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Impaired Lower Limb Muscle Synergies Post-Stroke

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Abstract—**Given the impaired motor output of the lower limb post-stroke, we argue that cortical damage alters the modular control structure underlying muscle activation patterns during both static and dynamic tasks. Muscle synergies were extracted from EMG collected during isometric hip torque production performed by chronic stroke and control subjects. The stroke group presented a reduction in muscle synergies, consistently displaying only two of the four characteristic synergies identified in the control group. These findings do not support a generalized co-contraction of muscles underlying motor impairments. Our data also indicated that the activation coefficients of the muscle synergies retained by the stroke group were altered. Finally, muscle synergies extracted from the isometric torque production tasks were able to account for a large portion of the variance in the gait EMG at similar limb postures in both subject groups. We therefore propose that muscle activation patterns during the swing phase of gait are constrained by the set of muscle synergies identified under the isometric conditions.**

I. Introduction

FOLLOWING a neurological injury, the ability of the central nervous system to control individual degrees of freedom is often compromised [1-3]. There is compelling evidence that human movement may be produced through the flexible combination of a limited set of muscle activation patterns, or muscle synergies, which are differentially weighted according to task [4-6]. As defined here, a muscle synergy is composed of a fixed balance of activations across muscles. Effectively, muscle synergies may simplify motor control by reducing the dimensionality (degrees of freedom) of the control problem [7, 8]. The central thesis of this work is that cerebrovascular accidents (CVA) alter the number and composition of muscle synergies available for control of lower limb motor tasks, resulting in altered gait patterns of muscle activation.

II. Experimental Methods

A. Subjects

Nine control and nine stroke subjects were recruited. The primary inclusion criteria for stroke were evidence of a unilateral brain lesion and the ability to stand and ambulate without assistance with a gait velocity of less than 0.8 m/s. Exclusion criteria included severe osteoporosis; contracture limiting the range of motion; cardiorespiratory or metabolic diseases; unhealed decubiti; persistent infection; and significant cognitive or communication impairment that could impede the understanding of the study procedures. Subjects gave informed consent, as approved by the Institutional Review Board of Northwestern University.

B. Electromyography (EMG)

EMG was recorded from eight muscles acting at the paretic hip and knee: vastus medialis (VM), vastus lateralis (VL), rectus femoris (RF), adductor longus (AD), tensor fascia lata (TFL), semitendinosus (MH), biceps femoris (LH), and gluteus medius (GM). Surface electrodes were used to record from superficial muscles (VM, VL, & LH), while intramuscular electrodes were used for the remaining muscles. EMG signals were digitized at 1,000Hz, low-pass filtered (100Hz cutoff), rectified, filtered with a 4^{th} order Butterworth filter (6Hz cutoff) and normalized to maximum voluntary contraction.

C. Experimental Paradigm

Muscle activation patterns during isometric hip torque production were measured in standing postures using a previously described experimental paradigm [3]. Briefly, the pelvis and lower limbs were secured to a motorized exoskeleton (Hocoma, Switzerland) via cuffs instrumented with six-degree-of-freedom load cells (JR3). The joints of the exoskeleton were locked with mechanical stops to create an isometric setup in the toeoff posture (15^o hip extension and 45^o knee flexion). The nonparetic limb was placed in full extension and supported the body during the experiment.

Prior to the initiation of a trial, subjects were instructed to relax and the load cells were zeroed. Subjects were asked to produce maximum voluntary isometric torques (MVIT) in eight directions (flexion, extension, abduction, adduction, flexion\abduction, flexion\adduction, extension\abduction, and extension\adduction) while receiving visual feedback of the magnitude and direction the hip torque. Then subjects were instructed to produce the hip torque (40% MVIT) in the eight target directions and hold for 200ms. Five repetitions of the eight target directions were presented in random order for a total of 40 targets. EMG data during treadmill walking at 2.0 kmph was also collected.

