L), moment of inertia (I), shank's mass (m), damping (B) and stiffness (K).

$$I\theta'' + B\theta' + K\theta = mgLsin(\theta) \quad (1)$$

As this equation has been shown to be inadequate to model the PKD trajectories of subjects with spasticity, Lin and Rymer [5] as well as Fowler et. al [6] modified the stiffness and damping over the trajectory duration to form a piecewise nonlinear system. Cavorzin et. al. [7] added a model of the reflex hypersensitivity to produce the velocity-dependent spastic torques. In contrast Fee and Foulds [8] added bursts of extra energy, which they described as muscle reflexes, at optimized times. Our more recent work [9, 10, 11] explored the addition of both exponential and sigmoidal virtual trajectories (θ_{vt}) that modify Eq.1 and allow the stiffness term to produce the necessary additional torque to model the trajectories of subjects with spasticity. Unlike the non-disabled trajectory, which oscillates about a constant angle, the spastic trajectory oscillates about reference angles that change over time. This correlates with the equilibrium point hypothesis (EPH), which states that a virtual reference trajectory (θ_{vt}) is defined by the neuromuscular system and serves as an attractor that the target limb follows [12]. By incorporating a time-varying velocity of θ_{vt} along with the changing reference trajectory (θ_{vt}), the clinical definition of spasticity become synchronous with engineering terms and provides an adequate representation that can be rewritten as:

$$I\theta'' + B\theta' + K(\theta - \theta_{vt}) = mgLsin(\theta) \quad (2)$$

Neither the extracted values of K and B separate the additional torque according to its neural and intrinsic components. In a highly influential paper, Stein et. al [13]

examined this separation by employing both the PKD and a pull test. Both Least Squares and an autoregressive model with external input were used to estimate K for each subject in the PKD. The intrinsic K was also computed from data in which subjects' shanks were pulled by experimenters from flexion to extension, while the applied torque was measured. They reported a similar intrinsic stiffness in the disabled and non-disabled subjects of 14-16 N-m/rad.

Separation of neurally controlled and intrinsic stiffness requires an additional stiffness term to be added to the model. In most studies, including our own, the intrinsic and active muscle stiffness are lumped into single stiffness term, with the angular displacement either with respect to a final position of the knee (zero vertical) or θ_{vt}. However there may be significant intrinsic stiffness that is relative to neither gravity nor the virtual trajectory [10, 14]. Therefore, the determination of a separate intrinsic spring set point term is crucial in achieving a more accurate approximation of the joint's biomechanics. This angle is not obvious in the trajectory data. The typical non-zero angle at the final resting position (θ_{FR}) seen in the trajectory, is the result of the intrinsic and gravitational stiffness moments (M_G and M_I respectively). M_I is defined as $K_I*(\theta-\theta_I)$, where K_I is the intrinsic stiffness and θ_I is its intrinsic set point. By revising equation 1 to differentiate between the active (K_a) and intrinsic stiffness, the equation of motion is modified as follows:

$$I\theta'' + B\theta' + K_a(\theta - \theta_{vt}) + K_I(\theta - \theta_I) = mgLsin(\theta) \qquad (3)$$

This holds true for subjects with and without neurological disability. To determine the intrinsic terms in the equation of motion, we applied a push-pull test [10] and a PKD on 4 subjects without disability (1 female and 3 male) and 2 female subjects with spasticity (CP) to evaluate their active and intrinsic knee stiffness parameters. The objective of this study is to obtain sound estimates of K_I and θ_I as they are important in providing plausible assessments of the level of impairment.

II. Methodology

A. Experimental Procedure

Four subjects without neurological disability and two subjects with disability (CP) were examined and presented in this paper. Data represent the knee's angular displacement (push-pull and PKD) and moments required to hold the shank in quasi-static positions (push-pull). Subjects were seated on table with the femur oriented horizontally and the shank allowed to hang freely over the edge. The torso was at an angle of 120˚ with respect to the thigh.

Data were collected using a single Ascension Technologies trakSTAR electromagnetic tracker at 100 f/s. The sensor was attached to the subject's shank to measure the angular displacement of the knee. The holding moment (M_H), exerted by the experimenter, was measured at 100 f/s with a force transducer (ATI-Mini 20/40) that was also attached to the subject's shank. The trackSTAR and force transducer data were filtered using a second order, low pass, zero lag, Butterworth filter with a cut-off frequency of 10 Hz.

