

Constraints imposed by the lower extremity extensor synergy in chronic hemiparetic stroke: Preliminary findings

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Abstract— In the present manuscript we implemented the MultiLEIT, a lower extremity isometric torque measurement device to quantify spontaneous joint torque coupling during maximal torque generation in the paretic leg of in chronic hemiparetic stroke. We quantified extension/adduction coupling (coincident with the clinical extension synergy) during the generation of hip extension and ankle plantarflexion maximum voluntary torques. Subjects were then instructed to generate torques outside the synergy by combining hip extension+ hip abduction or ankle plantarflexion + hip abduction. During the hip dual task, the paretic hip torques were significantly different from those measured in the non-paretic and control leg ($F = 22.9719$, $p = 0$) and resulted in the inability to generate torques outside the extensor synergy patterns. During the dual ankle/ hip task, the paretic extremity generated significantly smaller hip abduction torques compared to controls and to the non-paretic extremity ($F = 15.861$, $p = 0$). During this task the paretic extremity was capable of neutralizing the spontaneous adduction torque and generate a net albeit small abduction torque. Results may indicate an increased descending drive from brain stem pathways, particularly during hip extension, responsible for constraints in generating hip abduction torques after stroke.

I. INTRODUCTION

Stroke is the leading cause of long-term adult disability in the United States with almost 800 000 new and recurring strokes occurring each year¹. Impairment in the lower extremity manifests as asymmetrical distribution of weight during standing, rotation of the longitudinal axis of the body towards the unaffected side, impaired control of postural stabilization², decreased ability to withstand perturbations³, impaired transition between the phases of gait (torque weakness in specific degrees of freedom-DOF) and impaired foot clearance and forward displacement of the paretic extremity⁴⁻⁸. Yet, the underlying mechanisms responsible for these impairments have been sparsely researched.

Previous studies have aimed to quantify joint-torque coupling possibly due to altered supraspinal descending drive to the lower extremity⁹⁻¹⁷. However, results have been contradictory. On one hand, studies of isometric lower extremity joint-torque coupling in cerebral palsy quantified stereotypical spontaneous coupling between hip and knee

extension¹². In contrast, studies in individuals post-stroke have not obtained consistent findings with regards to the expression of abnormal joint torque coupling patterns. The use of an instrumented Lokomat^{8, 9} proved to be a novel approach to quantify spontaneous coupling between hip adduction and hip and knee extension^{8, 9, 12} (coincident with what is described clinically as the “extension synergy”: coupling of hip extension/adduction with knee extension and ankle plantarflexion⁴); however, other studies found only weakness in specific DOFs but no stereotypical joint torque coupling patterns across subjects^{10, 11}. The behavior of the ankle as part of these spontaneous coupling patterns has not been quantified to date.

We will describe a new Multi-joint Lower Extremity Isometric Torque measurement device, the MultiLEIT. This system can accurately quantify lower extremity joint torque coupling strategies during voluntary maximal and submaximal torques at the hip, knee and at the ankle (tasks include hip abduction – HABD, hip adduction – HADD, hip flexion – HFLEX, hip extension – HEXT, knee flexion – KFLEX, knee extension – KEXT, ankle dorsiflexion – ADF and ankle plantarflexion – APF). We will employ the MultiLEIT in the paretic and non-paretic lower extremity of post-stroke individuals and in the right leg of healthy, age matched control individuals. If abnormal stereotypical coupling torque patterns in the paretic extremity are observed, the ability of test participants to combine joint torques outside these patterns will be investigated. This will determine whether post-stroke individuals are indeed constrained to the coupling patterns defined clinically: the extensor or flexor synergies. We hope to provide a consensus about the existence of abnormal stereotypical joint torque coupling synergies in the paretic lower limb.

Accurate characterization of joint torques and associated coupling patterns could aid the development of therapies to address complications that result from constraints in the ability to generate certain torque coupling patterns post-stroke.

