

Improving impaired intermuscular coordination after stroke through synergy-guided human-machine interaction: a pilot study

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Abstract— Stroke survivors often experience motor impairments that include abnormal intermuscular coordination, which negatively affects individualized joint control. This lack of individualization is one of the fundamental motor control problems that largely explain voluntary movement impairment after stroke. Over the last decade, the concept of muscle synergy, here defined as a consistent ratio of muscle co-activation across muscles necessary to perform motor tasks, has been effectively applied to characterize intermuscular coordination following stroke. Developing non-invasive neurorehabilitation strategies to improve altered muscle synergy holds promise for advancing rehabilitative therapies; however, it remained largely unexplored. Here, we hypothesize that stroke survivors can improve the individualized activation of synergistic muscle coordination to decrease motor impairment through human-machine interaction guided by customized muscle synergies. In this study, three chronic post-stroke participants went through six weeks of a synergy-guided exercise that focused on independent control of each synergy identified from the major arm muscle activation. This study revealed three main findings: 1) chronic post-stroke participants could improve independent activation of synergistic muscle groups in the arm; 2) it was possible to modulate abnormal intermuscular coordination to resemble the intermuscular coordination patterns found in age-matched individuals; and 3) improving intermuscular coordination patterns showed the potential for reducing motor impairment. These results suggest that our novel synergy-guided exercise through human-machine interaction can improve stroke-induced neuromuscular coordination to reduce motor impairment.

Keywords—muscle synergies, synergy-guided training, neuromotor stroke rehabilitation

I. INTRODUCTION

Stroke is one of the leading causes of permanent disability in the USA [1] and worldwide [2]. The major upper extremity (UE) motor impairments after stroke involve muscle weakness, spasticity, and abnormal intermuscular coordination. Even after successful treatment or spontaneous recovery of weakness and spasticity after stroke, motor impairment often persists [3]. For this reason, developing a rehabilitation strategy that helps improve intermuscular coordination holds promise for innovating clinical solutions to enhance motor function.

The deficit in individualizing multi-joint movements in the arm post-stroke, a notable factor contributing to post-stroke abnormal intermuscular coordination, is associated with a reduction in motor function [4]. Previous classic studies

consistently revealed that the absence of individual joint control is exhibited as fixed movement patterns, accompanied by difficulties in isolating movements to perform voluntary motor tasks [5,6]. Later experiments have shown an unusually strong coupling between the shoulder and elbow after stroke [7,8]. For instance, a previous study on isometric torque production in the arm found that abnormal elbow flexor torque occurs as a secondary effect during shoulder activity after stroke [8], which is associated with a lack of individualized activation of intermuscular coordination around the shoulder and elbow. This lack of individualization of multi-joint intermuscular control can be quantified by dimensional reduction methods, such as non-negative matrix factorization (NNMF). NNMF can identify a few intermuscular coordination patterns, also known as muscle synergies, recruited to perform a variety of movements.

To reduce abnormal intermuscular coordination, resistive training through human-machine interaction has emerged as a promising rehabilitation tool for stroke recovery. For example, our previous study showed that stroke survivors could improve motor control by developing new intermuscular coordination through EMG-guided isometric resistive human-machine interaction [9]. Furthermore, EMG-guided computer interfaces have been used to reduce abnormal co-activation of a pair of arm muscles after stroke [10,11]. However, it is still unknown 1) whether UE muscle synergies could be used as feedback for developing rehabilitation strategies to reduce atypical joint coupling and multi-muscle coordination abnormalities after stroke, and 2) whether decreasing these abnormalities would improve motor impairment in the UE. In this study, a muscle-synergy-guided exercise protocol was designed to improve the abnormal intermuscular coordination to reduce motor impairment after stroke by training the individual control of each muscle synergy in the UE.

