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Abstract:

The purpose of this study was to use high density surface EMG recordings to quantify stroke-related abnormalities in motor unit firing behavior during repeated sub-maximal knee extensor contractions. A high density surface EMG system (sEMG) was used to record and extract single motor unit firing behavior in the vastus lateralis muscle of 6 individuals with chronic stroke and 8 controls during repeated sub-maximal isometric knee extension contractions. Paretic motor unit firing rates were increased with subsequent contractions (6.19±0.35 pps vs 7.89±0.66 pps, P <; 0.05) during task phases of torque decline as compared to controls (6.95±0.40 pps vs 6.68±0.41 pps). In addition, corresponding rates of torque decline were decreased for the paretic leg as compared to the non-paretic leg. These results suggest that regulation of declining forces may be impaired post stroke due to prolonged firing of paretic motor units.

SECTION I. Introduction

Many stroke survivors have long term motor impairments of their legs that limit walking at home and in the community[1]. Although the mechanisms of motor impairments are multi-factorial, deficits in force generating capabilities of the paretic leg have been associated with abnormalities in the kinematics and kinetics of walking [2]–[3][4]. After stroke, there is an obvious loss in force production in the muscle [5] due to altered *net* descending drive from the damaged motor cortex[6]–[7][8][9][10][11][12][13]. In addition, there is indirect evidence of plastic changes to the intrinsic electrical properties of motoneurons post stroke that result in inappropriate activation of the musculature and force generation, during resting conditions and during relaxation phases of contraction [14], [15]. The authors attribute the prolonged activation of paretic musculature to motoneuron hyperexcitability due to dysregulated persistent inward currents.

In humans, motoneuron firing behavior cannot be directly measured, however, measurements of muscle fiber motor unit firing is representative of spinal motoneuron firing behavior because they fire in a 1: 1 manner [16]. One mechanism of force regulation of the muscle is through rate coding of the motor units (increasing or decreasing the rate of motor unit firing). Studies examining motor unit firing behavior in chronic stroke survivors during non-fatiguing contractions have shown abnormalities [17]–[18][19][20] compared with controls and the non-paretic leg that could interfere with descending

commands such as (1) lower recruitment thresholds, (2) more depolarized resting membrane potential and (3) spontaneous firing.

Many of the previous studies have focused on alterations in paretic motor unit firing behavior during single repetitions of a force matching task. Less is known about paretic motor unit firing behavior across multiple repetitions. Quantifying the firing behavior over multiple repetitions of sub-maximal contractions is not only functionally relevant (i.e. walking, ascending stairs), but may expose changes in paretic motoneuron excitability due to a wind-up phenomenon seen in other patient populations[21], [22].

SECTION II. Methods

A. Subjects

The experimental procedures involving human subjects described in this paper were approved by the Institutional Review Board. All participants gave informed consent before participating in the study activities. Six subiects with chronic hemiparetic stroke (4 male, 2 female, 58.67±3.91 years, LE Fugl-Meyer 25/34±3 and eight age matched neurologically intact subj ects participated in the study (5 male, 3 female, 60.63±3.43 years). Stroke subject inclusion criteria included: single, unilateral stroke (obtained through verbal communication from the physician and consistent with neurological physical examination); able to ambulate at least 30 feet (with or without an assistive devicej. ≥6 months post stroke. Stroke subject exclusion criteria included: history of multiple stroke; brainstem stroke; any uncontrolled medical condition; contractures of any lower extremity joints; inability to follow 2–3 step commands.

B. Torque Measurements

Participants sat on a Biodex chair with their knee flexed to 70° and the leg securely attached to the Biodex attachment four cm above the lateral malleolus. This enabled the testing of isometric knee contractions. The isometric torque was measured with a six degree-of-freedom load cell (JR3 force-torque transducer) sampled at 2048 Hz. An EMG-USB2+ amplifier (256-channel regular plus 16-auxiliary channels, OT Bioelettronica, Turino, Italy) was used to record the force channel. This set-up was used to accurately measure the torque (Nm) produced by the knee extensor muscles in the sagittal plane (z-axis).

C. Surface EMG Measurements

Surface EMG was obtained using a 64 channel 2-D electrode array (13 rows, 5 columns). A double sided adhesive sticker designed for and compatible with the array was placed over the array. The holes within the adhesive sticker were filled with conductive electrode paste. The array was placed over the vastus lateralis, midway between the patella and the greater trochanter. The signals for each channel were bandpass filtered between 10 and 500 Hz and amplified by 1000 (v/v) using the EMG-USB2+ amplifier. The signal was then sampled at 2048 Hz and recorded using the OT Biolab software throughout the duration of the experimental protocol.

