

Temporal Features of Muscle Synergies in Sit-to-Stand Motion Reflect the Motor Impairment of Post-Stroke Patients

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Abstract—Sit-to-stand (STS) motion is an important daily activity, and many post-stroke patients have difficulty performing STS motion. Previous studies found that there are four muscle synergies (synchronized muscle activations) in the STS motion of healthy adults. However, for post-stroke patients, it is unclear whether muscle synergies change and which features primarily reflect motor impairment. Here, we use a machine learning method to demonstrate that temporal features in two muscle synergies that contribute to hip rising and balance maintenance motion reflect the motor impairment of post-stroke patients. Analyzing the muscle synergies of age-matched healthy elderly people ($n = 12$) and post-stroke patients ($n = 33$), we found that the same four muscle synergies could account for the muscle activity of post-stroke patients. Also, we were able to distinguish post-stroke patients from healthy people on the basis of the temporal features of these muscle synergies. Furthermore, these temporal features were found to correlate with motor impairment of post-stroke patients. We conclude that post-stroke patients can still utilize the same number of muscle synergies as healthy people, but the temporal structure of muscle synergies changes as a result of motor impairment. This could lead to a new rehabilitation strategy for post-stroke patients that focuses on activation timing of muscle synergies.

Index Terms—Muscle synergy, sit-to-stand, post-stroke, rehabilitation, random forest.

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I. INTRODUCTION

THIS study aimed to investigate the muscle synergy structure of post-stroke patients during sit-to-stand (STS) motion and to determine the primary features in muscle synergies that reflect the motor impairment of the patients. Stroke is the second leading cause of death and a major leading cause of disability [1]. The absolute number of post-stroke patients has increased because of the aging of the world's population [2]. As one of the most common causes of long-term disability, stroke causes an immense economic burden and places strain on caregivers [3]. Stroke survivors often present sensorimotor impairments that limit their motor ability to perform activities such as walking [4], standing [5], and STS motion [6]. To date, disability, impairment, handicap, and quality of life in post-stroke patients have been evaluated using clinical scales [7], of which one of the most adopted to evaluate impairment is the Fugl-Meyer Assessment (FMA). Some post-stroke patients may have the same evaluation score but different problems associated with their movement. Thus, it would be helpful to develop a quantitative evaluation method with respect to the specific motion of post-stroke patients that can reveal the patients' deficits and provide advice regarding their rehabilitation.

STS motion is a fundamental functional ability that is greatly affected in post-stroke patients. The important factors in STS motion, such as muscular activation, angular displacement, and center of mass (CoM) movement, have been investigated for healthy adults [8] and post-stroke patients [9]. These factors explain how STS transfer is accomplished and provide important information that may improve the STS performance of post-stroke patients. Some post-stroke patients employ a compensatory strategy, where they incline their CoM forward more before raising hips than healthy adults [6]. This study shows that post-stroke patients alter their STS movement by changing their movement strategy to avoid falling. Ada et al. reported that some post-stroke patients showed a lack of coordination between hip and knee joints; they completed knee

extension earlier while their hips were still extending [10]. Cheng *et al.* found that when post-stroke patients extend their body, the muscle soleus is activated earlier, almost simultaneously, with the quadriceps and hamstrings [11], [12]. It was found that post-stroke patients have delayed muscle activation compared with healthy adults [13]. These studies found that there is abnormal muscle co-activation during the STS motion of post-stroke patients. However, prior studies typically focused on the kinematic information or muscle activation characteristics in individual muscles. The neural aspects involved in the STS control of post-stroke patients remain elusive. Stroke causes lesions in the central nervous system that may essentially affect the central controllers, leading to abnormal coordination of muscles. Abnormal muscle coordination would directly result in impaired biomechanical output. Therefore, investigating the muscle coordination in the STS transfer of post-stroke patients is fundamental to developing improved rehabilitation strategies and may provide greater insight into the mechanisms that can improve STS performance.

To clarify how human movement is achieved by muscle coordination, muscle synergies were first proposed by Bernstein, who suggested that human movements could be generated from a limited number of modules (called muscle synergies) [14]. Bernstein decomposed the complex control of individual muscles into a modular organization. Previous studies have shown that the muscle activation of human motor behaviors, such as locomotion, postural control, and STS transfer, can be explained as the linear-summation of a small number of muscle synergies [15]–[18]. These studies have also suggested that these muscle synergies may exist in the spinal cord [19], [20]. Ivanenko *et al.* found that muscle synergy structures were similar in healthy humans walking with different speeds and gravitational loads; however, they adaptively changed the timing activation of muscle synergies to adapt to different conditions [15]. These findings suggest that humans may utilize different combinations or different ways to activate the limited number of muscle synergies to accomplish adaptive movements. In patients with motor impairment, the question is whether these muscle synergies are invariant. Previous studies analyzed the muscle synergy structure in motor-impaired patients, such as those with brain damage, spinal cord lesions, and other motor disturbances. Clark *et al.*, studying post-stroke patients, Rodriguez *et al.*, studying Parkinson’s disease, and Fox *et al.*, studying patients with spinal cord injuries, found that patients have fewer muscle synergies compared with healthy subjects, suggesting that the former have decreased neuromuscular complexity as a result of the dysfunction of parts of the central nervous system [16], [21], [22]. Clark *et al.* also suggested that the decreased muscle synergy numbers in human locomotion lead to the more compensatory walking strategies used by post-stroke patients. Previous studies have also emphasized plasticity and solutions geared at reorganizing muscle patterns in patients with motor impairment [23]. Therefore, clarification of the muscle synergy structure in post-stroke patients with motor impairment will provide useful information for research on rehabilitation.