II. METHODS

A. The MultiLEIT

The MultiLEIT measures all isometric torques from hip to ankle in a single leg, with the intention to quantify inter- and intra joint torque couplings in individuals after stroke. The setup provides information about the strategies used by subjects under well-controlled static conditions.

The MultiLEIT consists of a rigid aluminum structure instrumented with two 6-DOF force/torque sensors

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positioned above the knee and below the ankle, as shown in figure 1. The 12-DOF measurements are converted through

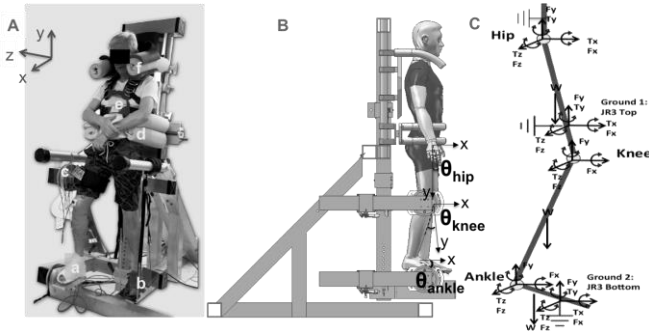


Figure 1. The MultiLEIT. A) Experimental setup. (a) Rigid attachment to bottom sensor. (b) Adjustable support for non-tested leg (bungee cord attached to placing surface). (c) Adjustable thigh cuff and top sensor. (d) Pelvic clamps. (e) Trunk harness (f) Shoulder clamps. B) Definition of joint angles. C) Free body diagram for equilibrium equations.

Jacobian transformations to torques in the hip, knee and ankle. The 6-DOF force/torque sensors have a $\pm 2000\text{N}$ force range in F_x and F_y and $\pm 4000\text{N}$ F_z range and a torque range of $\pm 400\text{Nm}$ in x , y and z (Model 75E20A4, JR3 Inc., Woodland, CA) to accommodate the 95th percentile maximum extension torque that can be generated at the hip by males (419Nm^{18}). Raw forces and torques measured from the sensors are transformed to joint torques and displayed on a monitor to provide visual feedback for the subject. Based on the assumption of a static system, the principle of virtual work for static equilibrium¹⁹ was used to calculate the joint torques as a function of the forces and torques measured at each of the load cells:

$$\boldsymbol{\tau} = \mathbf{J}^T(\mathbf{q}) [\mathbf{F}^T \mathbf{M}^T] \quad (\text{Equation 1}).$$

\mathbf{J}^T is the Jacobian transformation matrix determined by the geometry of the system. If \mathbf{F} and \mathbf{M} are the x , y and z forces and torques measured by the sensor, joint forces and torques are defined as:

$$\begin{bmatrix} F_x \\ F_y \\ F_z \\ M_x \\ M_y \\ M_z \end{bmatrix} = \begin{bmatrix} \mathbf{R}(\theta) & \mathbf{zeros}(3) \\ \begin{bmatrix} 0 & -dz & dy \\ dz & 0 & -dx \\ -dy & dx & 0 \end{bmatrix} & \mathbf{R}(\theta) \end{bmatrix} * \begin{bmatrix} F_{xJR3} \\ F_{yJR3} \\ F_{zJR3} \\ M_{xJR3} \\ M_{yJR3} \\ M_{zJR3} \end{bmatrix} \quad (\text{Equation 2}).$$

$\mathbf{R}(\theta)$ is the 3×3 rotation matrix defining the orientation of the sensor coordinate system with respect to the joint coordinate systems and d_i for $i=x,y$ or z is the distance between the geometrical origin of the sensor and the origin of each joint center of rotation in the corresponding direction (figure 1).

