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Analysis of Muscle Fiber Conduction Velocity during Finger Flexion and Extension after Stroke

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Abstract

Background—Stroke survivors experience greater strength deficits during finger extension than finger flexion. Prior research indicates relatively little observed weakness is directly attributable to muscle atrophy. Changes in other muscle properties, however, may contribute to strength deficits.

Objectives—This study measured muscle fiber conduction velocity in a finger flexor and extensor muscle to infer changes in muscle fiber type after stroke.

Methods—Conduction velocity was measured using a linear EMG surface electrode array for both extensor digitorum communis and flexor digitorum superficialis in 12 stroke survivors with chronic hand hemiparesis and 5 control subjects. Measurements were made in both hands for all subjects. Stroke survivors had either severe (n=5) or moderate (n=7) hand impairment.

Results—Absolute muscle fiber conduction velocity was significantly lower in the paretic hand of severely impaired stroke patients compared to moderately impaired patients and healthy control subjects. The relative conduction velocity between the two hands, however, was quite similar for flexor muscles across all subjects and for extensor muscles for the neurologically intact control

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subjects. However, muscle fiber conduction velocity for finger extensors was smaller in the paretic as compared to the non-paretic hand for both groups of stroke survivors.

Conclusions—One explanation for reduced conduction velocity may be a type II to type I muscle fiber, especially in extrinsic extensors. Clinically, therapists may use this information to develop therapeutic exercises targeting loss of type II fiber in extensor muscles.

Keywords

Stroke; Muscle; Conduction Velocity; Finger; EMG; Fiber

Introduction

Stroke survivors may experience profound impairment of the contralesional upper extremity. While a number of mechanisms contribute to varying degrees to the diminished motor control, such as contracture¹, muscle atrophy^{2,3}, spasticity⁴, and excessive coactivation⁵, weakness plays a fundamental role⁶. This weakness is especially noticeable in the hand where isometric force generation can be reduced by 80% or more^{7,8}. We have observed that force deficits during isokinetic movements are even greater⁹. These power deficits are of substantial functional importance as force generation during dynamic movement is a common requirement for task completion. Due to the massively parallel actuator structure of muscle, both force and length can be controlled simultaneously in the limbs. For example, for a given speed of hand closing either a large or a small grip force can be created, as needed.

Alternatively, changes in the motor unit morphology may contribute to the observed strength impairment. A change in muscle fiber type may have considerable effects on weakness by altering both the timing of force generation and strength capacity of the muscle. Specifically, a shift toward type I (slow twitch) muscle fibers from type II (fast twitch) fibers would decrease the ability of stroke survivors to generate force, particularly at high movement velocities. Indeed, some studies have suggested preferential atrophy of type II muscle fibers following stroke^{10,22,12} and even show signs of type I hypertrophy in response to physical activity¹³. Similarly, Dattola and colleagues³ observed an actual shift in the ratio of type I to type II fibers in the gastrocnemius muscle post-stroke suggesting a denervation-reinnervation process. Yet, other studies have shown an increase in the proportion of fast-twitch to slow-twitch fibers in the hemiparetic leg^{14,15}. Thus, possible changes in muscle composition in stroke survivors are not well understood and may vary depending upon individual muscles or muscle groups. Additionally, certain motoneurons, such as those innervating type II fibers, may be more difficult to recruit voluntarily after stroke, as they are generally larger and therefore require greater synaptic excitation to activate. We have previously seen that while muscle atrophy was fairly limited post-stroke², voluntary activation deficits were large¹⁶.

The purpose of this study was thus to examine potential changes in activation of different fiber types in key hand muscles after stroke. We employed muscle fiber conduction velocity (MFCV) as a means to infer changes in muscle fiber type. Using surface EMG to evaluate MFCV is a non-invasive means of evaluating differences in muscle fiber characteristics^{17,18}.