Although there are many studies regarding muscle synergies in human locomotion in patients with motor impairment, it is

TABLE I
PARTICIPANT DEMOGRAPHICS

	Mean \pm SD (Range)
Post-stroke patients’ group (n=33)	
Age (yrs)	58.1 \pm 11.8 (34-79)
Lower extremity FMA score (out of 34)	23.8 \pm 6.9 (9-34)
Gender (male/female)	23/10
Side affected (left/right)	23/10
Control group (n=12)	
Age (yrs)	64.2 \pm 3.2 (60-68)
Gender (male/female)	10/2
Side measured (left/right)	5/7

not clear whether muscle synergies were altered in the STS movement of post-stroke patients. For human STS movements, our research group employed both forward dynamic simulation [18] and experimental measurement [24] to clarify the muscle synergy structure in healthy young adults. In the present study, we first recorded the muscle activation data from post-stroke patients with different severities and from a subgroup of age-matched healthy controls, and then extracted the muscle synergies. After this, we compared the features in muscle synergies that led to the differences between the STS movements of healthy controls and post-stroke patients. We investigated the features that caused different STS movements in post-stroke patients with different severities. The first aim of the present study was to determine if there were differences between the muscle synergies of post-stroke patients and healthy subjects. The second aim was to clarify the important features in muscle synergies that reflected motor impairment. This study hypothesized that muscle synergies might be abnormal in the STS motion of post-stroke patients. The results indicated some important features in muscle synergies that primarily reflect the motor impairment in the STS movement of post-stroke patients and could be used as physiological markers for evaluation.

II. METHOD

A. Subjects

Thirty-three post-stroke patients and twelve healthy age-matched controls participated in this study. Both the healthy elderly participants and post-stroke patients were asked to stand up from their own comfortable feet location. All of the subjects could stand up from a chair by themselves without any support. For post-stroke patients, the average value of the lower extremity FMA score was 23.8 ± 6.9 (see Table 1 for demographic information). In order to clarify muscle synergy feature that reflect motor impairment, patients were divided into two groups based on FMA scores (predefined FMA threshold is 20): the “mildly impaired” group ($n = 24$): FMA ≥ 20 , and the “severely impaired” group ($n = 9$): FMA < 20 . To avoid arbitrary determination of the FMA threshold and to investigate the effect of FMA threshold on results, we utilized different FMA thresholds to divide the post-stroke patients into two groups. If the prepared two groups were clearly divided by muscle synergy feature, it indicated that the muscle synergy features reflected motor impairment. Each participant of the control group and each post-stroke patient performed

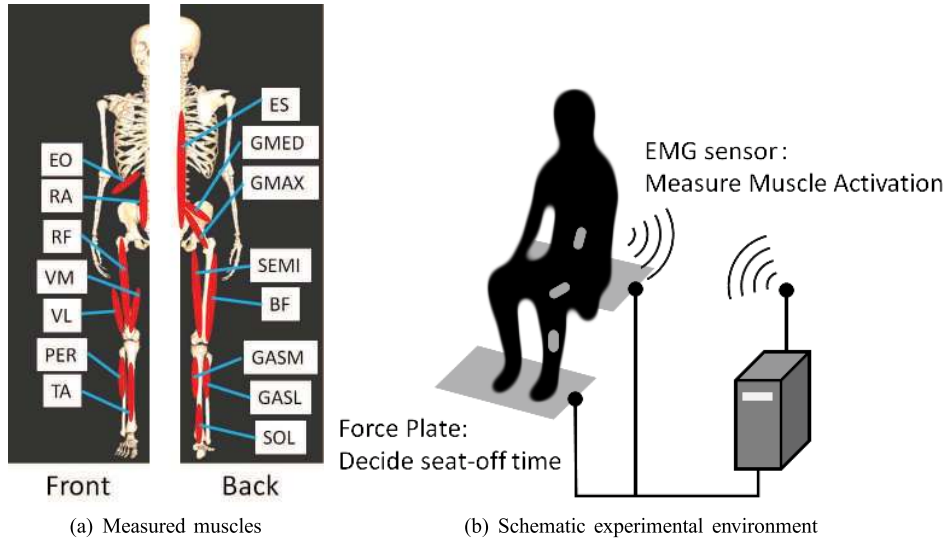


Fig. 1. Experimental setting.

10 trials. Some measured trials were deleted because of signal noise. The chair height was adjusted to the height of the lower leg. The participants finished the motion without moving their feet in all the trials. The informed consent of all participants was obtained, according to the protocol of the Institute Review Board of The University of Tokyo and Morinomiya Hospital, Japan.

B. Experimental Setting

Two force plates (TechGihan Corp.) were used to record the reaction force data at 2,000 Hz. The participant sat on one force plate and placed his or her feet on the other. The collected reaction force data was filtered with a low-pass filter at 20 Hz. The force data was used to define the seat-off time (when the vertical force on the chair became less than 10 N). A wireless surface EMG device (Cometa Corp.) was used in this experiment to obtain the muscle activities data at 2,000 Hz. Fifteen muscles related to STS motion were measured according to their contributions to the extension and flexion of the ankle, knee, hip, and lumbar: the tibialis anterior (TA), gastrocnemius lateralis (GASL), gastrocnemius medialis (GASM), peroneus longus (PER), soleus (SOL), rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), biceps femoris long head (BF), semimembranosus (SEMI), gluteus maximus (GMAX), gluteus medius (GMED), rectus abdominis (RA), abdominal external oblique muscle (EO), and erector spinae (ES), as shown in Fig. 1 (a). For post-stroke patients, the muscles on the affected side were measured. For healthy controls, five of them were measured on the left side and seven of them were measured on the right side to match the side of the corresponding post-stroke patients. All of the EMG signals were band-pass filtered (4th-order zero-lag Butterworth digital filter, passband 40–400 Hz) to attenuate DC offset and high-frequency noise [16], [25], [26]. Then, the filtered signals were rectified and low-pass filtered (4th order, cut-off frequency 4 Hz) [16]. Participants repeated sit-to-stand motion ten times, and there were at least 2 to 3 s intervals

between each trial. EMG data was measured throughout the experiment. The information from the force plate was used to define each repetition. Each repetition was cut from the whole recorded EMG signals 1 s before and 2 s after the seat-off time. Afterward, the EMG signal from each muscle was normalized based on its peak value in each repetition of each participant [16]. The schematic experimental environment is shown in Fig. 1 (b).