B. Experimental Protocol

Five chronic post-stroke individuals were recruited for this study from the Clinical Neuroscience Research Registry. Individuals post-stroke should have had a unilateral brain lesion from a single stroke at least one year prior to participation in this project and no muscle tone abnormalities, sensory impairment, severe wasting or contracture, severe cognitive or severe concurrent medical problems. Three age and gender matched healthy control individuals were recruited from the community. All experimental protocols were approved by Northwestern

University's Institutional Review Board.

Subjects were fitted into the MultiLEIT and their trunk was strapped using a skydiving harness. The leg was fitted to a memory foam padded attachment cuff located above the knee. The foot was attached to the setup using fiberglass cast. Joint angles for the tested leg were set to 30° hip and knee flexion, 0° ankle flexion and 10° hip abduction. The contralateral lower extremity was placed on an elastic sling to prevent subjects from pushing down with the non-tested leg while keeping the leg from dangling. The sling provided both mechanical and neural decoupling of both legs, thus preventing the non-tested lower extremity from driving the tested lower extremity due to inter-limb coupling⁷. Subjects' pelvis, trunk and shoulders were fitted into the setup to minimize movement.

Subjects were asked to perform maximum voluntary torques (MVTs) in either H ABD, H ADD, H FLEX, H EXT, K FLEX, K EXT, A DF or A PF for a total of 8 tasks (2 directions per DOF). MVTs were performed in randomly ordered blocks to discard learning effects. Primary (voluntary instructed) torques and secondary (concurrent spontaneous) torques were computed online based on the raw forces and torques measured from the two 6-DOF load cells. Visual feedback of the DOF being maximized was provided. No instructions were given for any of the other DOFs. In each trial, the subject was asked to start in a relaxed state, ramp up to maximal torque production and sustain it for 2 seconds. A trial was considered successful if the MVT plateaued for at least 250ms. Two trials with MVT values within 10% of each other, with the second trial being of smaller magnitude than the first trial were required to ensure MVTs were achieved. Only two MVT trials were required to avoid fatigue.

We identified extension/adduction coupling during hip extension MVT and ankle plantarflexion MVT on the paretic extremity of post-stroke individuals. Thus, for the dual DOF task, all individuals were asked to combine maximum H ABD with 25, 50 and 75% of their H EXT torque and maximum H ADD torque with 25, 50 and 75% of their A PF torque. These values were mapped on a visual display to provide feedback to the subjects during the task. Four post-stroke individuals and two control individuals completed the tasks.

C. Data processing and analysis

Data was acquired via a National Instruments Single Differential Legacy Device (PCI 6031E, NI, Austin, TX). Digital signal processing and data transformation was performed in a custom Matlab graphical user interface (Mathworks Inc., Natick, MA). Data were acquired for 8s at a 1000Hz sampling frequency. Force and torque data were filtered using a 250ms moving average window.

Joint torques were calculated using Jacobian transformation matrices applied to the smoothed force and torque data. The maximum torque in the instructed direction was obtained from the 2s where the torque magnitude was held constant. The time window when the Maximum Voluntary Torque (MVT) was generated was then identified

and the spontaneous torques in the other DOFs were extracted for the same time window. Spontaneous torques were normalized to the maximum torque in that DOF and direction obtained during the entire experiment, to express spontaneous coupling as a percentage of the maximum