D. Experimental Protocol

All control subjects performed the protocol with the right leg, and all stroke subjects performed the protocol with the paretic leg. Each participant first performed 3-5 baseline maximum voluntary contractions (MVC) of the knee extensor muscles. The peak force of all the trials was used as the MVC.

After at least one minute rest period following the final MVC, subjects performed the ramp and hold protocol. The subjects were instructed to contract their knee extensors and generate torques in order to trace a trapezoid trajectory displayed on a computer screen. Real-time visual feedback was provided to the subject indicating the torque produced by the knee extensor muscles. The trapezoid was 16 s in duration consisting of a 4 s rising phase ("Incline," beginning at 5% of MVC), 4 s hold phase ("Hold," 20% of MVC), a 4 s decline phase (back to 5% of MVC), and a 4 s "valley" phase (holding at 5% of MVC). One trapezoid cycle was repeated 5 times before completely relaxing the muscle. At least 1 week prior to the testing session, subjects completed a familiarization session where they were allowed to practice all study procedures.

E. Data Processing and Statistical Analysis

Data was processed with Matlab. Torque was zero phased lowpass filtered at 10Hz using a 2^{nd} order Butterworth filter prior to analysis. The torque was normalized to the ratio of the torque value (Nm) over the max torque value of a ramp and hold cycle. A linear fit was performed over the 4 s phases of each trapezoid trajectory and the slope was recorded for each phase.

Individual EMG channels were visually examined to remove noisy channels. The remaining channels were decomposed to attain information of single motor units. Motor unit discharge rates were extracted from the surface EMG recordings. Multichannel blind source separation using convolution kernel compensation (CKC)[23] was used to identify the motor unit discharge patterns. Identified motor units with a pulse-to-noise ratio (PNR) [24]of less than 35 dB were removed from further processing.

Firing rates (pulses per second - pps) of single motor units were calculated by taking the inverse of the inter-spike interval. The mean and coefficient of variation of the firing rates for each motor unit were determined. Further, the mean and coefficient of variation of the firing rates were determined for each 4 s phase (incline, hold, decline, and valley) of the trapezoid trajectory for the ramp and hold cycles.

The firing rates of all the motor units were combined and smoothed using a 4th order, loWpass Butterworth filter with a cutoff frequency of 2 Hz. The mean firing rate and coefficient of variation was determined. The smoothed data was normalized to the ratio of the firing rate over the max firing rate and time locked with the torque trace. A linear fit was performed on the smoothed data coinciding with each 4s section of the trapezoid trajectory and the slope was recorded for each phase.

Data are reported as the mean ± standard error. Separate t-tests (α=0.05) Were performed to compare the following variables for the first and fifth cycles of the trapezoid trajectory for both groups (stroke and control): mean firing rates during the (1) Incline, (2) Hold, and (3) Decline phase, and the normalized slope during the Decline phase.

SECTION III. Results

The average number of motor units processed after decomposition for the pre-fatigue and post-fatigue ramp and hold cycles were greater for the controls than for the stroke subjects. An average of 10.75 and 8.20 motor units were processed for the ramp and hold cycles for the controls and stroke subjects, respectively (Table 1).

Figure 1 shows the average firing frequencies for cycle 1 and cycle 5 of the ramp and hold cycles for both stroke and controls. The motor unit average firing frequency for stroke subjects significantly increased during the decline phase of cycle 5 when compared to the decline phase of cycle 1 for the ramp and hold ($P<0.05$). The decline phase of cycle 1 had an average firing frequency of 6.19±0.35 pps compared to 7.89±0.35 pps for the decline phase of cycle 5. Cycle 1 had firing frequencies of 7.96±0.41 pps and 8.07±0.36 pps for the incline and hold phases, respectively. Cycle 5 had firing frequencies of 8.86±0.35 pps and 8.91±0.53 pps for the incline and hold phases, respectively. The incline and hold phases for the stroke subjects were not significantly different for cycle 1 and cycle 5. Motor unit firing frequencies for control subjects were not significantly different between cycle 1 and cycle 5 of the ramp and hold cycles. Control's incline firing frequencies for cycle 1 and cycle 5 were 7.74±0.36 and 8.02±0.34, respectively. The hold phase firing frequencies were 9.82±0.37 and 9.89±0.36 for cycles 1 and 5, respectively. Cycle 1 had a decline phase firing frequencies of 6.95±0.40 compared to a firing frequency of 6.68±0.41 for cycle 5.