III. Data Analysis

We propose that EMG patterns from the isometric task may be produced through the weighted combination of a small number of muscle synergies [9, 10]. That is, a smaller number of neural drives than individual muscles may

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account for most of the observed EMG for a given task. Each synergy specifies fixed activations of different muscles within the limb, and multiple synergies may be active simultaneously. The EMG pattern under isometric conditions can be described as the linear combination of N synergies:

$$\vec{x} = \sum_{i=1}^{N} c_i \vec{w}_i + \varepsilon \quad (1)$$

where $\vec{x}$ is the M-dimensional muscle data vector, $\vec{w}_i$ an M-dimensional vector of the i^{th} muscle synergies (i = 1 , .. N, where N is the number of synergies), c_i is the scalar weighting coefficient of the i^{th} muscle synergy, and $\vec{\varepsilon}$ is the M-dimension noise vector. Eq. (1) written in a matrix form is

$$X = WC + E \quad (2)$$

where X is the (M x T) matrix of EMG data from M muscles and T observations, W is the muscle synergies (M x N), C is the activation coefficients (N x T), and E is the error (M x T).

Principal component analysis (PCA) and independent component analysis (ICA) were used to achieve uncorrelated synergies activated by a low number of independent sources [11, 12]. PCA reduced the dimensionality of a dataset by only retaining the components associated with the most variance (e.g. >90%) [13]. ICA was then used to decompose the reduced dimensionality dataset into muscle synergies (W) and activation coefficients (C) [14]. While the test trials were performed at random, the data was reordered *post hoc* by target direction. Activation coefficients may be interpreted as spatial weighting toward a particular target direction as displayed in polar plots. EMG from 75% of the trials was used to estimate W and cross-validation was achieved using Variance Accounted For (VAF) for the prediction of the remaining 25% of the trials [5, 8].

A. Muscle Synergies (W)

To investigate motor control behavior in the intact system, similarities in muscle synergies across control subjects were assessed. Similarity is defined as scalar products of synergies of unit length greater than 0.90 [15]. To determine if a synergy may be considered "characteristic" of the control group, similarity at random was identified across subjects. Random permutations of the muscle loadings within the synergies were created for each subject and the frequency of similarity of the random synergies across subjects was determined. If the scalar product of two synergies is greater than "similarity at random", the two synergies are classified as characteristic synergies of the control group. Groupings of similar synergies were also made in the stroke group using the same procedure. Systematic comparisons of characteristic stroke muscle synergies to the characteristic synergies of the control group were made by calculating the scalar product of the synergies of unit length. Again, values of greater than 0.90 were considered similar.

B. Activation Coefficients (C)

Activation coefficients from similar muscle synergies were averaged across subjects and compared between groups. Comparisons of activation coefficients were made using linear regression of the mean values ordered by target direction. An $r^2 > 0.55$ indicates similar recruitment of a given muscle synergy across groups [16].

Finally, EMG data observed during treadmill walking was used to determine if the estimated muscle synergies are indeed a reflection of an intrinsic motor control structure that is task independent. Walking EMG data at the toeoff posture was predicted using the synergies derived from the isometric tasks at the same limb posture. The upper bound of the proposed prediction was defined by the step to step variability of the EMG while the lower bound was defined by the VAF from random permutations of the muscle loadings within the synergies.

IV. RESULTS

Four muscle synergies were sufficient to account for ~90% of the variance in the eight-muscle EMG dataset for each subject in both groups, effectively reducing the control problem dimension by half. This number was significantly greater than the VAF by the random permutations of muscle loadings within each synergy ($p<0.0001$).

A. Muscle Synergies (W)

The stroke group did not display all the characteristic muscle synergy types found in the control group. Four characteristic muscle synergy types were observed across subjects in the control group and are described as the abductor, flexor/abductor, adductor, and the extensor synergies. Subject-specific examples of these characteristic synergies along with the spatial distributions of their activation coefficients can be found in Figure 1. Specifically, five control subjects displayed the flexor/abductor and extensor synergies, and four control subjects displayed the adductor and abductor synergies. The remaining muscle synergies could not be grouped with greater frequency than observed by chance.

Muscle synergies of the stroke group were more diverse than the control group. Only two of the four characteristic synergy types presented with frequency greater than expected by chance in the stroke group - the flexor/abductor and adductor synergies, see W_3 and W_1 in Figure 1, respectively. Six subjects displayed the flexor/abductor synergy and four subjects displayed the adductor synergy.