C. Muscle Synergy Model

Human STS motion is a result of multi-joint movements achieved by muscle coordination. For the muscle synergy model, muscle activation can be expressed as the linear summation of spatiotemporal patterns in a mathematical expression, as in Eq. (1):

$$\mathbf{M} = \mathbf{W}\mathbf{C}, \quad (1)$$

where matrices \mathbf{M} , \mathbf{W} , and \mathbf{C} indicate muscle activation, spatial pattern, and temporal pattern matrices, respectively. Matrix \mathbf{M} consists of muscle activation vectors m_i ($i = 1, 2, \dots, n$) to represent the activation of n different muscles.

Figure 2 shows the schematic design of the muscle synergy model. Three muscle synergies are used to express n muscle activations. They are composed of spatial and temporal patterns. Spatial patterns $\mathbf{w}_{1,2,3}$ show the contribution of each muscle to the synergy. Temporal patterns $\mathbf{c}_{1,2,3}$ show the timing activation of the synergy. During motion, the spatial patterns are constant, but the temporal patterns change over time. Muscle activation is generated from the linear production of spatial and temporal patterns of muscle synergies. Muscle activation is shown in the gray areas; muscle synergies 1, 2, and 3 are described by solid, dashed, and circled green lines, respectively. To calculate the elements of the matrices \mathbf{W} and \mathbf{C} , the non-negative matrix factorization (NNMF) [27] was used. The muscle synergies were extracted from each repetition of each subject. The order of muscle synergies extracted by the NNMF algorithm can differ among subjects

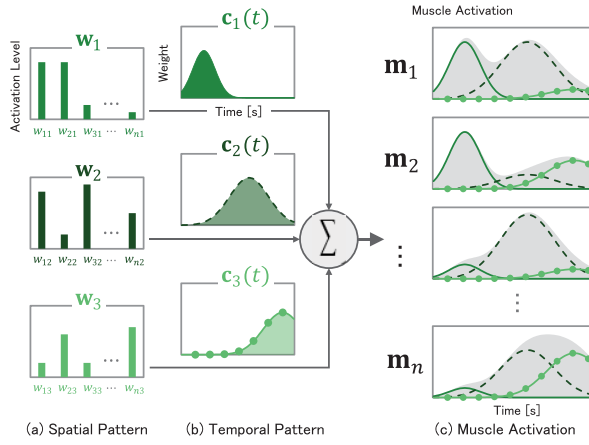


Fig. 2. Muscle synergies model.

and repetitions. Therefore, in order to cluster the similar muscle synergies extracted by NNMF algorithm, it was necessary to analyze the spatial and temporal patterns of healthy controls and sorted the results based on the peak time of each temporal pattern. We also checked the sorted results manually. Then, we computed the Pearson correlation coefficient of spatial and temporal patterns in every two synergies between the averaged healthy controls and each repetition of each post-stroke patient. The results of Pearson correlation coefficient were ranked, and the muscle synergies of post-stroke patients were clustered with the highest related muscle synergies of healthy controls. We also manually checked the sorted results of each repetition and post-stroke patient.

To investigate the change in muscle synergy after stroke onset, this study first examined the number of muscle synergies that could represent the muscle activation of the measured muscles. Therefore, the coefficient of determination R^2 was calculated for different numbers of muscle synergies [28], as in Eq. (2).

$$R^2 = 1 - \sum_{i=1}^n \frac{\sum_{t=1}^{t_{\max}} (m_0^i(t) - m^i(t))^2}{\sum_{t=1}^{t_{\max}} (m_0^i(t) - \bar{m}_0^i)^2}, \quad (2)$$

where $m_0^i(t)$ is the measured EMG of muscle i at time t after pre-processing, \bar{m}_0^i is the mean EMG value in muscle i . $m^i(t)$ is the EMG of muscle i at time t regenerated from the muscle synergy structure obtained by the NNMF algorithm. Afterward, the number of muscle synergies that could well represent muscle activation was determined [29]. The muscle synergy analysis was performed in MATLAB (Matlab R2017a). The algorithm used was ALS, and the number of iterations of the NNMF function was set to a default number of 100. The number of repetitions was 50. After analysis, the solution with the highest R^2 value was selected.

D. Similarity of Muscle Synergies

To determine if spatial patterns changed after stroke onset, the similarity between the muscle synergies of each post-stroke patient and healthy controls was quantified using the cosine of principal angles. Similarity represents the dimensionality of the subspace shared between the spaces spanned by the muscle

synergy sets [29], [30]. Similarity between the i -th and j -th muscle synergies were calculated using Eq. (3).

$$s_{ij} = \frac{\mathbf{w}_i \cdot \mathbf{w}_j}{|\mathbf{w}_i| |\mathbf{w}_j|}. \quad (3)$$

In addition to assessing absolute value of cosine similarity between the healthy control and stroke patients, inter-subject and intra-subject similarities were used to evaluate the level of statistic significance. The same procedure was used as the previous study [31]. When the inter-subject similarity is significantly lower than the intra-subject similarity, it indicates that the post-stroke group may have larger variances than the healthy control group and utilize different spatial patterns of muscle synergies from control healthy group.