A linear EMG electrode array was used to measure changes in MFCV for both an extrinsic finger flexor muscle, flexor digitorum superficialis (FDS), and an extrinsic extensor muscle, extensor digitorum communis (EDC). By using EMG, rather than other techniques such as biopsy, we were able to address questions regarding the types of fibers actually being voluntarily activated, not just present in the muscle. *In accordance with previously observed strength and power deficits in the stroke hand, we hypothesized MFCV measured by the EMG signal would be lower in paretic compared to the non-paretic hand muscles.*

Materials and Methods

Subjects

Twelve chronic stroke survivors (9 male, 3 female) between age 41 – 71 (mean $\pm$ SD; 55 ± 7 yrs) and 5 healthy control subjects ranging from age 21 - 36 (mean $\pm$ SD; 28 ± 7 yrs; $n=5$) participated in this research study. Stroke survivors had experienced a single stroke, were 1 to 7 years post-injury and had chronic hemiparesis which included the hand (7 right, 5 left). Prior to the experiment the subjects were rated by a clinician as having moderate (Stage 4 or Stage 5, $n = 7$) to severe (Stage 2 or Stage 3, $n = 5$) hand impairment using the Stage of Hand portion (maximum 7 stages) of the Chedoke-McMaster Stroke Assessment¹⁹. In this assessment, Stage 2 indicates spasticity is present and felt as a resistance to passive movement, Stage 3 has marked spasticity but voluntary movement is present within synergistic patterns, in Stage 4 spasticity diminishes, and in Stage 5 spasticity wanes but is sometimes evident with rapid movement particularly at the extremes of range. All subjects were tested in both hands and were required to have no diagnosis of other neurological or orthopaedic disorders interfering with neuromuscular function. Informed consent was obtained from each subject in accordance with the protocol approved by the Northwestern University Institutional Review Board and experiments were conducted in accordance with the Declaration of the World Medical Association. Table 1 summarizes individual subject characteristics.

Experimental Protocol

Muscle fiber conduction velocity was measured using surface EMG. Prior to the experiment the subject was briefed on the study objectives. Subjects were seated with their arm relaxed at their side; the desired muscle for testing (FDS or EDC) was located through palpation. A custom, linear, 20-channel bar electrode array with an inter-bar distance of 5 mm was affixed to the skin over the desired muscles. A single low amplitude electric pulse (< 10 mA) was applied to the nerve (median or radial) innervating the desired muscle (FDS or EDC, respectively) using a Digitimer High Voltage Stimulator (Model DS7A, Digitimer, Inc., Hertfordshire, England) to locate the motor point with respect to the electrode array. The pulse was administered using a stimulating bar electrode with two 9mm contacts with 30 mm inter-electrode distance. The position of the bar electrode on the nerve was adjusted by the experimenter until resulting movement produced index finger flexion (for FDS activation) or index finger extension (for EDC activation). After isolating appropriate movement, EMG data were recorded from the linear bar electrode (Fig 1) with a Refa128 advanced high-density EMG recording system (TMS International, Amsterdam, Netherlands). EMG data were bandpass filtered (10 – 450 Hz) prior to sampling at 2000 Hz.

Next, the subject exerted a weak contraction by either lightly flexing (for FDS recordings) or extending (for EDC recordings) the MCP joint for 6 s while EMG data were recorded.

Data Analysis

For all trials, nineteen bipolar signals were calculated from the 20 monopolar channels using:

$$\overline{B}_n = \overline{M}_{n+1} - \overline{M}_1, \text{ for } n=1 \text{ to } 19 \quad (1)$$

where $\overline{B}$ represents an array of bipolar signals and $\overline{M}$ represents the arrays of monopolar data directly sampled. Data collected during stimulation trials were visually scanned to estimate the motor point by identifying the bipolar signal with the least change in amplitude. MFCV was calculated from the voluntary contraction trials using the first and third bipolar signals distal to the motor point:

$$CV = \frac{\Delta d}{\Delta t} \quad (2)$$

where d is the fixed distance (10 mm) between the two electrodes and t is the time delay between the two signals estimated using the maximum of the cross-correlation technique. For each subject, MFCV was determined as a mean of 8 samples of data analyzed spanning the 6 s trial (1 sample at every 0.5 s intervals).