II. METHODS

A. Participants

Three chronic stroke volunteers (one female (P1), and two males (P2 & P3); 68, 47, and 69 years old, respectively) with a single unilateral event participated in this study with mild to severe UE impairment (UE Fugl-Meyer assessment (FMA) scores before training: 49, 33, and 6). Exclusion criteria included the presence of any other neurological pathology or orthopedic disorders in UE. The study was conducted in accordance with the Declaration of Helsinki, with the approval of the University of Houston Institutional Review Board. Before each session, participants provided informed consent.

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B. Equipment

KAIST Upper Limb Synergy Investigation System (KULSIS) was used for isometric training and assessment sessions [12]. The three-dimensional forces measured at the hand and the surface electromyographic (EMG) (Trigno Wireless Biofeedback System; Delsys Inc., Boston, MA) signals were collected at a sampling rate of 1kHz. The eight UE muscles recorded included brachioradialis (BRD), biceps brachii (BB), triceps brachii (long (TrLo) and lateral (TrLa) heads), deltoids (anterior (AD), middle (MD), and posterior (PD)) fibers, and pectoralis major clavicular head (Pect).

C. Muscle Synergy Analysis

Surface EMG data were collected while participants performed isometric force target matches in 54 directions, equally spaced in 3D, from both arms in separate assessment sessions. The 54 target matching task was scaled to be matched when participants applied 40% of their maximum lateral force and maintained the force for one second. During the assessment, participants grasped the handle, which was positioned so that the participants' hand was aligned with their ipsilateral shoulder. The distance between the handle and the participant's acromion was 60% of the full arm length. The raw EMG signals were wavelet filtered to remove any electrocardiogram artifact visually inspected, demeaned, and full-wave rectified. The mean baseline was subtracted from the processed signal and then low pass filtered (Butterworth, 4th order, 10Hz as cutoff frequency) to obtain a signal envelope.

The preprocessed one-second holding period EMG signals were concatenated across trials. All negative values were set as zero to meet the non-negative constraint of NNMF, and each muscle's EMG data were normalized to the unit variance to avoid bias towards muscles with high variance. NNMF was applied to identify muscle synergies from both arms separately. By applying NNMF, the EMG matrix from each arm was modeled as $EMG_{isometric} = W \cdot C$, where W was an eight (number of muscles) by N (number of muscle synergies) matrix, and C was an N by D (number of data points) matrix. The W matrix is the time-invariant muscle weights or muscle synergies, while the C matrix is their corresponding time-varying activation profile. The predefined criteria to set the optimal number of synergies included the global variance accounted for (gVAF) greater than 90%; an additional synergy would increase the gVAF by at least 5%, and each muscle $VAF > 60\%$ [13,14]. The similarity index (SI) between a pair of muscle synergy vectors was calculated by obtaining their scalar product. For each of the four synergies, the linear summation of the synergy activation profile as a function of the target force vector, acquired from the EMG data of the more affected arm, defined the preferred direction of the synergy activation (PDS) in the 3D force space.

D. Design of Synergy-Guided Feedback

To design customized target (or model) synergies that would guide the training, healthy, age-matched participants' muscle synergies [15,16] were referred. These four synergies and their major muscle excitation were: Elbow Flexor (EF) (BRD & BB), Elbow Extensor (EE) (TrLo & TrLa), Shoulder

Flexor/Adductor (SF/Ad) (AD, MD, & Pect), and Shoulder Extensor/Abductor (SE/Ab) (MD & PD, or PD only). The muscle synergies in previous studies underlay the 3D unbiased isometric force generation, the same as the one adopted in the current assessment sessions.

Muscle synergies were identified from the EMG data of each stroke participant's less- and more-affected arms, respectively, during the isometric force generation task, described in Section II-C. More-affected arm synergies were assessed before and after the training as one of the outcome measures to identify potential changes in intermuscular coordination. Also, the muscle weights of the synergies identified from the less-affected arm were used to define the muscle weights of the target muscle synergy matrix for customized training (e.g., Fig. 3B).

