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Alterations in Spike Amplitude Distribution of the Surface Electromyogram Post-Stroke

Xiaoyan Li, Aneesha Suresh, Ping Zhou, William Zev Rymer

Abstract- We examined surface electromyogram (EMG) characteristics during voluntary isometric activation of the first dorsal interosseous (FDI) muscle in stroke survivors. Five stroke subjects participated in the study. They were instructed to generate isometric contraction at different force levels. The recording was performed in both paretic and contralateral muscles using a matched force protocol. Comparisons of the spike amplitude distribution of the surface EMG signals were made between paretic and contralateral muscles. For a given contraction level, a widened or narrowed spike amplitude distribution was observed in paretic muscles of stroke subjects. Such differences may be induced by degeneration of some spinal motoneurons and/or disorganization of motor unit control properties after stroke.

Key works: motor unit, surface EMG, stroke

I. INTRODUCTION

Degenerative changes in motoneurons following a hemispheric brain lesion were first reported by Charcot in 1879 [1]. Since then, many researchers have investigated changes in properties and control of motor units after a stroke. Concentric needle electromyogram (EMG) studies have reported fibrillation and sharp waves in stroke subjects [2-3]. Reductions of motor unit number and reduced maximum compound muscle action potentials (M wave) have been described in the paretic limbs after a stroke in the electrophysiological studies [1, 4]. Evidence of motor unit degeneration was also reported in muscle biopsies of paretic limbs as atrophy of type II fibers, small angular fibers, and grouped atrophy [2, 5].

It has also been suggested that following motoneuron lesions and muscle denervation, the remaining motor units can be reinnervated through collateral sprouting and branching of the motor neurons during the chronic stage of stroke [6-7]. An increase in motor unit fiber density in the paretic upper limb indicates a rearrangement of the muscle fibers within the motor unit of hemiparetic patients [6, 8]. As a result of reinnervation, fiber type grouping or enlarged motor unit sizes were observed in both morphological and electrophysiological studies [2, 9].

The aforementioned techniques either involve invasive recording (e.g., biopsy or intramuscular EMG), or intensive electrical stimulations, thus limiting their convenient applications. In the current study, we introduce a new method using surface EMG recording to investigate motor unit characteristics post stroke. The method is based on an examination of surface EMG spike amplitude distribution. As a reflection of motor unit property changes, we expect to observe alterations in EMG spike distribution in paretic muscles of stroke survivors, in favor of larger amplitude spikes in paretic muscles.

II. METHODS

A. Subjects

Five stroke subjects with mild to severe weakness of the extremities contralateral to the cerebral lesion side participated in the study. All subjects were recruited using the Clinical Neuroscience Research Registry at the Rehabilitation Institute of Chicago (Chicago, IL, USA). All subjects gave their written consent before the experiment, which was approved by the Institutional Review Board of Northwestern University (Chicago, IL, USA). Clinical assessments were performed prior to the experiment, including spasticity measures at the elbow and assessment of motor recovery after stroke using the Fugl-Meyer test and the Chedoke-McMaster assessment. Table 1 summarizes the demographic information of the 5 stroke subjects.

Table 1. Demographic information of stroke subjects.

ID	Sex	Age	A	FT	C	Duration (years)	Paretic side
1	F	59	1+	29/66	3	19	L
2	F	52		27/30(h)	5	7	R
3	M	62	1	66/66	7	1	L
4	M	53	3+	16/66	2	11	R
5	M	48	1+	24/30(h)	6	1.5	R

A: Ashworth test, FT: Fugl-Meyer test, C: Chedoke-McMaster test, h: hand only

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Xiaoyan Li is with the Rehabilitation Institute of Chicago, 345 E Superior, Chicago, IL, USA (phone: 312-238-1273, email: xiaoyan-li-1@northwestern.edu)

Aneesha Suresh is with the Northwestern University, Evanston, IL, USA (email: aneesha@u.northwestern.edu)