E. Muscle Synergy Features

After evaluating the similarities in the spatial patterns of the post-stroke patients and healthy controls, differences in the temporal patterns were also investigated. Previous studies found that temporal patterns were merged in the locomotion of some post-stroke patients [16]. It has been suggested that post-stroke patients change their temporal patterns to achieve motion [26]. From these previous studies, it was found that post-stroke patients prolonged and delayed the activation time of some muscle synergies. However, it has been unclear which temporal features primarily affect the movement performance of post-stroke patients. Thus, several features in the temporal patterns were selected to describe the temporal features of post-stroke patients. First, the start, end, and duration time of temporal patterns were chosen because previous studies found that some post-stroke patients delayed or extended synergy activation, compared with other patients [26]. In addition, our previous study revealed that humans change the duration of muscle synergy to realize adaptive STS motion when their sensory information is impaired [32]. Then, the peak time was selected because our previous study found that the peak time affects STS strategies in healthy adults [24]. Therefore, we suggested the peak time might also affect the STS of post-stroke patients. Finally, the overlap time between two synergies may affect the STS performance. Clark et al. found that temporal patterns were merged in the locomotion of post-stroke patients [16]. These features are more interpretable than spatiotemporal patterns in muscle synergies and may be physiological markers that can be used to evaluate STS performance. First, the k -th muscle synergy was determined to be activated at time t , when its timing activation $c_k(t)$ was above the mean activation \bar{c}_k . \bar{c}_k was obtained during each trial from the following equation:

$$\bar{c}_k = \frac{\sum_{t_0}^{t_{\max}} c_k(t)}{t_{\max} - t_0}. \quad (4)$$

After it was determined whether each synergy was activated, each of the selected temporal features was obtained as follows:

- 1) Start time t_k^{st} : the first activated time of the k -th muscle synergy.
- 2) End time t_k^{ed} : the last activated time of the k -th muscle synergy.

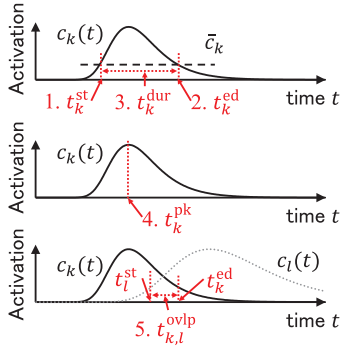


Fig. 3. Features selected in temporal patterns.

- 3) Duration time t_k^{dur} : the length between the start time t_k^{st} and end time t_k^{ed} . It is obtained as follows: $t_k^{\text{dur}} = t_k^{\text{ed}} - t_k^{\text{st}}$.
- 4) Peak time t_k^{pk} : the time when the maximum muscle activation is achieved. It is obtained as follows: $t_k^{\text{pk}} = \text{argmax } c_k(t)$.
- 5) Overlap time between every two muscle synergies k and l : $t_{k,l}^{\text{ovlp}} = t_k^{\text{ed}} - t_l^{\text{st}}$.

Figure 3 shows the features selected in the temporal patterns.

F. Classification

The selected temporal features had high variances in post-stroke patients because of different severity levels. For example, some patients mostly delayed the peak time of specific synergies, while others had longer overlap time. It was difficult to evaluate which features primarily affected motor improvement in STS. To clarify which temporal features were important, this study employed the random forest classifier. The random forest classifier can multi-compare the importance of all the features and sort the features based on their importance. Furthermore, the random forest classifier can also provide a result that is more robust to data size than other methods, such as K-means or hierarchical clustering. The random forest classifier is one of the most popular boosting methods and has performed well in many applications [33], [34]. It employs training set bagging and random subspaces based on decision trees [35]. A single decision tree computes the Gini impurity to find the best features in muscle synergies and split the data. This algorithm takes a top-down, greedy approach that is known as recursive binary splitting. It may produce good predictions on the training set, but also overfit the data and have low performance on the testing set. However, the random forest builds a large number of decision trees and randomly uses a subset of the features p of m input features ($p < m$). The randomness allows the training to avoid getting stuck at a local minimum, improves accuracy, and controls overfitting. Random Forest in Rstudio was used to train 500 trees [36].

The feature importance was computed using the mean decrease in both the Gini index and accuracy. In decision trees, every node is a condition regarding how to split values in a single feature, so that similar values of dependent features end up in the same class-set after splitting. This condition is based on the Gini impurity [35]. The Gini impurity is computed as

follows:

$$I_G(p) = \sum_{i=1}^J p_i \sum_{k \neq i} p_k = 1 - \sum_{i=1}^J (p_i)^2 \quad (5)$$

where p_i is the probability of an item with label i ($i \in \{1, 2, \dots, J\}$) being chosen and p_k ($k \neq i$) is the probability of an item being wrongly categorized. Therefore, when training a tree, it computes how much each feature contributes to decrease the weighted impurity or prediction accuracy. In the Random Forest method, feature importance is computed by averaging the condition over trees.

The input datasets were designed as follows. The first aim of this study was to find the main effective features that might cause differences in the STS movements of healthy controls and post-stroke patients; the data from both the control group and post-stroke patients' group were merged to build the first dataset. The labels of this dataset were "healthy" and "stroke". The second aim was to find important features that led to mildly or severely impaired motor performance in post-stroke patients. The second dataset contained data from the post-stroke patients' group. These patients were divided into two groups based on their lower extremity FMA threshold. Furthermore, "mildly impaired" and "severely impaired" were used as two labels in this dataset. "Downsampling" was used in random forest to solve problems that might be caused by an imbalanced number of samples. The input features were the selected timing features in the temporal patterns. The random forest classifier was trained based on these features and two labels in each dataset. After training, the random forest classifier outputted the importance of each feature in each dataset. For one dataset, the data were split into two parts. One part, 70%, was designated as the training data, which was used to build the random forest classifier. The other part, 30% of the data, was used to test the performance of the random forest classifier.

