Quantifying the Peak Amplitude Distributions of Electromyogram in Bicep Brachii muscle after Stroke

Taimoor Afzal, Andrew Lai, Xiaogang Hu, William Z. Rymer, and Nina L. Suresh

Abstract—The objective of this study was to quantify the differences in surface electromyogram (EMG) signal characteristics between affected and contralateral arm muscles of hemispheric stroke survivors. EMG signals were recorded from the biceps brachii muscles using single differential electrodes. Four chronic stroke subjects performed isometric elbow flexions at sub-maximal voluntary contraction levels on both the affected and contralateral limbs. The force generated on the contralateral side was matched to the force generated on the affected side. We observed different types of EMG activation on the affected side compared to the contralateral side.

Specifically, two subjects showed lower RMS EMG activity on the affected side whereas two subjects showed greater EMG activity on the affected side compared to the contralateral side. Analysis of the peak amplitudes of the EMG activity showed greater number of peaks in the EMG on affected side compared to the contralateral side in all subjects. The histogram of the peak amplitudes showed greater number of smaller peak amplitudes in subjects with lower EMG activity on the affected side suggesting a reliance on smaller motor units. Our combined EMG signal analysis techniques on one set of recorded signals provides insight regarding potential mechanisms of weakness.

Clinical Relevance—Decoding neural information from surface EMG signals without decomposition into individual motor units could provide clinicians with quick insight about disease progress and potential treatment.

I. INTRODUCTION

Stroke is the leading cause of disability in the United States with nearly 800,000 people suffering a stroke each year. Muscle weakness on the side contralateral to the damaged cerebral hemisphere is a significant post-stroke symptom. The level of weakness is dependent on the severity of the neural damage ranging from reduced muscle strength to complete paralysis. The muscle weakness is potentially due, in part, to the changes in the descending cortical commands or reduced neural drive. Other potential causes of muscle weakness could be due to the alterations at the motoneuron level, including motoneuron control. Some of these changes include potentially disordered motor unit firing patterns [1], increased motoneuron excitability on the affected side [2], and disordered motor unit recruitment order and compression of recruitment threshold [3], i.e. larger motor-units recruited earlier.

Assessing motor unit behavior requires decomposition of electromyography (EMG) signals. Typical methods of assessing the weakened muscle is by recording EMG signals through invasive or non-invasive procedures. Invasive methods rely on inserting needle electrodes into an affected muscle and these provide precise recording of single motor unit action potentials (MUAPs)[4]. A limitation of using the invasive method is that to access large pool of MUAPs, multiple needles have to be inserted, thereby making the procedure an unpleasant experience for the patient. Non-invasive methods allow the recording of muscle activity using surface electrodes. Although, surface EMG signals lack the resolution provided by the needle electrodes, decomposition of the surface EMG signal provides a global picture of the underlying muscle activity. Typically, a surface EMG signal is the summation of individual MUAPs from different muscle fibers innervated by their respective motoneurons. Thus higher amplitudes in the EMG signal could be a result of either superposition or the presence of action potential from muscle fibers in close proximity of the electrode. In addition, the subcutaneous tissue and fat layer act as a low pass filter thus smoothing the motor unit action potentials. Despite these limitations, frequency and/or time domain analysis of the EMG signal can be used to indirectly assess the behavior of the motor units [5-7].

Peak amplitude distributions of the EMG signal is a powerful and expedient method to assess motor unit action potential properties. It has been previously tested in the first dorsal interosseous muscle in stroke survivors using a sensor array (Delsys, Inc) [6]. The authors reported a narrower band of peak amplitude distribution in one-third of stroke sample and a wider band of peak amplitude distribution in the remaining two-third sample. They attributed the alterations in peak amplitude distributions to post-stroke motor unit pathological changes. The results from these studies indicate that the EMG signal has the potential to provide valuable information about the underlying activity without the need of decomposition into individual motor units.

The objective of this study was to examine the peak amplitude distribution of the surface EMG signal as well as to estimate the root-mean-square EMG (RMS-EMG) and characterize the differences in the stroke affected side compared to the contralateral side at matched forces in the biceps brachii muscle. We recorded EMG signals from the biceps brachii muscle using a single-differential electrode. We demonstrate...
that the EMG signal collected from this electrode provides meaningful information about the composition of MUAPs.

II. METHODS

A. Subjects

Four chronic stroke subjects (3 male, 1 female), right hand dominant, participated in the study. The study was approved by the Institutional Review Board at Northwestern University Chicago, IL. All subjects provided written consent to participate in the study. The subject demographics are presented in table 1. Modified Ashworth Scale (MAS) score for elbow flexion and Fugl-meyer (FM) assessment of the arm motor function were performed. The inclusion criteria was a single hemispheric stroke occurring at least 6 months before participation in the study.