Each participant had his or her own target muscle synergy vectors. The muscle weights of the four synergies (EF, EE, SF/Ad, and SE/Ab) vectors were obtained to guide each participant's training. For example, the two antagonistic elbow synergy targets (EF and EE) were deemed to be matched if the participant generated the following EMG signals, respectively.

$$EF = \left(\alpha_{LA} * \frac{BRD_{MA}}{MVC_{MA BRD}} + \beta_{LA} * \frac{BB_{MA}}{MVC_{MA BB}} \right) * \min \left(\alpha_{LA} * \frac{BRD_{MA}}{MVC_{MA BRD}}, \beta_{LA} * \frac{BB_{MA}}{MVC_{MA BB}} \right)$$

$$EE = \left(\gamma_{LA} * \frac{TrLo_{MA}}{MVC_{MA TrLo}} + \delta_{LA} * \frac{TrLa_{MA}}{MVC_{MA TrLa}} \right) * \min \left(\gamma_{LA} * \frac{TrLo_{MA}}{MVC_{MA TrLo}}, \delta_{LA} * \frac{TrLa_{MA}}{MVC_{MA TrLa}} \right)$$

, where α_{LA} , β_{LA} , γ_{LA} , and δ_{LA} are the muscle weights for BRD, BB, TrLo, and TrLa, respectively, identified from the less-affected arm. Also, BRD_{MA} , BB_{MA} , $TrLo_{MA}$, and $TrLa_{MA}$ are the real-time low-pass filtered (Butterworth, 3rd order, 5Hz as cutoff frequency) EMG data of each UE muscle from the more-affected arm. The same idea was applied to define the SF/Ad and SE/Ab targets. The maximum voluntary contraction of the more-affected arm (MVC_{MA}) per muscle and the maximum voluntary synergies (MVS) were determined at the beginning of each training session by assessing the maximal synergy activation three times in each of the 4 PDSs (12 trials, in total). Finally, to visually guide the training, these four synergy-driven signals were mapped to the direction and magnitude of the cursor movement in the 2D display. The EF and EE activation was mapped to upward and downward cursor movement along the positive and negative y-axis, respectively. Similarly, the SF/Ad and SE/Ab activation resulted in medial and lateral movement of the cursor along the negative and positive x-axis, respectively (Fig. 1).

E. Training Protocol

Each participant performed one hour of synergy-guided isometric training per session, three times per week for six weeks (18 training sessions in total). To adjust the task difficulty, the visual feedback screen was adjusted to 70% of each premeasured MVS. The objective of the training task was to match one of the four individual synergy targets on a two-dimensional visual display. For instance, to match Target 1 or Target 2 (Fig. 1), a participant should activate EF or SE/Ab synergistic muscle group, while suppressing the activation of the other three synergies.

The four targets were provided in a pseudorandomized order. The horizontal and vertical target margins were set as

15% of the MVS to create a target match zone where the cursor needed to stay for one second to make a successful trial. The severely impaired participant (UE FMA=6) could not apply 70% of MVS and hold it for the one-second holding period; therefore, the training protocol was customized in the way that a successful trial requires the target force magnitude as 40% of MVS, maintained for a 0.5s holding period. During training, participants explored and developed their own strategies to match the targets. However, general verbal instruction was provided as needed to facilitate the participant to activate a single muscle synergy at a time. For instance, to match the EF target, the participants were suggested to generate an isometric force in the upward and backward direction, activating their elbow muscles while trying to keep their shoulder relaxed. If multiple muscles underlying more than a single synergy were activated simultaneously, the cursor moved in a diagonal direction on display. For example, if EF and SF/Ad were co-activated, the cursor would move to the top-left corner. Also, if EF and EE were co-activated, the cursor location would be close to the center. These resulted in missed trials. Finally, to assess clinical motor impairment and motor function in the arm, the UE FMA and the Action Research Arm Test (ARAT) were performed before and after training, respectively.