III. RESULTS

A. Muscle Synergy Number

Figure 4 shows the coefficient of determination R^2 of different muscle synergies numbers. The black dashed line (Fig. 4 (a)) and the black solid line (Fig. 4 (b)) respectively represent healthy participants and the affected side of the post-stroke patients. When the number of muscle synergies was four, the coefficient of determination R^2 was 88.7% and 88.4% for the control group and post-stroke patients, respectively. This indicated that four muscle synergies could represent most of the muscle activation during the STS motion of the control group. In addition, there were significant differences in R^2 between one and two, two and three, and three and four muscle synergies in both post-stroke and control groups (one-factor repeated measure ANOVA and post-hoc test, $p < 0.05$). There was no significant difference in R^2 between four and five muscle synergies in the two groups. To compare the characteristics of muscle synergy structure between the control group and the post-stroke patients, four muscle synergies were used to represent the muscle activation in STS motion.

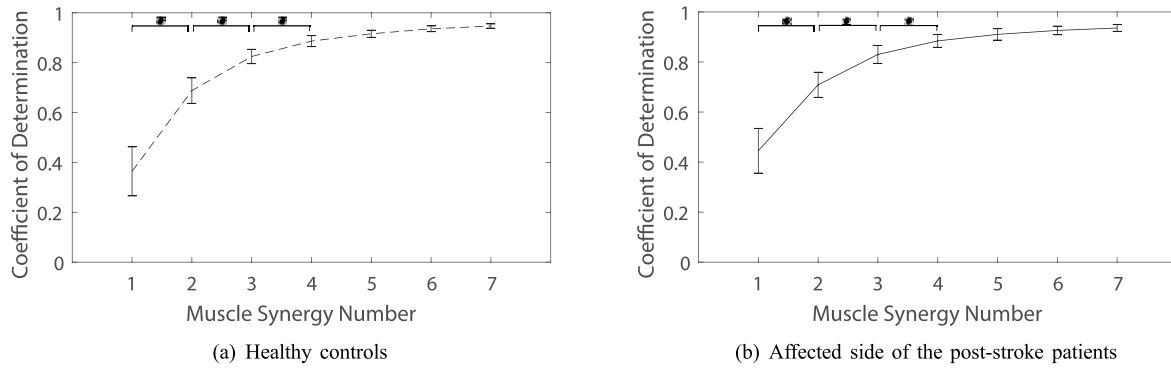


Fig. 4. Muscle synergy number. (a) Averaged R^2 value of muscle synergies in different numbers obtained from 120 trials of 12 healthy controls. (b) Averaged R^2 value of muscle synergies in different numbers obtained from 320 trials of 33 post-stroke patients.

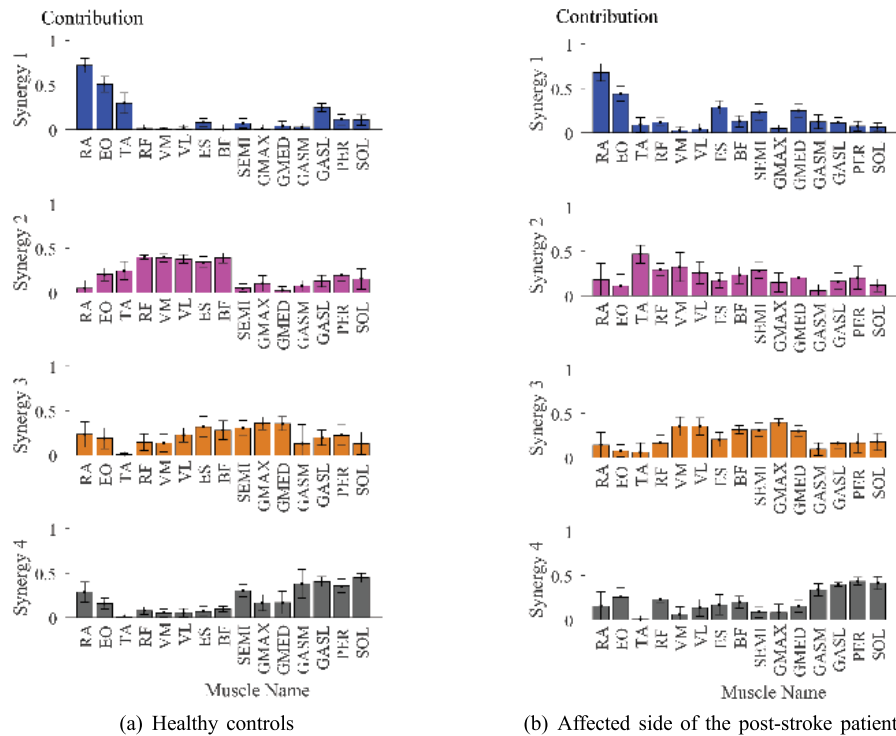


Fig. 5. Example of spatial patterns of muscle synergies during STS in one healthy subject and one post-stroke patient for 15 muscles. (a) Averaged spatial patterns of muscle synergies 1-4 (top to bottom) obtained from 10 trials of one healthy control. (b) Averaged spatial patterns of muscle synergies 1-4 (top to bottom) obtained from 10 trials on the affected side of one post-stroke patient.