Ping Zhou is with the Northwestern University and the Rehabilitation Institute of Chicago, Chicago, IL, USA (email:p-zhou@northwestern.edu)

William Zev Rymer is with the Northwestern University and the Rehabilitation Institute of Chicago, Chicago, IL, USA (email: w-rymer@northwestern.edu)

B. Experiments

Subjects were seated upright in a mobile Biodex Chair. Their shoulders and waist were tightly strapped to the chair to minimize trunk and shoulder movements. The upper arm was placed on a plastic support and the forearm was casted and strapped in a ring-mount interface (Figure 1a). The proximal phalanx of the index finger was casted and fixed to a small ring-mount interface attached to a six degree-of-freedom load cell (ATI, Apex, NC). This standardized position served to minimize activation of unmeasured muscles.

Surface EMG signals were recorded from the first dorsal interosseous (FDI) muscle using a sensor array (Delsys, Boston, MA, Figure 1b). Detailed description of the sensor can be found in [10]. The output signals from four pairs of electrodes were differentially amplified and filtered with a bandwidth of 20 Hz to 2000 Hz. The forces and surface EMG signals were sampled at 20 kHz using EMGWorks® (Delsys, Boston, MA).

In the beginning of the experiment, subjects performed three trials of isometric maximum voluntary contraction (MVC) of the paretic FDI muscle using index finger abduction. This paretic muscle MVC was used as a standard to calculate the target forces for both paretic and contralateral muscles. The target force trajectory and feedback of the contraction force were displayed on a computer monitor. An example of the target trajectory is shown in Figure 1c. Subjects were instructed to match the target force trajectory using visual feedback of the force output, and to hold the force for 10 s. The target force was set from 20% to 80% of the paretic MVC at 10% MVC increments. Depending on the amplitude of target force, the total duration of trials varied from approximately 20 s to 30 s. All target forces were presented randomly, at least twice in one session. Practice trials and substantial rest period between trials were provided. The recording was performed for both paretic and contralateral muscles.

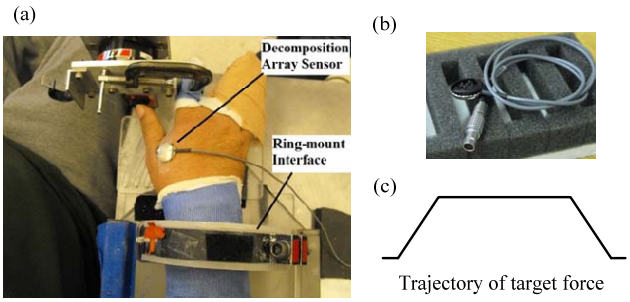

Figure 1. (a) Experimental setup; (b) Four-channel array sensor (Delsys, Boston, MA); (c) an example of trajectory of target force.

C. Data Analysis

All signals were exported and analyzed offline in Matlab® (MathWorks, Natick, MA). An algorithm was developed to detect all the spikes in the surface EMG signals within constant force period. We used the traditional threshold approach. The threshold was determined by

$$Th=u_0 + 3 * \delta \quad (1)$$

where u_0 and δ are the mean and the standard deviation of the background noise of surface EMG.

Then, for each designated matched force, the amplitude of the spikes was detected and the distributions of the spike amplitude were compared between the paretic and contralateral muscles. Due to the large inter-subject variability in the surface EMG signals, comparisons were made within the same subject. The range of the spike amplitude was categorized into 10 equal discrete intervals (or bins). To quantify observations of spike distribution at the right side, the accumulate histogram of bins was calculated:

$$n = \sum_{i=8}^{10} m_i \quad (2)$$

where i is the index of bins, and m_i is the number of observations falling into the ith bin. Spikes within bin 8 to bin 10 were averaged and the value was used to represent the amplitude of larger action potential spikes. The averaged spike amplitude was computed across all force levels for all subjects. Then, the Student's T-Test was performed to compare the differences of the averaged spike amplitude between the paretic and contralateral side for each subject. Statistical significance was set as $p<0.05$.