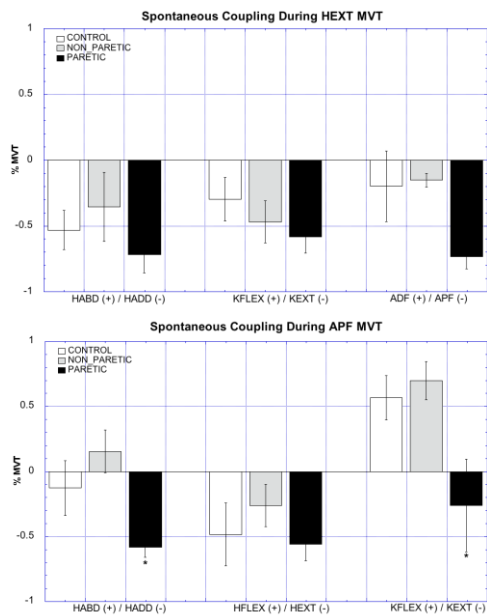


Figure 2: Normalized spontaneous joint torque coupling. HEXT (top) and APF (bottom). Control: white, non-paretic: grey, paretic: black. * $p < .05$ torque. All statistical analyses were run in SPSS (version 21, SPSS Inc., Chicago, IL). Data was tested for normality using Q-Q plots. Homogeneity of variance was tested using Levene's Statistic. One-way ANOVAs for each task was run with group (control, paretic, non-paretic) as the independent variable and spontaneous secondary torque as the dependent variable. Post-hoc Bonferroni analyses were run at $p = 0.05$ significance. For the dual DOF task, we performed two-way ANOVAs with group and level (hip extension or ankle plantarflexion) as independent variables and percentage of hip abduction as the dependent variable. Post-hoc comparisons were done using Bonferroni analyses.

III. RESULTS

A. Spontaneous Joint Torque Coupling

None of the MVT tasks in the abduction or flexion directions elicited a grouped flexor synergy. In contrast, coupling in accordance to the extensor synergy (extension/adduction coupling) was quantified during hip extension MVT in the paretic and non-paretic extremity of post-stroke individuals. Extension/adduction coupling was also observed in the control lower extremity when maximizing hip extension torque. No significant differences in spontaneous coupling were quantified across groups (figure 2).

Extension/adduction coupling was observed during ankle plantarflexion MVT only on the paretic extremity of post stroke individuals. Greater hip adduction torque was observed in the paretic extremity ($F = 5.372$, $p = 0.022$).

Greater paretic knee extension torque was also quantified ($F = 4.478$, $p = 0.033$) as shown in figure 2.

B. Voluntary hip extension/abduction coupling

For the dual submaximal HEXT + HADD MVT, there was

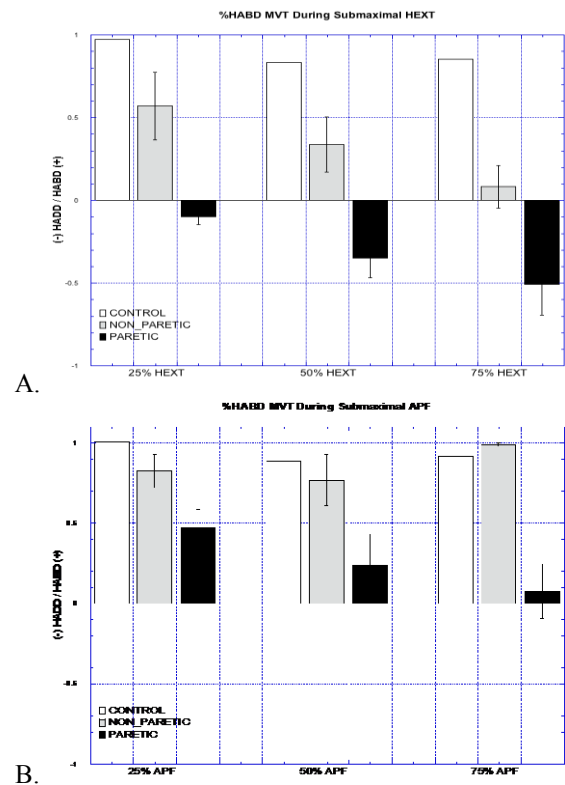


Figure 3. Results dual DOF task. A) 25, 50 and 75% HEXT + maxHADD torque. Significantly smaller HADD torque was observed on the non-paretic extremity compared to controls. On the paretic extremity, spontaneous HADD overcame net HADD torque. B) 25, 50 and 75% APF torque + maxHADD. No significant differences were observed between controls and non-paretic. Significantly decreased paretic HADD. (averaged 2C, 4P, 4NP)

a significant main effect of group on HADD/ADD level ($F = 29.719$, $p = 0.000$, post-hoc Bonferroni comparisons showed significant differences across all groups $p < 0.05$). There was no significant main effect of hip extension level or of the interaction group*HEXT (Figure 3-A).