B. Muscle Synergy

1) *Spatial Patterns:* Figure 5 shows spatial patterns with two individuals respectively selected from healthy controls and post-stroke patients as examples. The bars show the contributions of muscles in the control group and affected side of post-stroke patients, respectively. The horizontal axis in the graphs shows the name of the fifteen selected muscles, and the vertical axis shows the relative activation level of each muscle in the muscle synergy. Each muscle synergy has a particular contribution to human movement, according to the anatomical characteristics of muscles. The average spatial patterns of muscle synergies 1-4 (top to bottom) are represented by blue, pink, brown, and gray bars. Muscle synergy 1 was demonstrated to primarily activate the muscles RA and EO,

which flex the lumbar to bend the body forward and produce the necessary momentum for STS motion. Muscle synergy 2 mostly activated the muscle TA to dorsiflex the ankle joint to move the body forward, and the muscles RF, VM, and VL activated to extend the knee joint and raise the hip. Muscle synergy 3 primarily activated the ES, BF, SEMI, GMAX and GMED to move the whole body upward. Muscle synergy 4 mostly activated the muscles GASL, GASM, PER, and SOL to flex the knee and to plantarflex the ankle to decelerate the movement of the CoM and control the body posture.

The similarity between each muscle synergy, compared between the control group and every post-stroke patient, was computed using cosine similarity, as shown in Table 2. The results showed that the similarity between the spatial patterns of healthy controls and the affected side of post-stroke patients

TABLE II
SIMILARITY OF SPATIAL PATTERNS

	Healthy	Stroke	P-value
Synergy 1	0.88 ± 0.05	0.84 ± 0.07	0.094
Synergy 2	0.90 ± 0.03	0.83 ± 0.06	1.18E-05
Synergy 3	0.88 ± 0.03	0.85 ± 0.04	0.0027
Synergy 4	0.88 ± 0.03	0.84 ± 0.04	0.0022

was greater than 0.83. However, the comparison between inter-subjects and intra-subjects groups showed that there are significant difference in the similarity of muscle synergies 2, 3, and 4. This shows that stroke patients had larger variability in spatial pattern than did healthy subjects.

2) *Temporal Patterns*: For the temporal patterns of muscle synergies, Fig 6 shows the results obtained from the same subjects that were used to depict the results of Fig. 5. The horizontal axis in the graphs shows the duration time of the STS motion, normalized to 100%, and the vertical axis shows the timing activation of the muscle synergy. The blue, pink, brown, and gray solid lines represent the mean of the temporal patterns in muscle synergies 1, 2, 3, and 4, respectively. The dashed lines show the variance in the temporal patterns. For all the participants, muscle synergy 1 was first activated to bend the upper trunk; muscle synergy 2 was activated next, to flex the knee and raise the hip. The two muscle synergies contribute to move the body forward. After the hip was raised, muscle synergy 3 started to become activated to extend the knee and trunk to move the whole body upward. Finally, muscle synergy 4 was activated to plantarflex the ankle and knee and decelerate the horizontal movement of the CoM to maintain balance. Compared with the healthy control group, the start and peak times of the temporal patterns in post-stroke patients were delayed. Duration was also longer in the post-stroke patients group. The features that primarily affected the STS performance were classified using the random forest classifier, as described in the next subsection.

C. Performance of Random Forest and Feature Importance

1) *Accuracy of Random Forest Classifier*: In this study, the random forest classifiers were trained and tested with two datasets. In total, 22 features were selected as the input features for the classifier, including the start, end, duration, and peak time of four muscle synergies and six overlap times between every two synergies. The results obtained from the two classifiers are listed in Table 3. One dataset consisted of the data from both the control and post-stroke patients' groups. This dataset was used to find the main effective features that may cause the difference in the STS movements between healthy people and post-stroke patients. Both the training and testing accuracies were 84.5%. Another dataset only contained the data from the post-stroke patients' group. These patients were divided into two groups based on their FMA scores ("mildly impaired" group: FMA ≥ 20; "severely impaired" group: FMA < 20). The training and testing accuracy were 82.9% and 83.0%, respectively.

TABLE III
PERFORMANCE OF THE RANDOM FOREST CLASSIFIER

Dataset	Cases	Training accuracy	Testing accuracy
Post-stroke and control groups	440	84.5 ± 1.5%	84.5 ± 3.3%
Post-stroke group	320	82.9 ± 1.7%	83.0 ± 3.5%

TABLE IV
PERFORMANCE OF THE RANDOM FOREST CLASSIFIER

FMA threshold	Number of trials: mild/severe	Train accuracy	Test accuracy
19	256/64	83.2 ± 1.5%	82.9 ± 3.5%
20	237/83	82.9 ± 1.7%	83.0 ± 3.5%
21	237/83	82.9 ± 1.7%	83.0 ± 3.5%
22	217/103	78.0 ± 1.9%	78.5 ± 3.7%
23	207/113	76.1 ± 2.3%	76.3 ± 4.0%
24	187/133	73.0 ± 2.9%	73.3 ± 4.3%
25	157/163	70.0 ± 2.5%	69.6 ± 4.0%
26	147/173	70.9 ± 2.4%	70.8 ± 4.1%
27	128/192	69.6 ± 2.4%	69.7 ± 3.8%
28	98/222	72.9 ± 2.0%	73.4 ± 3.5%
29	98/222	72.9 ± 2.0%	73.4 ± 3.5%
30	89/231	72.6 ± 2.1%	72.7 ± 3.6%

To investigate the effect of FMA threshold on training and testing accuracies, we calculated the classification accuracy using different FMA thresholds (1st Qu.: 19, Median: 24, Mean: 23.9, 3rd Qu.: 30, Min: 9, Max: 34). Table 4 shows results of training and testing accuracies depending on different FMA thresholds. The test accuracy decreased when the FMA threshold increased from 20, and it particularly decreased when the FMA threshold was above 23. This might indicate that the temporal features of muscle synergies are useful for distinguishing patients when the FMA score is less than 23, but other important features might exist in the higher FMA score group. The above results were calculated using the whole dataset, but we also checked the result using "Downsampling" in random forest and found the imbalanced dataset did not affect the results. Therefore, we determined an FMA score of 20 as the threshold to divide the mild and severe groups.