III. RESULTS

Using the sensor array, we were able to collect four channels of surface EMG in one single trial. Examples of one-channel surface EMG recording from the paretic FDI muscle and contralateral side are shown in Figure 2 for a matched force. Consistent recordings were found in the other three channels. In this subject, the amplitude of surface EMG was visibly larger on the paretic side than on the contralateral side.

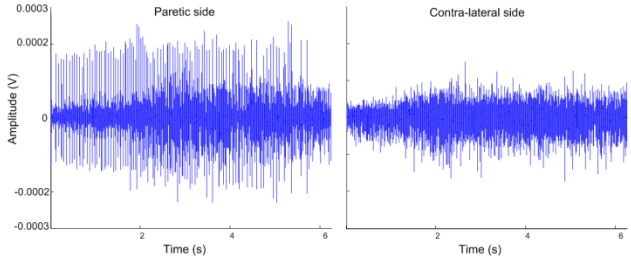

Figure 2. Example of surface EMG signals from the FDI muscles of a stroke subject at matched force. Left: paretic side, Right: contralateral side.

Figure 3 shows examples of the distributions on the paretic and contralateral sides from all subjects. Visual inspection of the histogram showed that three of the five subjects demonstrated a broader range of spike amplitude on the paretic FDI muscles compared with the contralateral side (Figure 3 a-c). We also noticed a shift towards the larger amplitude part in paretic muscles (see the circled area). In table 2, these three subjects showed larger averaged spike amplitude than the contralateral side.

Two subjects demonstrated a narrower range of spike amplitude on the paretic side (Figure 3d-e), and they had smaller averaged spike amplitudes than the contralateral side (Table 2).

Table 2. Comparison of averaged spike amplitude between paretic hand and contralateral side

ID	Paretic (mV)	Contralateral (mV)	p value
2	0.49±0.076	0.358± 0.12	0.03
3	1.36±0.35	1.18± 0.17	0.19
4	0.36±0.04	0.27±0.028	0.04
1	0.13±0.04	0.36±0.21	0.04
5	0.26±0.07	0.567±0.367	0.07

IV. DISCUSSION

The recording and analysis methods used in this study offer a convenient approach towards understanding disorders of motor unit activation in stroke, in that only surface EMG recordings are required. By applying these techniques, we have examined global motor unit characteristics in the paretic hand muscles of stroke survivors and are able to assess the consequences of motor unit alterations following a lateralized brain lesion.

In three subjects, we observed a shift towards larger spike amplitude values on the paretic side, as compared with the contralateral side. This shift may be due to enlarged surface motor unit action potentials, which may conceivably follow motoneuron loss and subsequent enlargement of many remaining motor units as a result of motor axon sprouting and collateral reinnervation in the chronic phase of stroke [9]. This finding is consistent with previous electrophysiological studies in stroke [7, 9, 11].

An alternative explanation for the finding of larger spike amplitudes on the paretic hand could be attribute to inefficient activation patterns of motor units. Gemperline et al. reported that in 50% (3 of 6) of their hemiparetic stroke subjects, there were significant reductions in motor unit mean discharge rate in the paretic muscle compared to the contralateral muscle [12]. Inefficient activation of motor units would necessitate the recruitment of more and larger motor units. As a result, the EMG produced by the muscle on the paretic limb in their study was greater than that produced by the contralateral limb for a given force [13].

The overall shift towards smaller spike amplitude on the paretic side could be an indication of muscle atrophy or activation of low-threshold motor units. Histochemical study of muscle biopsies from the hemiplegic patients showed that type II fibers were most affected by motor neuron degeneration, while the percentage of type I muscle could increase [2].