B. Major Components of the Push-Pull Test Model

This test starts with the shank at its resting angle (θ_{FR}). It is then displaced with increments of 0.1 radian to maximum flexion. After that, the experimenter allows the shank to stabilize back to θ_{FR} and repeats the process to maximum extension. At each increment, the experimenter holds the shank static, maintaining a constant angle for approximately 5 sec. This represents a quasi-static equilibrium state at which there is negligible velocity and acceleration ($\theta''=\theta'=0$). The net holding moment (M_H) which maintains the shank in static equilibrium can be represented by Equation 4. Winter's anthropometric equations [15] were used with the body mass and shank length to calculate the gravitational torque (M_G). Output of the force transducer was used to compute M_H. M_I could then be calculated. Estimation of the term K_I is found by graphically calculating the slope between clusters on the M_I plot that represent the periods of static equilibrium. Clusters are non-linear over the entire range of possible knee motion, therefore, the central region in which K_I is quasi-linear was used. This range was chosen as the shank's resting angle ± 1 standard deviation of the PKD excursions. The angle at which M_I = zero or ($M_H = M_G$) is identified as θ_I.

$$M_H = M_G \pm M_I \qquad (4)$$

The shank's angular trajectory of as it is examined in the push-pull test is illustrated in Fig.1, where plateaus on the plot show the quasi-static holding of the shank.

C. Pendulum Knee Drop test (PKD)

This is a dynamic test to investigate the biomechanical parameters of knee joint and provide evaluation of the person's impairment. The shank hangs freely while subjects are seated on an examining table and are asked not intervene while evaluated. The experimenter administers the PKD test by lifting the examined shank to a specific angle then releases it, producing an angular trajectory as the limb falls due to gravity. The parameters K, B, θ_{vt} are extracted from the data using our Least Squares optimization model [9].

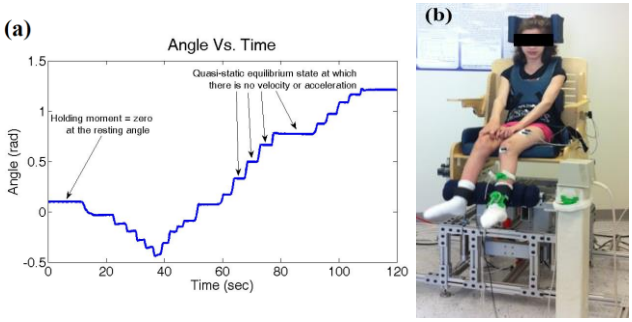

Fig. 1: (a) Example of knee angular position (Quasi-static push-pull test), (b) experimental set-up.