Muscle synergies not classified into the characteristic synergies categories were assessed for co-contraction between muscles acting in the hip frontal plane and the knee sagittal plane, according to the previously reported abnormal torque couplings [3]. Specifically, coupling of hip adductors (AD) with knee extensors (VM, VL, or RF) (muscle loading >0.10) and hip abductors (GM or TFL) with knee flexors (MH or LH) (muscle loading >0.10) were sought. Five stroke subjects displayed either type of across-joint muscle coupling.

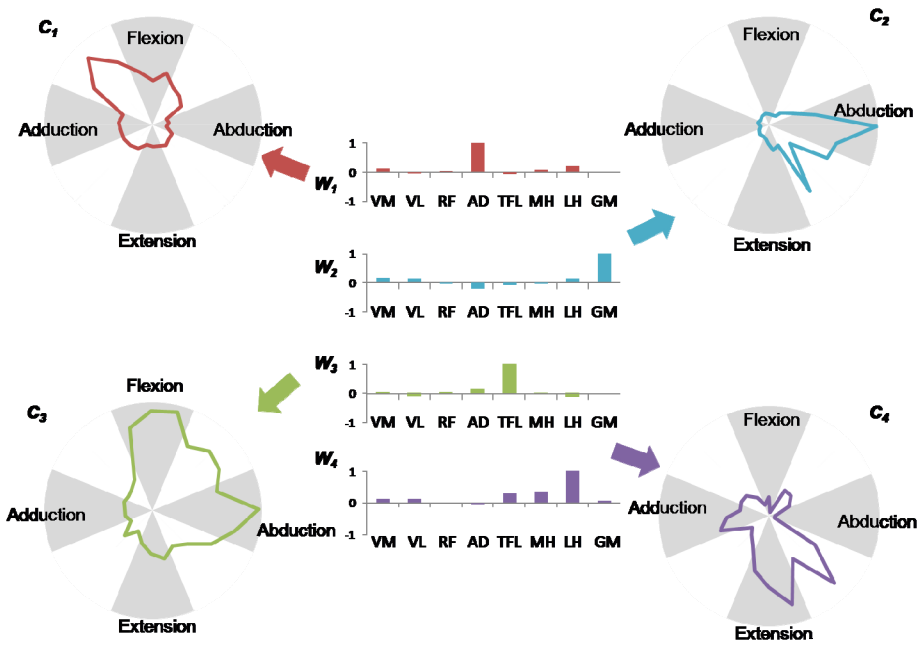

Figure 1: Subject-specific examples of the characteristic synergy types W_i from the control group and the associated activation coefficients C_i. The muscle synergies W_i show the fixed activations of the eight muscles. Due to the *post hoc* re-ordering of the EMG matrix, the activation coefficients C_i may be interpreted as spatial weighting toward a particular target direction. These polar plots display the mean coefficients over 20 data point bins as a function of target direction. Note that the target directions are discrete directions but are presented here as a continuum.

B. Activation Coefficients

The activation coefficients of some muscle synergies retained by the stroke group were altered. The mean activation coefficients for the adductor synergy are statistically similar across groups (r^2 = 0.76); however, the mean activation coefficients of the flexor/abductor synergy are not similar (r^2 = 0.22), see Figure 2.

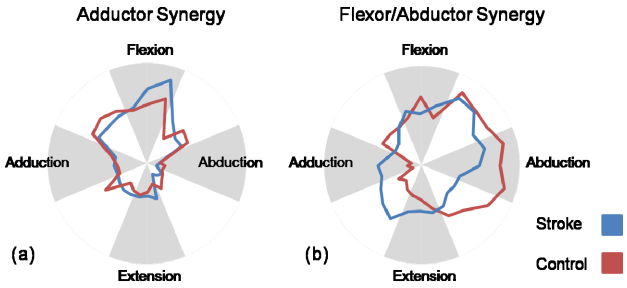

Figure 2: Mean spatial distribution activation coefficients for the muscle synergies consistent between groups.