In keeping with this possibility, in two subjects we found an overall shift of the spike amplitude towards the left (or to smaller values) at the matched force (Figure 3d-e). Previous studies suggested a selective degeneration of the large motor units following a stroke, for example, no differences was found between the motor unit action potentials at high and low force output on the paretic side

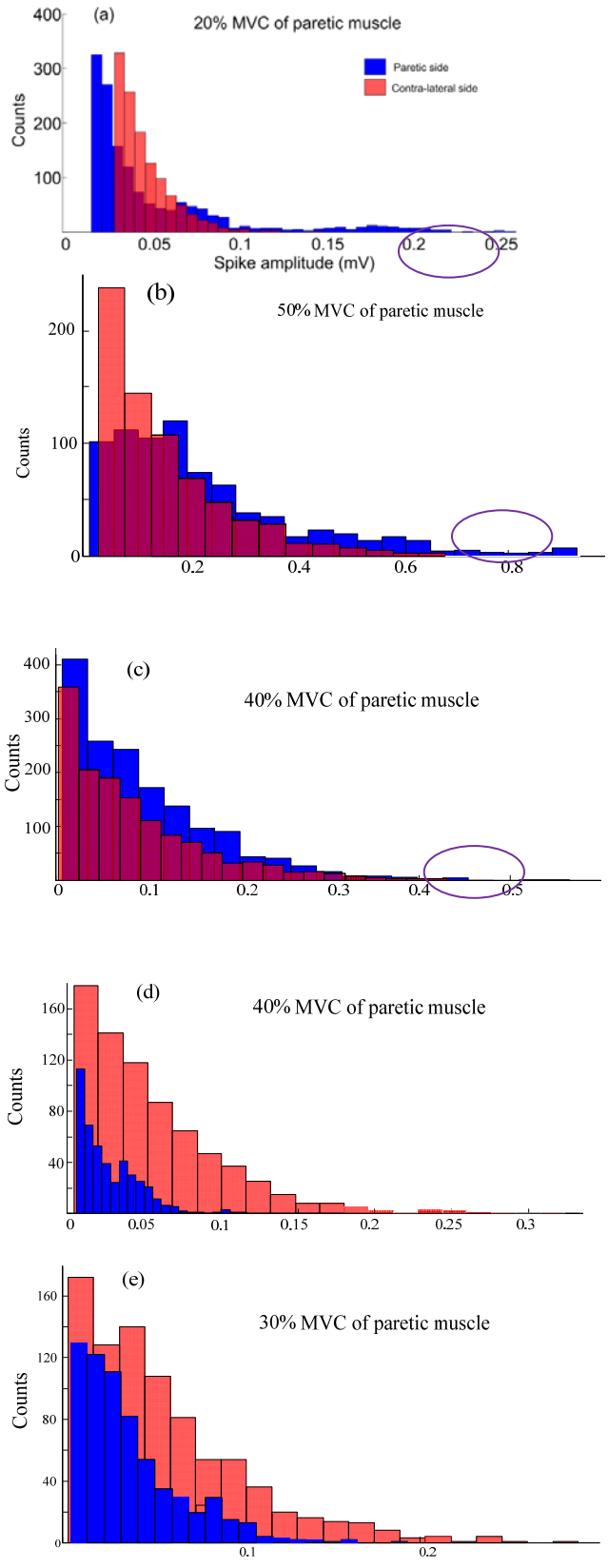

Figure 3. Histogram of spike amplitude of five stroke subjects. The contraction level varies in subjects. (a): subject 4; (b): subject 3; (c): subject 2; (d): subject 1; (e): subject 5. Overlapping columns are displayed in purple.

in the macro EMG recording [14]. The larger motor units were reported to be located in more superficial regions of the muscles [15]. Therefore, it is possible that the surface EMG signal recorded from the two subjects were drawn from smaller motor units located in relatively deep regions of the muscle, generating the same force as the contralateral muscle.

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