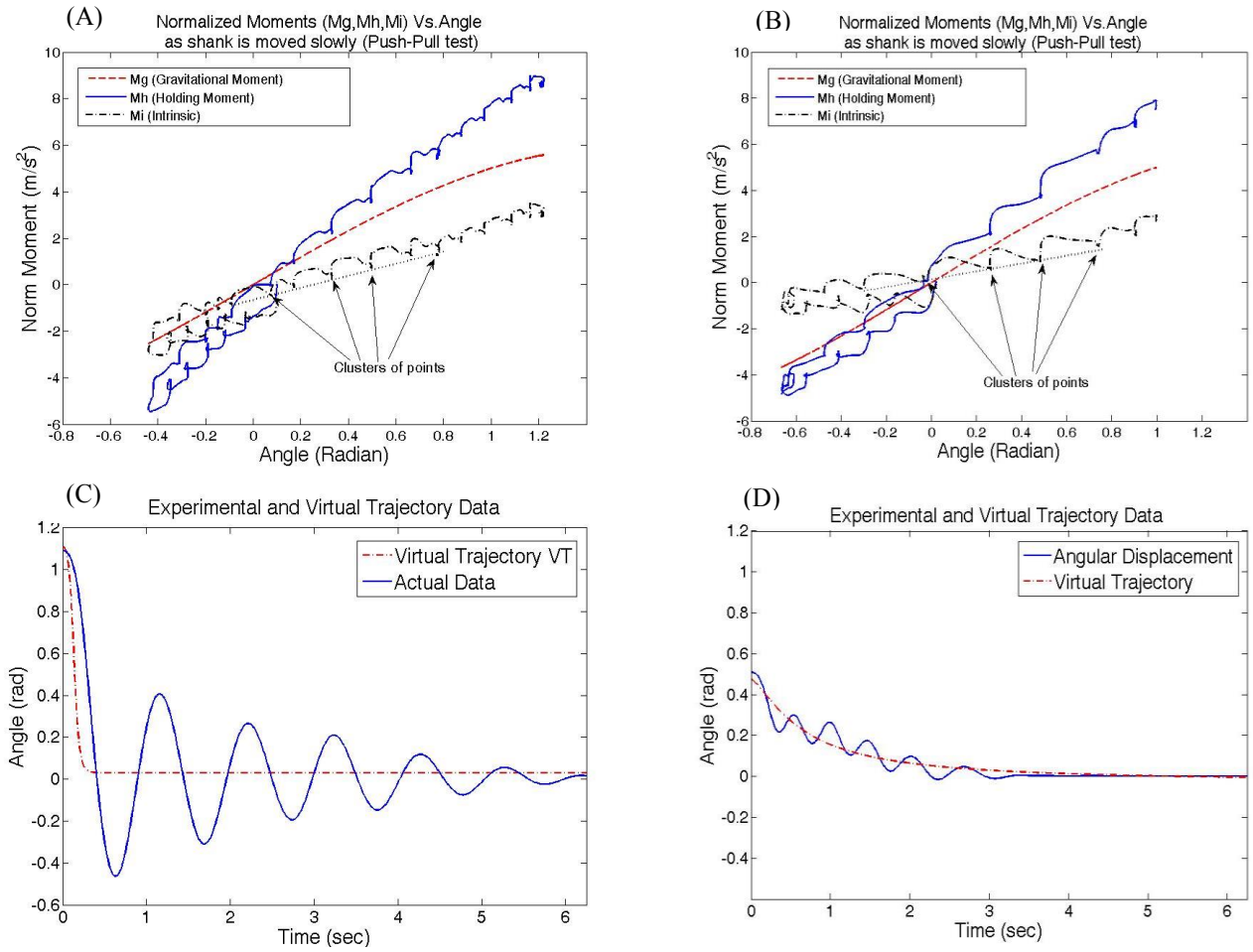

Fig. 2 (A) Illustrates an example of the gravitational, holding and the intrinsic as a function of angle (rad) for one subject without disability; (B) with disability. It is noticed that there are point clusters every ~ 0.1 rad representing the holding angles which are used in determining the intrinsic the stiffness of knee joint K_I. The neutral knee angle Θ_I can be graphically found by the intersection of M_G and M_H. The slope between the clusters points offers estimation of K_I. (C) Illustrates an example of the PKD data for one subject without disability; (D) with disability. In A and B, the rhythmic arcs present in the M_H and M_I plots represent shank movement, followed by quasi-static clusters of equilibrium points. The slope of M_I can therefore be computed between these adjacent clusters to provide values of K_I in relation to angle [10].

III. RESULTS

The optimization of PKD data of each subject without disability revealed lumped K values that are quite similar to the K_I values of the same subject found from graphical calculation in the push-pull. Very interestingly, these are also similar to the K_I values computed graphically for subjects with disability from the push-pull test. On the other hand, the optimized K values found in the PKD test for subjects with disability are approximately 19 times larger than the K values found graphically from the push-pull test.

To eliminate variability between both populations, the push-pull test data were normalized to the mass and length of the subject's shank. This produces comparable results in Fig.2 (A and B).

Table 1 The optimized parameters from the PKD and push-pull tests for both populations

Assessment	PKD				Push-Pull		Ratio
Parameters	Subject	θ_{VT}* (sec)	B (N.m.sec /rad)	K_{PKD} (N.m/rad)	θ_I (rad)	K_I (N.m/rad)	K_{PKD} / K_I
Individuals without disability	1	0.392	0.160	3.164	0.312	3.586	0.882
	2	0.511	0.618	4.820	0.328	1.777	2.712
	3	0.413	0.271	7.244	0.314	8.179	0.886
	4	0.340	0.745	3.429	0.051	2.719	1.261
Individual with disability	1	0.650	0.108	31.13	-0.035	1.687	18.45
	2	0.402	0.030	24.23	-0.045	1.222	19.80

***Time to the sigmoidal virtual trajectory's inflection point.**

IV. Discussion

The push-pull test used in this study shows a very interesting way of examining passive, intrinsic limb stiffness and can be used to allow dynamic techniques such as PKD to more effectively model spasticity.

The intrinsic stiffness of our spastic subjects has approximately the same magnitude as that of our non-spastic subjects. This is in agreement with Gordon et. al. [3] in which a pull test was used with the elbow joint of spastic and non-spastic subjects. The stiffness values were very similar for both groups when the angular velocity equaled 0, and consistent with the results discussed by Stein et. al. [13] where spastic and non-spastic subjects show similar stiffness during low velocity passive motion. This confirms that spasticity is truly a velocity dependent, and is not present in disabled subjects at rest or when joint velocity near zero.