B. Experimental Protocol

Subjects were seated on a Biodex chair and the trunk was secured by Velcro straps across their torso from shoulder to hip. To avoid unwanted movement, the forearm on the testing side was casted and fixated at the wrist to a plastic interface attached to a six-degree of freedom Delta load cell (ATI, Apex, NC). The wrist was pronated at an angle of 45°. The isometric flexion force from the load cell was calculated using the root-sum-square of the horizontal (Fx) and vertical (Fz) components of force. The forces and surface EMG signals were sampled at 2 kHz using EMGWorks (Delsys, Boston). To ensure consistency between the affected and the contralateral sides, EMG sensors were placed at similar location on the two sides. Furthermore, we adjusted the casted limb to achieve a shoulder flexion of 20°, elbow extension of 120° and lower arm abduction of 45°. EMG signals were recorded using single differential surface electrodes (Delsys, Boston, MA, USA) placed on the medial and lateral heads of the biceps brachii. The skin was cleaned with alcohol pads prior to electrode placement.

The subjects were asked to perform isometric elbow flexion to produce maximum voluntary contraction (MVC). The MVC trials were repeated twice and the maximum value of the two trials was chosen as the MVC of the affected limb. After determining the MVC, target forces were assigned as a percentage of the MVC i.e. from 30% to 60% with increments of 10%. The same contraction levels as performed by the paretic side were used for the contralateral side. For each subject, the target force was displayed on the screen as a trapezoidal trajectory. The subjects were instructed to match the target force trajectory for a maximum of 10 seconds. Each trial consisted of a 3 s rest period followed by a ramp period of increasing flexion force at 10% paretic MVC per second, a 10 s steady-state stable portion and a final ramp period of force decrease again at 10% MVC per second of the paretic side. Subjects 1 and 4 generated forces at all sub-maximal levels. Subject 2 and 3 were unable to generate a force of 30% MVC on the affected side and therefore were only tested at 40%, 50%, and 60% of MVC of the affected side.

The order of target forces was randomized. Each target force was executed three times. The subjects were allowed to practice to familiarize with the task and make appropriate adjustments of the force. Subjects were provided rest between trials to prevent fatigue. The experiment was conducted on two separate days for the affected and contralateral side.

TABLE I. SUBJECT DEMOGRAPHICS

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs.)</th>
<th>Years since Stroke</th>
<th>Affected side</th>
<th>MAS</th>
<th>FM</th>
<th>MVC Aff/Cov</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>63</td>
<td>2</td>
<td>L</td>
<td>0</td>
<td>55</td>
<td>173 N/277 N</td>
</tr>
<tr>
<td>2</td>
<td>71</td>
<td>21</td>
<td>R</td>
<td>1</td>
<td>-</td>
<td>62/257</td>
</tr>
<tr>
<td>3</td>
<td>66</td>
<td>3</td>
<td>R</td>
<td>0</td>
<td>59</td>
<td>50/69</td>
</tr>
<tr>
<td>4</td>
<td>72</td>
<td>14</td>
<td>L</td>
<td>2</td>
<td>17</td>
<td>69/53</td>
</tr>
</tbody>
</table>

C. Data Analysis

EMG and force signals were collected in Spike 2 and analyzed offline in MATLAB (MathWorks, Natick, MA). EMG signals were filtered (4th order Butterworth) with a bandpass filter of 10-450 Hz. The peak amplitudes from the EMG signals were calculated during a 5 s steady-state period of constant force generation for each trial. Only those peak amplitudes were detected that were above the mean + 3 standard deviations (SD) of the baseline noise level. We also selected the negative peaks with a threshold of mean – 3 SD of the baseline level. For analysis purposes, positive peaks and absolute values of the negative peaks were used.

D. Statistical Analysis

We used un-paired sample t-tests to compare the differences between the elbow flexion force between the affected and contralateral side, EMG-RMS between the affected and contralateral side, and Peak counts between the affected and contralateral side for each subject.

III. RESULTS

A. Comparison of sub-maximal forces between sides

We calculated the ratio of the recorded MVC forces on the affected as compared to the contralateral side. We found a considerable decrease in MVC force on the stroke-affected side compared to the contralateral side. The MVC ratio varied from 0.24 to 0.72 indicating different levels of weakness on the affected side. To ensure the subjects produced matched sub-maximal forces on the contralateral side, we compared the forces between the sides. We compared the isometric forces between the affected and contralateral side including all force levels, separately for each subject. For each subject, we found no significant difference between the average forces between the two sides. As subjects performed 3 trials at each force level, we performed a correlation among the trials performed on the affected side with those on the contralateral side. For subjects 1, 2, and 4 the correlation between the forces was > 0.97. For subject 3, the correlation was 0.61.