2) *Important Features of Muscle Synergies*: The most important features were chosen based on the mean decrease in the Gini impurity in predictions. For the dataset that consisted of the control and post-stroke groups, the main features that affected the STS performance were the peak, duration, and start time of muscle synergy 2; peak, start, and end time of muscle synergy 3; and overlap between synergies 2 & 4, as shown in Table 5. Almost all of these features showed significant differences between the post-stroke and healthy control groups, except for the peak time of synergy 3.

For the post-stroke dataset, the main features that affected the STS performance were the peak and end time of muscle synergy 4; start, end, peak, and duration time of muscle synergy 2; and overlap time between muscle synergies 1 & 2, as shown in Table 6. All of these features showed significant differences between the mild and severe patients groups. These results also showed that the characteristics in the temporal pattern of muscle synergy 2 affected the STS performance. In addition, muscle synergy 4 played an important role in distinguishing the movements of the

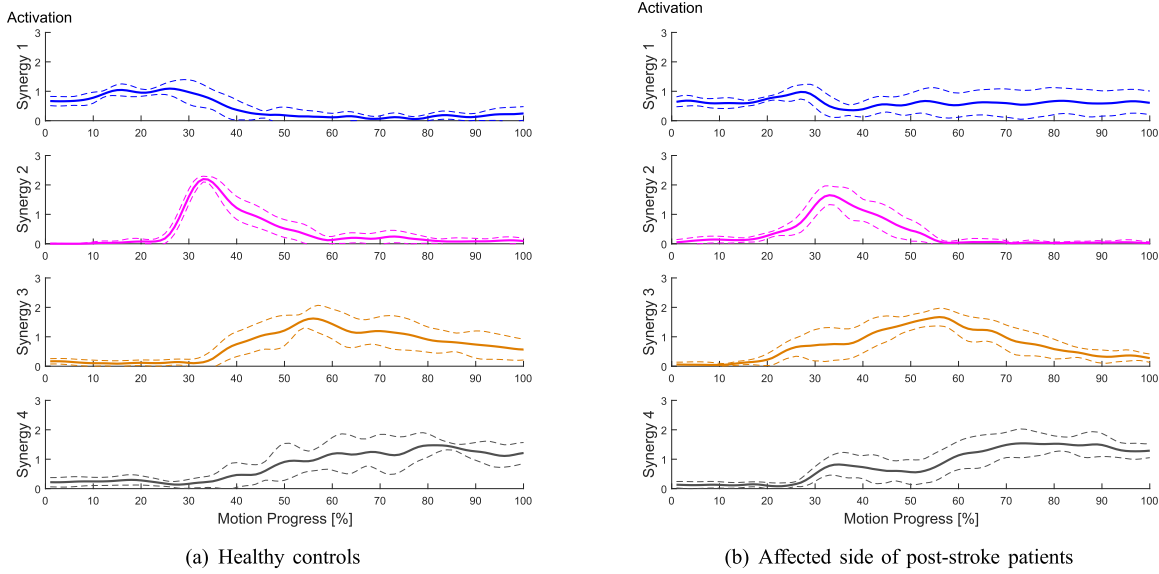


Fig. 6. Example of temporal patterns of muscle synergies during STS in one healthy subject and one post-stroke patient. Solid lines show the average timing activation, and dashed lines show the standard deviation. (a) Average temporal patterns of muscle synergies 1-4 (top to bottom) obtained from 10 trials of one healthy control. (b) Average temporal patterns of muscle synergies 1-4 (top to bottom) obtained from 10 trials on the affected side of one post-stroke patient.

TABLE V
IMPORTANCE OF FEATURES: POST-STROKE AND HEALTHY CONTROL GROUPS

Feature	Decrease Gini	Post-stroke (normalized motion progress [%])	Control (normalized motion progress [%])	Change from Control to Post-stroke	P-value
Peak of muscle synergy 2	25.3	36.3±11.1	32.9±3.3	Delay	9.45E-04
Duration of muscle synergy 2	17.1	36.2±10.6	28.6±6.2	Longer	5.21E-13
Peak of muscle synergy 3	14.3	56.9±20.4	55.7±9.4	Delay	5.33E-01
Start of muscle synergy 2	12.0	21.4±8.6	23.2±4.9	Earlier	3.11E-02
Start of muscle synergy 3	11.6	31.9±13.8	35.2±9.2	Earlier	1.26E-02
Overlap between muscle synergies 2 & 4	9.6	12.3±10.0	6.3±7.2	Longer	3.01E-09
End of muscle synergy 3	8.9	85.5±16.7	80.3±11.5	Delay	1.61E-03
⋮	⋮	⋮	⋮	⋮	⋮

TABLE VI
IMPORTANCE OF FEATURES: MILD AND SEVERE GROUPS

Feature	Decrease Gini	Mild (normalized motion progress [%])	Severe (normalized motion progress [%])	Change from Severe to Mild	P-value
Peak of muscle synergy 4	10.3	69.3±17.9	77.6±18.2	Earlier	1.81E-08
Start of muscle synergy 2	9.3	20.4±8.0	22.9±9.1	Earlier	3.15E-04
End of muscle synergy 2	8.7	62.3±16.7	73.4±18.9	Earlier	2.31E-11
Overlap between muscle synergies 1 & 2	7.9	15.6±7.2	12.4±8.3	Longer	2.88E-02
Peak of muscle synergy 2	7.9	34.9±10.2	38.1±12.1	Earlier	6.06E-05
End of muscle synergy 4	6.9	95.9±10.2	97.4±9.6	Earlier	2.17E-08
Duration of muscle synergy 2	6.2	34.6±8.9	38.1±11.0	Shorter	1.93E-06
⋮	⋮	⋮	⋮	⋮	⋮

post-stroke patients because of the postural control function. For the important feature, the results showed that the features of muscle synergy 2 always had higher ranking than other features when the FMA threshold to divide patients into two groups changed from 19 to 30.

