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A Preliminary Study of Action Potential Propagation in Paretic Muscle of Stroke Survivors

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***Abstract*—Individual motor unit action potential (MUAP) propagation along the muscle fibers was assessed in paretic muscle of stroke survivors. Single motor unit activity was recorded from the biceps brachii muscle on the paretic side of stroke survivors using a surface electrode array. Among the 15 clearly identified low-threshold motor units, six units showed little delay between MUAPs on different electrodes aligned along muscle fibers, resulting in abnormally high values of calculated propagation velocities, while clear action potential delay was observed for the other motor units. Several factors that possibly lead to this observation are discussed.**

I. Introduction

AFTER a hemiparetic stroke, motor control of one or more extremities on one side of the body may be affected. Many patients suffer a variety of physical symptoms, such as spastic hypertonia, muscular weakness, and impaired movement coordination. Muscle weakness after a stroke could be the result of different causes such as disuse, vasomotor changes, and loss of central motor neuron trophic influences. In addition, trans-synaptic degeneration of lower motoneurns after an upper motor neuron lesion, has also been suggested, mainly based on electrophysiological studies, e.g., needle electromyogram (EMG) based quantitative motor unit action potential (MUAP) analysis [1][2], and incremental stimulation based motor unit number estimation [3]. In this study, a 2-dimensional surface electrode array was used to record single motor unit activity from paretic muscle of stroke survivors. Our aim was to take advantages of the multi-channel recording to assess, in these muscles, the possible pathological alterations in MUAP propagation patterns.

II. Materials and Methods

A. Subjects

Nine hemiparetic stroke subjects participated in the study. They all gave informed consent via protocols approved by the Institutional Review Board under the Office for Protection of Human Subjects at Northwestern University.

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B. Data Recording

Each subject was seated upright in a Biodex chair and positioned in a standardized posture with the forearm resting comfortably on an arm base [4]. A flexible 2-dimensional surface electrode grid (Figure 1) was used to record EMG signal from the affected side. The electrode array was placed on the medial biceps brachii, such that each column ran along the muscle fibers (Fig. 1). Subjects were asked to isometrically contract the biceps to recruit an identifiable single motor unit and maintain a steady unit firing rate. EMG signals were amplified by a Refa128 system (TMS International, Amsterdam, the Netherlands), with a sampling rate of 2000 Hz/channel. The EMG data were stored in a laptop for off-line analysis.

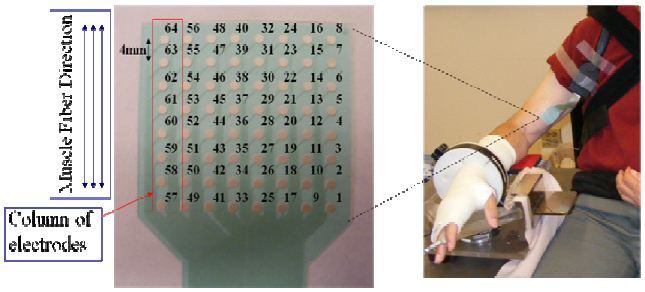

Fig. 1: Experimental setup. The flexible high density 2D surface EMG electrode (left) is labeled by channel for reference. The grid was placed on the medial biceps with electrode columns oriented along the muscle fibers (right).

C. Data Analysis

Each trial of surface EMG data was digitally filtered through Spike2 software (CED Ltd, Cambridge, UK) using IIR notch filters to remove 60Hz noise and its harmonics. Instances of single motor unit firing were identified on each channel. Motor unit conduction velocity was estimated to assess MUAP propagation patterns. Cross-correlations of two signals within a linear electrode column (along muscle fibers) were calculated. Twenty 1-second data segments were used for this calculation. The time shifts of the correlation maxima represent the time used to travel the specified inter-electrode distance. This distance divided by the time shift represents an estimation of propagation velocity. All possible correlations from two electrodes within an electrode column were calculated. The values from the measurement distance generating the lowest standard error of the estimation were adopted.