Statistical Analysis

After a Shapiro-Wilk Test was conducted to assess normality of data, a repeated measures ANOVA was performed to test the effect of the independent variables subject Group (3 levels), Muscle (2 levels) and Hand (2 levels) on the dependent variable (MFCV) using SPSS Statistics 20.0 software (SPSS, Chicago, IL). Posthoc Tukey tests were conducted when a variable was determined significant according to the initial ANOVA. Significance was accepted at $p < 0.05$. Subsequently, individual within-group comparisons were made using separate ANOVAs to account for severe stroke subjects showing a significant effect in contrast with the other groups. In this case, a Bonferroni correction was taken to account for the separate analyses and the significance level was set to $\alpha/2 = 0.025$.

Results

This study assessed MFCV in paretic and non-paretic flexor and extensor muscle of stroke survivors comparing it to that recorded from the dominant and non-dominant hand of healthy control subjects. The initial ANOVA revealed that Group ($p = 0.038$) and Muscle ($p = 0.005$) had a significant effect on MFCV. Posthoc analyses indicate that mean MFCV from the severely impaired stroke survivors (mean MFCV = 1.34 ± 0.75 m/s) were significantly lower than those in the moderately impaired group (1.85 ± 0.78 m/s, $p = 0.033$). Additionally, the overall mean MFCV measured in the FDS muscle (1.42 ± 0.73 m/s) was significantly lower than that recorded from the EDC muscle (1.90 ± 0.64 m/s, $p = 0.005$).

Figure 2 depicts MFCV summary data for control, moderately impaired stroke, and severely impaired stroke subjects.

For healthy control subjects, MFCV recorded from FDS ($\text{MFCV}_{\text{Dom}} = 1.52 \pm 0.49$ m/s; $\text{MFCV}_{\text{Non-Dom}} = 1.41 \pm 0.22$ m/s; $p = 0.656$) and EDC ($\text{MFCV}_{\text{Dom}} = 1.93 \pm 0.43$ m/s; $\text{MFCV}_{\text{Non-Dom}} = 2.02 \pm 0.49$ m/s; $p = 0.201$) muscle is very similar between the dominant and non-dominant arms. Additionally, FDS data from stroke subjects with moderate ($\text{MFCV}_{\text{Non-Paretic}} = 1.53 \pm 0.93$ m/s; $\text{MFCV}_{\text{Paretic}} = 1.75 \pm 1.01$ m/s; $p = 0.262$) or severe ($\text{MFCV}_{\text{Non-Paretic}} = 1.03 \pm 0.32$ m/s; $\text{MFCV}_{\text{Paretic}} = 1.12 \pm 0.69$ m/s; $p = 0.777$) impairment indicate no significant difference between MFCV in the paretic and non-paretic arm. No significant trends are noticeable in individual subject data either for FDS MFCV (Fig. 3). However, MFCV recorded from the EDC muscle in both moderately ($\text{MFCV}_{\text{Non-Paretic}} = 2.27 \pm 0.21$ m/s; $\text{MFCV}_{\text{Paretic}} = 1.86 \pm 0.38$ m/s; $p = 0.021$) and severely ($\text{MFCV}_{\text{Non-Paretic}} = 2.04 \pm 0.64$ m/s; $\text{MFCV}_{\text{Paretic}} = 1.17 \pm 0.71$ m/s; $p = 0.006$) impaired subjects was significantly lower in the paretic arm. These findings were very consistent across subjects (Fig. 3).

Discussion

Loss of strength in the hand is profound after stroke and contributes substantially to overall impairment⁶. Weakness in the fingers is asymmetrical, such that relative deficits in generation of extension force are greater than relative deficits in peak flexion force. This study, through measured changes in muscle fiber conduction velocity, suggests a change in muscle fiber type may be at least partially responsible for the severe extension deficits observed in finger extensor muscles.