IV. DISCUSSION

A. Spatial Features of Muscle Synergy

In this study, we found that post-stroke patients who can perform STS transfer independently can control the same

number of muscle synergies as the healthy controls. However, in other related work on muscle synergy, it was found that some post-stroke patients had decreased muscle synergies in locomotion [16]. This suggested that post-stroke patients who have less independently timed muscle synergies walked more slowly and had more asymmetrical step lengths. We suggest that this occurred because some post-stroke patients had asymmetric movements on different sides of their body when they walked. Although the number of muscle synergies decrease, and post-stroke patients cannot accomplish locomotion with

normal joint trajectories, they still can walk using compensatory strategies. For example, some post-stroke patients lift their hips and move the affected leg forward. However, the STS motion of post-stroke patients is relatively symmetric, where the non-affected side can lead the movement and the affected side follows. The joints on both sides have similar moving trajectories. These movements required similar control of muscle synergies on both the affected and non-affected sides; thus, the post-stroke patients retain the ability to control four muscle synergies. In addition, this study only measured patients who could at least stand up by themselves. Other patients with more severe symptoms, who have asymmetric movements in their STS motion, may have decreased muscle synergy number.

B. Temporal Features of Muscle Synergy

The results showed that the random forest classifier performed well at classifying the control and post-stroke groups, as well as the mild and severe groups. The two random forest classifiers investigated several main features that affect the motor improvement in STS. By comparing the post-stroke patients and healthy controls, it was found that the temporal features related to muscle synergies 2 and 3, such as the peak time, start time, and duration time, changed after post-stroke onset. The peak time is significantly delayed in muscle synergy 2, and the activation time is significantly longer in both muscle synergies 2 and 3 in the stroke group. The two muscle synergies contribute to raising the hip and extending the body during STS transfer. Eriksrud et al. found that knee extension is a strong predictor of independence during STS transfer [37], and Lomaglio et al. also showed that both knee extension and ankle dorsiflexion are related to STS performance [38]. In our previous study, it was found that post-stroke patients delayed the peak time of synergy 2 and required larger hip and lumbar flexion to move forward compared with healthy controls [39].

The results of the random forest classifier trained only based on post-stroke patients' data also showed that the temporal features related to muscle synergy 4 play important roles in STS transfer. The important features, the peak and end time of muscle synergy 4, were significantly delayed in the "severely impaired" group. Muscle synergy 4 primarily activated GASL, GASL, PER, and SOL and contributed to controlling the posture and maintaining stability. The abnormal activation of muscle synergy 4 showed that post-stroke patients belonging to the "severely impaired" group had difficulty maintaining balance during STS transfer. These post-stroke patients may not know the right time to control their posture. This result suggests that training the activation of synergy 4 may improve the STS performance of patients with severe post-stroke impairments. Previous studies also found similar results. Cheng et al. showed that post-stroke patients activated muscle SOL earlier, at almost the same time as they activated the hamstrings [11].

In addition to synergy 4, muscle synergy 2 is a key factor to distinguish motor severity. Delayed activation of muscle synergy 2 leads to delayed acceleration for moving

forward. Similar results were also found in our previous study [40]. Kogami et al. showed that the peak time of synergy 2 was earlier after a period of rehabilitation, and post-stroke patients who underwent rehabilitation also had better STS performance. Our study found that rehabilitation changed the peak of synergy 2, causing it to occur earlier during STS transfer. These phenomena can be explained by our analysis. The delay in peak time of muscle synergy 2 would delay the time when the individual raised his or her hip, resulting in movement whereby humans move forward before raising their hips. In other words, post-stroke patients tend to move their body closer to the base of support by delaying muscle synergy 2 and tend to choose a stabilized strategy post-stroke. Muscle synergies 2 and 4 play an important part in leading the transitions of patients from the severely impaired group to the mildly impaired group. The patients need to forward shift the activation time of muscle synergies 2 and 4 to raise the hip from chair and to decelerate movement and retain balance.

V. CONCLUSION

In this study, the muscle synergy model was employed to investigate the synergy features that primarily affect motor improvement during STS transfer. The muscle activation in post-stroke patients ($n = 33$) and age-matched healthy elderly subjects ($n = 12$) was measured. First, this study verified that post-stroke patients still utilized four muscle synergies, similar to the healthy controls. Muscle synergies 1 to 4 contribute to bending the upper body, raising the hips, extending the body, and controlling posture, respectively. Post-stroke patients had larger variability in the spatial patterns of muscle synergies compared with those extracted from healthy controls. In other words, different combinations of muscle activations might be utilized in post-stroke patients, whereas healthy people have more consistent coordinated muscle activation. The important temporal features that reflect the motor impairment of the STS motion were clarified by the random forest classifier. The random forest classifier showed that the temporal features of muscle synergies 2 and 4 primarily affect the STS performance improvement in post-stroke patients. The result suggests that it is necessary to teach post-stroke patients the right time to lift their hip during STS transfer. The results also show that the temporal features of muscle synergy 4 reflect the recovery of STS performance in severe post-stroke patients. This result shows that in the rehabilitation of severe post-stroke patients, it may be important to teach them the appropriate time to activate muscle synergy. For future work, the muscle synergy structure in post-stroke patients performing STS motion with the intervention of physical therapists will be clarified. The temporal features clarified by the random forest classifier will also be used to evaluate these post-stroke patients.

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