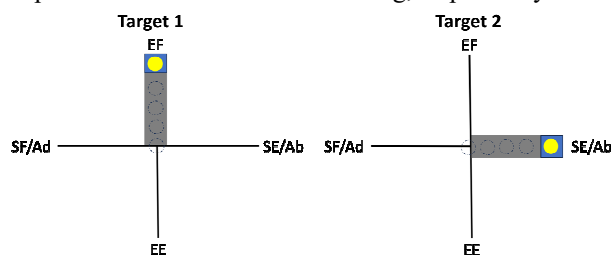


Figure 1. The mapping of four muscle synergies to 2D display and the schematic of the match of two sample targets (EF & SE/Ab). Participants controlled the cursor location to one of the four targets (up-EF, right-SE/Ab, down-EE, left-SF/Ad) by selectively activating each muscle synergy.

III. RESULTS

A. Task Performance

As opposed to the first week of the training, post-stroke participants were able to match the targets in all four directions during the last week, with the exception of P3, who never matched the SF/Ad target. Moreover, the number of target matches statistically increased ($p < 0.05$) across all three participants (Fig. 2).

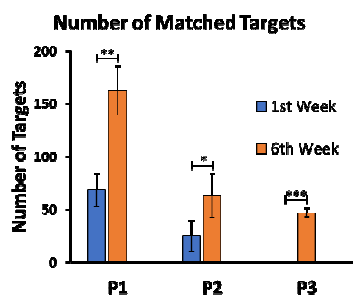


Figure 2. The mean and standard deviation ($n=3$, P1-P3 participants) of the number of matched targets per session calculated from the three sessions of the first (blue) training week and the three sessions of the last (orange) training week (t -test; *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.0001$).

B. Intermuscular Coordination

The number of muscle synergies for the more-affected arm did not change after training across the participants (typically four). The results of the pre-training assessment showed that the altered synergy vectors, reflected as low SI values between their target and more-affected arm synergies, were different across participants. The result indicates that the characteristics of impaired intermuscular coordination were individualized within the stroke group. The two lowest SI values and their associated synergy vectors per participant were: P1 (0.77 for EF and 0.82 for SF/Ad), P2 (0.68 for EE and 0.78 for SE/Ab), and P3 (0.84 for EE and 0.89 for SE/Ab). In contrast, the post-training assessment showed that changes in muscle synergy composition after the isometric exercise (e.g., Fig. 3A) increased SI values in multiple synergy vectors including the two ones with the lowest SI values. For example, P1's both EF and SF/Ad SI values increased up to 0.98 ($\max=1$). P2's EE and SE/Ab SI values increased up to 0.87 and 0.88, respectively. P3's EF and EE SI values increased up to 0.98 and 0.99, respectively (Fig. 3C).

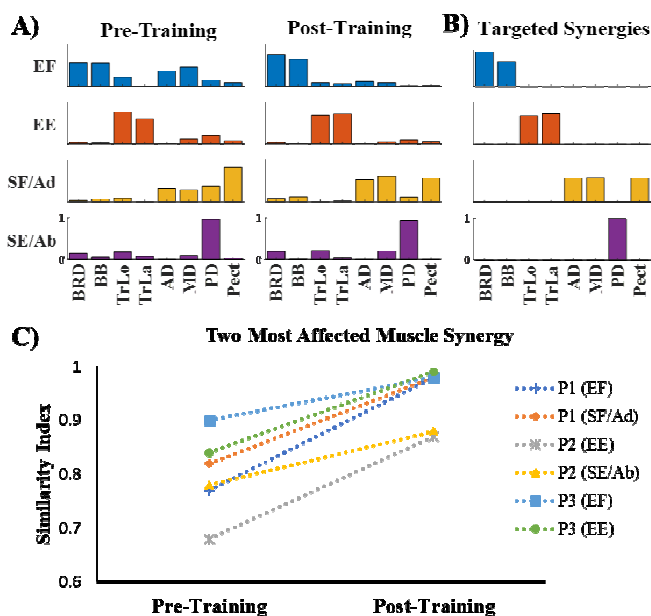


Figure 3. A) The composition of pre- and post-training muscle synergy patterns from the stroke-affected (or more-affected) arm and B) the associated target synergies underlying the less-affected arm of the same representative participant, P1. C) Similarity indices (SI) of the two most stroke-affected muscle synergies per participant (P1, P2, or P3). The increase in the similarity between the participant-specific target synergy and its corresponding pre- or post-training synergy indicates the improvement of intermuscular coordination after the training. EF, Elbow Flexor; SF/Ad, Shoulder Flexor/Adductor; EE, Elbow Extensor; SE/Ab, Shoulder Extensor/Abductor.