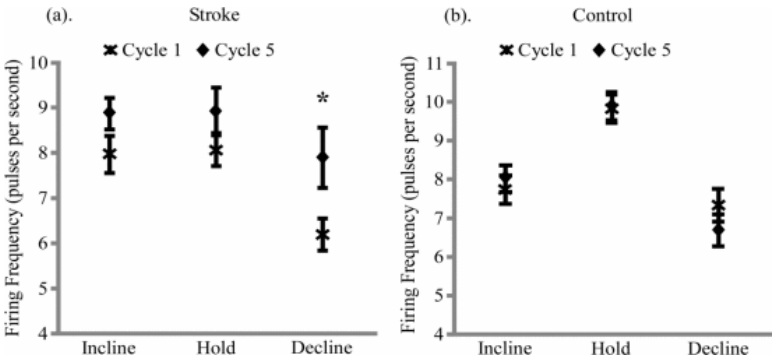

Figure 1. A comparison of the firing frequency rates for subsequent contractions for stroke and control subjects. (a) Paretic motor unit firing frequency increased during the torque decline phase with subsequent contractions (6.19±0.35 pps VS 7.89±0.66 pps, $P<0.5$);. (b) Control motor unit firing frequency rates were not significantly different during the torque decline phase with subsequent contractions (6.95±0.40 pps VS 6.68±0.41 pps)

Table 1. Average number of processed motor units

Number of Motor Units Processed	
Controls	***Stroke***
10.75 ± 3.61	8.20 ± 1.77

The average decrease in percent task torque per second for the decline phase was significantly greater in cycle 1 compared to cycle 5 of the ramp and hold for stroke subjects ($P < 0.05$). Stroke subjects decreased task torque by 13.51±1.66 %/s in cycle 1 compared to 12.34±0.94 %/s in cycle 5. Control subjects were not significantly different in decreasing task torque between cycles 1 and 5 of the

decline phase for the ramp and hold. On average, controls decreased task torque by 12.05±1.16 %/s for cycle 1 compared to 12.66±0.74 %/S for cycle 5 of the decline phase. Figure 2 shows the percent decline in task torque per second for the decline phase of the ramp and hold cycles 1 and 5 for both stroke and control.

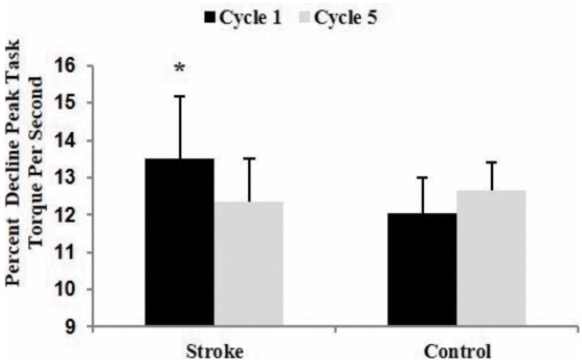

Figure 2. The average decrease in percent task torque per second during the decline phase in subsequent contractions for stroke and control subjects. Stroke subjects significantly decreased task torque in cycle 1 compared to cycle 5 (13.51±1.66%/svs12.34±0.94%/s,P<0.05). Control subjects decreased task torque with no significant difference between cycle 1 and cycle 5 12.05±1.16 %/s VS 12.66±0.74. %/s).

SECTION IV. Discussion

The novel findings of this study are that during repeated sub-maximal knee extensor contractions (1) paretic motorunits exhibit increased firing rates during volitional declines in torque as compared to control motor units and (2) increasing impairment in the ability to decrease knee extension torque as compared to control legs.

Our findings in the knee extensors are consistent with previous studies showing motoneuron hyperexcitability in the arm and hand musculature during tasks involving decreased volitional drive. As discussed in these papers, a plausible mechanism for paretic motoneuron hyperexcitability is that following a stroke there is decreased activity in corticomotor/corticobulbar descending pathways and increased activity in descending monoaminergic pathways originating from the brainstem[14], [15]. The monoamines are known to have a strong neuromodulatory effect on motoneurons - amplifying synaptic inputs to motoneuron and causing self-sustained firing[25]. We did not see effects of repeated contractions on the paretic motor unit firing behavior during the Incline or Hold phases. During both of these phases, descending drive is either increasing or being held steady and our findings may reflect a saturation in paretic motor unit firing rates seen by others[23],[24][25][26].

From a clinical standpoint, these data suggest that prolonged/inappropriate muscle activation may be worse when the individual is engaging in activities that require repeated contraction/relaxation of the musculature. Future studies will address whether this abnormal firing response can be modulated in order to improve force regulation post stroke.

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