Muscle synergies extracted from the isometric torque production tasks were able to estimate a large portion of the variance in the gait EMG at similar lower limb postures. The estimation was highly variable across subjects. In the stroke group, the isometric synergies from the toeoff posture could predict 66.7% (SE 9.3) of variance in the toeoff EMG data, while in the control group, the isometric synergies could predict 72.4% (SE 7.0) of variance in the walking EMG data. These values were significantly greater than those of the random synergies (49%, $p<0.008$, paired t-test on the pooled group data). Additionally, when placed in the context of step-to-step variability, relative performance of the synergies improves. The similarities of the EMG patterns step-to-step were 88% and 86% in control and stroke groups, respectively. Therefore the 66.7% VAF in the stroke group represents roughly 78% of variance that one would be expected to capture.

V. Discussion

The goal of this work was to quantify the influence of stroke-mediated cortical damage on lower limb motor control using muscle synergy analyses. Our findings suggest that a modular and hierarchical control structure with similar dimensionality can be used to describe motor output before and after a neurological injury. The elements contained within this structure, however, were altered. Stroke subjects were limited in the fundamental building blocks of muscle activations expressed by the intact motor system, as well as in the manner in which they were recruited. Motor templates with selective activation of hip musculature were present in individuals post-stroke in addition to synergies with across-joint co-activation. The across-joint co-activation synergies were consistent with abnormal across-joint torque coupling in the paretic lower limb [3]. The generalizability of the isometric muscle synergies was tested by estimating motor outputs associated with walking behavior. Muscle activations at select points of the gait cycle could be predicted with muscle synergies estimated from isometric data. These findings suggest that the isometric target matching paradigm proposed in this study was sufficient

to extract the fundamental muscle synergies available to both the healthy and impaired motor systems.

The similar number of synergies in the healthy and impaired motor systems suggests that the proposed modular structure of control is preserved after neurological injury. This is consistent with previous investigations in spinal cord injured and healthy control subjects [17] that identified similar dimensionality across groups during locomotion. While the exact neuro-physiological correlates of these synergies are unknown, similar muscle synergies have been observed in spinalized and brainstem frogs [12]. Thus it is tempting to suggest that the observed muscle synergies may be a manifestation of neural states at the spinal level [9, 10].

While the dimension of the motor control structure was similar across groups, our data indicated that only a sub-set of muscle synergies was present in both groups. Synergies featuring individual hip frontal plane muscles (AD, GM, or TFL) were shared templates across groups**.** However, unlike the control group, stroke data did not display muscle synergies dominated by hip flexor nor extensor muscles. The inability to isolate sagittal plane hip muscles during a hip torque generation task may contribute to the impaired sagittal plane kinematics observed during overground walking post-stroke [18, 19]. Our data also revealed the presence of abnormal co-activation synergies. Specifically, data from five of the nine stroke subjects showed muscles synergies combining knee extensors with hip adductors, a synergistic action consistent with previously observed torque coupling [3]. This class of synergies was infrequent in the control group (one of nine subjects). Taken together, our data indicate that the EMG patterns observed during the target matching task can be largely derived from two primary sets of synergies: selective and co-activated templates. These findings contrast with the traditional proposal that impaired motor activities following stroke are largely constrained by generalized co-contraction [20].

The change in the motor control options may stem from cortical reorganization post-stroke. Although only subjects with cortical CVA were examined, lesion size and shape may have contributed to the subject to subject variability. Specifically, the high variability in muscle synergies displayed by the stroke group suggests a strong subject-specific influence of neurological damage.

It has been proposed that some muscles synergies may control basic biomechanical functions of the limb, while other task-dependent synergies may meet unique biomechanical requirements of a specific motor output [5]. In this study, muscle synergies extracted from simple isometric behaviors were shown to estimate a large portion of the variability in EMG observed during treadmill walking. A plausible interpretation of these findings may be that muscle activation patterns observed during the swing phase of gait are constrained by the set of muscle synergies identified under the isometric conditions. To impact gait dysfunction, it may be possible to design novel isometric training protocols that can be administered during the pre-ambulatory phase of recovery to limit formation of disruptive linkages identified by the abnormal muscle synergies observed in this study.

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