Our estimates of intrinsic K are significantly lower than those reported by Stein et. al. This is explained by that paper's definition of intrinsic stiffness, which includes the joint's mechanical properties and gravitationally induced stiffness, and is therefore overestimated. Our evaluation on the other hand excludes the gravitational torque and reports only the contribution of mechanical tissue properties as the intrinsic stiffness. The Stein et. al. stiffness minus its respective gravitational term is of comparable magnitude to our findings.

This is evident when the ratios of holding to gravitational torques from the two studies are observed. The Stein et. al. ratio is close to 2:1, as compared to approximately 1.6:1 in our study. This small difference is likely due to the additional torque caused by inherent damping since the Stein et al data were collected at low velocity and not at static intervals.

This leads us to the conclusion that we can partition our previously lumped K as the sum of a neurally generated stiffness, K_a and K_I and complete a more accurate model defined in Equation 3.

Future work will include examining a larger population of subjects with spasticity and dystonia due to CP and subjects with spasticity due to other disabilities including stroke, SCI, TBI and MS. Additionally, more work is required in examining knee movement in the absence of gravity by orienting subjects horizontally.

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References

[1] Lance JW Symposium synopsis. In Feldman RG, Young RR, Koella WP eds. Spasticity: disordered motor control.Year Book Medical Publishers, 1980: 485-94.

[2] O'Dwyer, N. J., & Ada, L. (1996). Reflex hyperexcitability and muscle contracture in relation to spastic hypertonia. *Current opinion in neurology*, *9*(6), 451-455.

[3] Gordon, L. M., Keller, J. L., Stashinko, E. E., Hoon, A. H., & Bastian, A. J. (2006). Can spasticity and dystonia be independently measured in cerebral palsy?. *Pediatric neurology*, *35*(6), 375-381.

[4] Wartenberg, R. (1951). Pendulousness of the legs as a diagnostic test.*Neurology*, *1*(1), 18-18.

[5] Lin, D.C.; Rymer, W.Z.; , "A quantitative analysis of pendular motion of the lower leg in spastic human subjects,"*Biomedical Engineering, IEEE Transactions on* , vol.38, no.9, pp.906-918, Sept. 1991 doi: 10.1109/10.83611.

[6] Fowler, G. E., Nwigwe, A. I., & Wong Ho, T. (2000). Sensitivity of the pendulum test for assessing spasticity in persons with cerebral palsy. *Developmental Medicine & Child Neurology*, *42*(03), 182-189.

[7] Le Cavorzin P, Poudens SA, Chagneau F, Carrault G, Allain H, Rochcongar P, "A Comprehensive Model of Spastic Hypertonia Derived from the Pendulum Test of the Leg," Muscle Nerve, vol. 24, no. 12, pp. 1612-21, December 2001.

[8] Fee, J.W. & Foulds, R.A. Neuromuscular Modeling of Spasticity in Cerebral Palsy. IEEE Transactions on Neural Systems and Rehabilitation, 12(1), 55-64. (2004).

[9] Simon, D., Androwis, G. J., & Foulds, R. A. (2011, April). Equilibrium point model of knee joint spasticity. In *Bioengineering Conference (NEBEC), 2011 IEEE 37th Annual Northeast* (pp. 1-2). IEEE.

[10] Androwis, G. J., Simon, D., & Foulds, R. A. (2012, March). Assessment of passive knee stiffness and set point. In *Bioengineering Conference (NEBEC), 2012 38th Annual Northeast* (pp. 133-134). IEEE.

[11] Androwis, G. J., Foulds, R. A., & Jewaid, D. I. (2013, April). The Neural Contribution to Passive Joint Movement in Individuals with Cerebral Palsy. In *Bioengineering Conference (NEBEC), 2013 39th Annual Northeast* (pp. 223-224). IEEE.

[12] Feldman, A.G. (2011) Space and time in the context of equilibrium-point theory. New York, Wiley Interdiscip Rev: Cogn Sci 2:287-304.

[13] Stein, R. B., Zehr, E. P., Lebiedowska, M. K., Popovic, D. B., Scheiner, A., & Chizeck, H. J. (1996). Estimating mechanical parameters of leg segments in individuals with and without physical disabilities. *Rehabilitation Engineering, IEEE Transactions on*, *4*(3), 201-211.

[14] Bohannon, R. W., Harrison, S., & Kinsella-Shaw, J. (2009). Journal of NeuroEngineering and Rehabilitation. *Journal of neuroengineering and rehab*.

[15] Winter, D. A. (1979). *Biomechanics of human movement* (pp. 151-152). New York: Wiley.

[16] Burne, J. A., Carleton, V. L., & O'dwyer, N. J. (2005). The spasticity paradox: movement disorder or disorder of resting limbs?. *Journal of Neurology, Neurosurgery & Psychiatry*, *76*(1), 47-54.