MFCV as an Indicator of Muscle Fiber Type

MFCV has long been considered a parameter of the size principle of muscle fiber¹². Early research studies indicated the electric signal propagating across fast fibers moved at a higher rate than across slow fiber in both cats²⁰ and humans²¹. MFCV has previously been linked to several other factors such as temperature²², muscle length²³, fatigue^{24,25,26}, contraction force^{25,27,28}, and gender^{29,30}. Due to the light contraction utilized in this experiment and the controlled nature of the lab environment, we expect there was little effect of temperature, muscle length, fatigue or contractile force on individual readings. Additionally, at least one study of MFCV in myopathy found no significant difference between patient age or gender³¹. Therefore, we believe we can accurately deduce that observed changes in MFCV are linked to muscle fiber type. While no known studies have directly reported the values of MFCV in the EDC and FDS muscle, the values recorded in this study were similar, but relatively lower than MFCV reported in larger muscles such as the biceps^{18,29}. One explanation for lower MFCV calculations is that due to FDS and EDC muscle orientation the electrode array may not have been exactly parallel to the muscle fibers, such that the action potentials actually travel a slightly longer distance than the 10mm inter-electrode distance used for calculations. Additionally, it should be noted that because surface EMG records a summation of signals originating from several motor units, a wide range of MFCV contribute to the velocity recorded using surface electrodes. Despite the summation of

signals, a direct relationship between muscle fiber composition and MFCV still exists when measuring from the surface of the skin¹⁷.

Muscle Fiber Type

It is widely accepted that type I (slow twitch) fibers are present in muscles exhibiting high endurance and type II (fast twitch) fiber is favored in muscles exhibiting high power³². In actuality, the distribution of muscle fiber type varies significantly not only between muscle groups but also within a single muscle itself. For example, autopsy studies indicate the composition of peripheral fiber rather consistently has a higher proportion of type II than type I fibers³³ in comparison with that deep in the muscle. Therefore, when using surface EMG to evaluate MFCV it should likely be assumed the assessment most accurately evaluates the muscle fiber composition towards the surface of the muscle. The following is a discussion of the results as they may relate to muscle fiber type.

In both healthy subjects and stroke patients we recorded higher MFCV in the EDC muscle as compared to the FDS muscles. This may be an indication that the EDC has a tendency for relatively greater proportion of fast twitch fibers than is found in FDS muscle. Indeed, it would be intuitive that FDS muscle, due to the finger flexor's role in maintaining grip which could be considered an endurance activity. This is in accordance with findings of high proportions of type I fibers in intrinsic hand muscles that are highly involved in gripping³⁴. In contrast, precise, rapid finger extension is needed to control release of a thrown object³⁵. Another, secondary explanation could be the manner in which the muscle fiber is grouped within the muscle. In at least one toe extensor, the muscle fiber was found to have a different histology than toe flexors in that type II fibers were highly grouped towards the surface of the muscle³². Further autopsy studies of the muscle are needed to determine if such grouping is also present in finger extensors. If this were the case, it might explain this study's observations.

The most important finding of this study was that MFCV was lower in the paretic EDC muscle than in the non-paretic EDC of stroke patients. This finding correspond with EMG studies assessing hemiparetic biceps brachii muscles which attribute reduction in MFCV to altered motor unit and muscle fiber after stroke³⁶. The measured shift may indicate an increase in the proportion of active type I fibers to type II fibers, which could result from motor unit dysfunction, preferential atrophy of type II fibers and/or a denervation-reinnervation of fibers occurring over time. Some lower extremity studies, for example, point to preferential atrophy of type II fibers^{11,12,37} which may be accompanied by type I hypertrophy¹³. However, contradicting these results, an increase in the proportion of fast twitch fibers has been observed in the paretic vastus lateralis muscle³⁸. Thus, changes in fiber type after stroke may be specific to certain muscle groups or depend upon the action of a muscle. The seemingly contradictory results in the literature could also arise from the chosen methodology. Most research to date evaluating the muscle itself has been conducted using muscle biopsy. Biopsy typically samples only a very small portion of the muscle fiber which may not provide a globally accurate depiction of the muscle composition. Perhaps the most accurate indication of muscle composition are those studied by autopsy opposed to biopsy which only allows analysis of a small sampling of muscle fiber.