III. Results

First, the optimal measurement distance for MUAP propagation velocity estimation was determined by

comparing relative standard deviations of the estimated values from each distance. We found that velocities estimated from distances 4 mm - 12 mm generally produced highly variable results, while an inter-electrode distance of 16 mm or greater (5-8 electrodes) produced more stable estimation of action potential conduction velocities.

For each of the patients, we tracked the activity of one or multiple motor units. In total 15 low-threshold single motor units were identified from the paretic muscles for the propagation analysis. We observed that four of the 15 units had almost no delay between the recording electrodes on the column along the muscle fiber s. It is worth noting that those motor units were recorded when the subjects had no intension of voluntary contraction, i.e. they self-reported that they were at rest during the experiment.

In addition, we found that two of the 15 motor units had little delay between the adjacent electrodes along muscle fibers. This delay generated an abnormally high values (>10m/s) of calculated motor unit conduction velocities. For the rest of the motor units, a clear delay was observed between the MUAPs along the muscle fibers, generating motor unit conduction velocities within a physiologically reasonable range [5].

IV. Discussion

Several electrophysiological studies have investigated the pathological alterations in motor unit structural and functional properties in paretic muscles of stroke survivors. This was performed primarily using invasive needle EMG examinations [1][2] and motor unit number estimation [3]. For example, from needle EMG, it was found that the spontaneous activity and the mean number of MUAP phases and turns were significantly higher on the paretic side, and the outliers above maximum for MUAP duration and amplitude on the paretic side were significantly higher than those on the contralateral side. Very recently, Kallenberg et al. [6] used surface electrode arrays to examine hemiparetic stroke survivors. They found that the mean frequency of the power spectrum of the extracted surface MUAPs was smaller on the affected than on the contralateral side, indicating an increased contribution of low-threshold motor units, possibly related to degeneration of high-threshold motor units. Furthermore, the ratio of the root mean square value of the surface MUAPs on the affected side divided by that on the unaffected side correlated significantly with the Fugl-Meyer score, suggesting different extent of muscle fiber reinnervation on the affected side [6].

In our current study, we used a surface electrode array to track MUAP propagating patterns in the paretic muscle of stroke survivors. We found abnormally high propagation velocities could be observed for a limited number of motor units. One possible explanation of this observation is that this may be a manifestation of muscle fiber reinnervation resulting in a widening of the neuromuscular junction. Placing the electrodes on the neuromuscular junction significantly increases the calculated value of motor unit conduction velocity. Abnormal conduction velocities in the biceps brachii muscles were also reported in a surface EMG examination of Duchenne muscular dystrophy patients because of "pathological longitudinal spread of end-plates" [7]. Another possible explanation is that the motor units were located relatively deep in the muscle. Note that the high values of the calculated conduction velocities were observed most consistently in involuntarily firing 'spontaneous' units, which were most likely highly excitable, low threshold units [8]. A flexible surface electrode array, designed to facilitate recording from an uneven surface of a muscle, was used in this study. Although it is more suitable for recording from small muscles (e.g., hand muscles) compared with rigid electrode arrays, it may not have sufficient resolution to capture MUAP propagation of biceps, due to the extent and constitution of the subcutaneous tissues of the muscle. It is also worth noting that a special design of the recording system may be another factor causing abnormal MUAP propagation patterns. Although the multi-channel EMG amplifier has a common reference, the output of each channel uses the average signal of all the channels as the actual reference. This design may impose a common signal component for all the recording channels, resulting in observation of abnormal MUAP propagation patterns. Finally, this is a preliminary study. No motor unit data on contralateral side were recorded for comparison. Moreover, the flexible surface electrode array only covers a small portion of the biceps muscle. To more accurately track MUAP propagating patterns along the muscle fibers, an electrode array spanning the entire biceps is required to record motor unit activity [9]. All these will be included in our future experimental protocols.

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