C. Voluntary ankle plantarflexion/hip abduction coupling

For the dual submaximal APF + HADD MVT task, there was a significant main effect of group on HADD/ADD level ($F = 15.861$, $p = 0.000$, post-hoc Bonferroni comparisons showed significant differences between control and paretic $p = 0.004$ and non-paretic and paretic $p = 0.000$). There was no significant main effect of ankle plantarflexion level or of the interaction group*APF. Figure 3-B shows these results.

IV. DISCUSSION

In the present manuscript, we provided an accurate quantification of lower extremity joint torque coupling during standing. Joint torque coupling in the form of the extensor synergy was observed during HEXT and APF MVT on their paretic extremity. Subjects were then instructed to "break out" of the synergy by combining

these torques with HADD MVTs. Subjects were capable of overcoming the synergy during APF but not during HEXT. This may provide insights into the neural drive to muscles during these tasks: hip extension, is a more postural task, therefore it may potentially involve greater brain stem drive which causes HADD coactivation. In contrast, during APF, subjects may not use brainstem mediated postural motor pathways thus allowing for better isolated HADD activation.

Joint torque coupling strategies quantified during HEXT MVT generation coincided with the coupling patterns described in the extensor synergy. However, coupling of extension/adduction was observed during HEXT MVT across all lower extremities (control, non-paretic and paretic). This finding may just indicate the fact that when trying to maximize hip extension torque, both control and post-stroke individuals used the same strategy, which can be a result of mechanically coupled hip extensors and adductors. These results do not contradict the existence of a pathological extensor synergy. We hypothesize that due to the similarities in coupling during HEXT MVT between control and stroke subjects during may be because of mechanical coupling of hip extensors with adductors, which in controls can be overcome by increasing corticospinal drive. The upright posture in which subjects were tested, in addition to increased drive to postural muscles and increased brain stem drive²⁰ may be accounted for the constraint in post-stroke individuals during HEXT MVT.

Our results for the dual DOF task demonstrated that the extension/adduction coupling may in fact be a consequence of being constrained to the use of bulbospinal pathways post-stroke individuals were control subjects were not, presumably because of the use of corticospinal projections. Even for 25% HEXT, post-stroke individuals were only capable of minimizing the hip adduction torque without generating a net hip abduction torque. This may have functional implications for standing balance and gait: when an increased HEXT torque is required, this will generate HADD torques that cannot be volitionally overcome, thus, decreasing frontal plane stability. It is also worth noting the fact that the non-paretic extremity was also affected by the extensor/adduction coupling: subjects were not able to generate a HADD MVT but only generated on average 50% HADD even for 25% HEXT. This may point to bilateral changes in descending drive that can possibly affect the ipsilateral extremity albeit to a lesser extent the paretic leg.

During APF MVT, the coupling strategies and muscle activation patterns used between groups were significantly different. The greater amount of coactivation observed on the paretic extremity may indicate the fact that subjects post-stroke may sacrifice independent joint control to maximize distal torque by increasing all descending drive from remaining corticospinal pathways and brain stem pathways which are more diffuse and innervate multiple muscles simultaneously²⁰. However, in the dual DOF APF + HADD MVT task, the degree of

constrain to the synergy was less. Even though the HADD MVT could not be achieved, the net torque generated was in the HADD direction and even for the highest APF level, subjects were capable of neutralizing the spontaneous HADD torque. This may indicate use of remaining corticospinal pathways especially when activating a more distal joint. It is also interesting to note that the non-paretic limb behaves very similar to that of the control subjects who participated in this study. These results suggest that physical rehabilitation exercises to strengthen lower extremity muscles should be performed combining degrees of freedom away from the coupling patterns that involve HADD during functional leg tasks.

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