A limitation of this study is that finger movements were not standardized by force production or EMG biofeedback. A variation in contraction intensity or effect of high/low threshold motor units could confound the results. We believe that force level has little effect on our results as study participants were asked to lightly flex or extend their fingers at the MCP joint. We do not believe any were producing any significant magnitude of force in either movement direction. However, future studies may investigate changes in MFCV in passive versus active levels of varying force production in paretic muscle.

Clinical Implications

A relative decrease in type II fiber, representing a greater loss of strength in extensor muscles, provides one possible explanation for previous findings of relatively greater strength deficits during isometric⁷ or isokinetic⁹ **extension** tasks compared to **flexion** tasks. Clinically, this information could be very useful in the development of therapeutic exercises targeting the loss of type II fiber in extensor muscles. By using strength or endurance training targeted at specific fiber types a therapist could help improve strength in extensor muscles, or prevent the loss of strength over time.

At this time, further research is needed in three additional areas for the benefits of therapy: (1) to determine the time course of the loss of type II muscle fiber, (2) to determine if observed differences in MFCV are due to physical changes in the fiber, or the inability to activate fast twitch motor units, and (3) to evaluate various treatment approaches targeting type I and type II fibers in moderately and severely impaired patients. If the changes do not occur immediately with the onset of injury (as would likely be the case for reinnervation or preferential atrophy) a therapeutic regime could be developed to intervene before changes in muscle fiber composition occur. If the alterations occur immediately with injury, as may be the case with lost motor unit activation, appropriate exercises may be developed to restore function in the muscle to prevent further degradation of muscle function. Since evidence exists suggesting an inability to recruit Type II fibers post- stroke³⁹, tracking therapeutic interventions targeting activation of such fibers would provide valuable insight into appropriate approaches to rehabilitation. Potential interventions known to target Type II fibers include training on isokinetic tasks at various movement velocities⁴⁰ or electric stimulation of muscle fiber⁴¹.

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Figure 1. Experimental Set-Up

A) The desired nerve was stimulated in the upper arm. EMG activity was recorded in the FDS or EDC in the forearm. B) A 20-channel bar electrode array with an inter-bar distance of 5 mm was used to record muscle activity. C) Sample of EMG signals recorded for the EDC muscle across 20 channel linear electrode array. The location of the motor point is estimated to be near Ch 7 & 8 due to lack of amplitude change in signal.

Figure 2. MFCV Summary Data

MFCV recorded from the right and left arm of healthy control subjects, moderately impaired stroke subjects, and severely impaired stroke subjects.

Figure 3. MFCV Data by Subject

Individual subject data for MFCV recorded from EDC and FDS muscles in control and stroke subjects.

Summary of Subject Characteristics

Table 1

Subject ID ^a	Gender ^b	Age	Affected or Dominant Hand ^c	Years Post-Injury	Chedoke Staged ^d
S1	F	53	R	4	5
S2	M	71	L	3	4
S3	M	62	R	4	4
S4	M	49	R	2	5
S5	M	49	R	2	4
S6	M	56	R	1	5
S7	M	52	L	2	3
S8	F	41	L	6	2
S9	F	58	L	7	3
S10	M	51	R	4	2
S11	M	59	R	1	2
S12	M	54	L	3	2
C1	M	22	R		
C2	M	21	R		
C3	F	34	R		
C4	M	28	R		
C5	M	36	R		

^aS, Stroke; C, Control

^bF, Female; M, Male

^cR, Right; L, Left; Affected Hand, Stroke; Dominant Hand, Control

^dBased on stage of hand portion of the Chedoke-McMaster Stroke Assessment (max: Stage 7) (Gowland et al., 1993)