C. Clinical Assessments

After the treatment, UE FMA scores of the more-affected arm improved in all participants (P1 from 49 to 61, P2 from 33 to 43, and P3 from 6 to 14, respectively) (Fig. 4). Moreover, only P2 with moderate impairment improved the ARAT score of the stroke-affected arm from 23 to 33, while in both pre- and post-training sessions, P1 scored 54 and P3 scored 0.

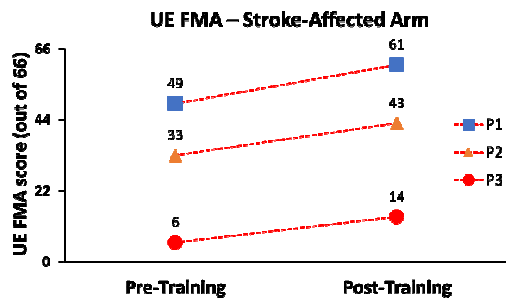


Figure 4. UE FMA scores of the stroke-affected arm of three participants (P1-P3) before and after the six-week synergy-guided exercise.

IV. DISCUSSION

This pilot study showed that UE intermuscular coordination after stroke could improve through an innovative muscle synergy-guided exercise performed through human-machine interaction. Traditionally, muscle synergies have been mainly used as an assessment tool to characterize stroke-altered neuromuscular coordination; nevertheless, this concept has been rarely applied as a tool that guides neuromotor rehabilitation therapies [17]. Recently, a small number of studies have focused on developing new rehabilitation strategies by targeting atypical co-activation of specific muscle pairs [10,18] without training all UE major synergistic muscles comprehensively. The current study provides three noteworthy observations. First, an increase in the number of targets matched after six weeks of training indicates stroke participants, through this exercise, could improve neuromuscular control of the more-affected arm by learning independent control of synergistic muscle groups. Second, similar to our previous study [9], participants demonstrated the capacity to modulate their muscle coordination patterns using a non-invasive exercise through human-machine interaction. In contrast, this study also revealed a notable change in stroke-induced muscle patterns, so that post-training intermuscular coordination became more similar to the targeted one. Third, this study showed that through holistic modulation of UE intermuscular coordination patterns to mimic those observed in healthy participants, stroke survivors, even those with severe motor impairment, could effectively decrease their UE motor impairment. These results suggest that muscle synergy principles might be useful as a rehabilitation strategy after stroke. Interestingly, the participant with a moderate level of motor impairment (P2) demonstrated an improvement in the upper limb motor function score (ARAT). In contrast, mildly and severely impaired volunteers (P1 and P3, respectively) maintained the same ARAT score after training, potentially as a ceiling or floor effect. P1's score was close to the maximum value (54 out of 57), indicating a high level of motor function even before training, while P3's score stated at the minimum score (0 out of 57), indicating severe limitation of the arm function.

In summary, this pilot study showed that through synergy-guided human-machine interaction, chronic stroke survivors could enhance their ability to individually activate synergistic muscle groups. Also, alternation in muscle synergy after stroke can be improved after the exercise; the stroke-affected

synergy composition became similar to that of the targeted intermuscular coordination patterns. These changes can lead to a decrease in motor impairment and an increase in motor function. Finally, we will perform a strengthening therapy without synergy-guided feedback in stroke as a control test to determine whether the observed improvements are specific to the synergy-guided training or are the general effects of a strengthening exercise.

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