B. Comparison of EMG activity

Next, we compared the EMG signals between the two sides. We calculated the RMS value of the EMG signal during the 5 s steady-state period. The average RMS value from all trials of the EMG signals in two stroke subjects was lower on the affected side compared to the contralateral side. For the other two subjects the RMS value of the EMG signal was higher on the affected side compared to contralateral side. Based on the differences between RMS values, we divided the subjects into two groups, i.e., subjects with lower EMG on the affected side (fig. 1(a-b)) compared to the contralateral side and subjects with higher EMG values on the affected side (fig 1(c-d)).
1) **Group 1: Lower EMG RMS on the affected side**

After assigning the subjects to two groups, we compared the peak amplitudes between the spastic and contralateral sides. Both subjects in group 1 showed higher number of peaks in the EMG signal compared to the contralateral side. The unpaired t-test showed significantly higher number of peaks in the affected side compared to the contralateral side. The distribution of peaks at each force level was further analyzed. We found a greater number of peaks at smaller amplitudes on the affected side compared to the contralateral side (fig. 1 e-f).

2) **Group 2: Higher EMG RMS on the affected side**

Although both subjects in group 2 showed greater RMS value on the affected side compared to the contralateral side, subject 3 showed a significantly higher RMS value on the affected side. Both subjects in group 2 also showed significantly higher number of peaks in the EMG signal compared to the contralateral side (fig. 1 g-h). The distribution of peaks at each force level was further analyzed. We found a greater number of peaks on the affected side compared to the contralateral side. The EMG-RMS vs force plot of the subject 3 shows a decrease in the EMG amplitude with increasing force levels. This could be potentially due to the activation of other muscles acting at the joint.

C. **Amplitude histogram analysis**

Figure 2 shows the absolute value of the amplitude distribution of the positive and negative peaks recorded in the EMG signal. In case of subject 1 and subject 2, the median amplitude is smaller on the affected side. For subjects 3 and 4, the median amplitude in the affected side is higher or similar to the contralateral side.

IV. **DISCUSSION**

In this study, we determined the peak amplitude distribution of the EMG signal in biceps brachii muscle to quantify post stroke changes in motoneuron and motor unit action potential behavior. In the relatively small sample, we found two contrasting behaviors in the EMG analysis. Specifically two subjects demonstrated lower EMG RMS on the affected side compared to the contralateral side at matched force levels. While two subjects showed a greater EMG RMS on the affected side compared to the contralateral side. In spite of these opposing trends, the peak count was greater for the affected side regardless of the EMG RMS ratio of the affected to the contralateral side.

The shift of the median peak amplitude towards the lower values in subject 1 and 2 is a potential indicator of muscle fiber atrophy. Our finding is consistent with a previous study in the FDI muscle [6]. The authors reported a shift of median peak amplitudes towards lower values, in one-third stroke subjects from the sample, indicating muscle fiber atrophy. It could also be possible that there is a degeneration of larger motor units. The higher number of peaks on the affected side, together with greater number of low amplitude peaks supports this idea that force production was primarily achieved by recruitment of mostly smaller motor units. A larger number i.e., increased recruitment of the smaller motor units as compared to the contralateral side could result in a higher number of peaks. The amplitude of these action potential of the motor units are lower, thereby resulting in lower EMG RMS. The greater number of peaks on the affected side could also be indicative of less superposition of MUAPs.

The results of the two subjects in group 2 could possibly be due to the presence and earlier recruitment of larger motor units. Previous studies have reported enlarged MUAPs in stroke subjects during chronic phase. Following stroke, there could be motoneuron denervation and reinnervation that leads to the reorganization of muscle fiber types. It has also been reported that larger motor units are recruited earlier than smaller motor units [8]. The amplitude histogram distribution, however, does not show significant difference between the affected and the contralateral side in subject 3 and subject 4. As neural drive is an ensemble of discharge timings of the motor neurons, the amplitude of the EMG signal depicts the strength of the neural drive [9]. The differences in EMG amplitude between the affected and contralateral sides may suggest greater neural drive in subjects with higher EMG RMS on the affected side and lower neural drive in subjects with lower EMG RMS on the affected side compared to the contralateral side. However, the EMG signal is also prone to...
amplitude cancellation (overlapping positive and negative phases cancelling each other) thus limiting the accuracy of neural drive estimation.

It has been suggested that under certain conditions EMG signals may reflect the neural drive, i.e. at low contraction levels or in conditions where there is high variability of the neural drive. Based on the above, we can assume that the amplitude of EMG signal may not be a direct estimate of neural drive. Based on the above, we can assume that the amplitude of EMG signal may not be a direct estimate of neural drive. The reduced EMG amplitude in two subjects and increased EMG amplitude in two subjects compared to their contralateral sides could potentially indicate occurrence of different post-stroke changes motor unit control and structure. On the other hand, the increase in peak counts on the affected side compared to contralateral side indicates similar changes in motor unit behavior on the affected side in all subjects that were not measurable with the RMS value of EMG. The presented analysis could be utilized in time-course analysis studies and could assist clinicians to understand the progression of underlying physiological changes in the muscle after stroke.

V. REFERENCES