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Delayed Muscle Activity in Stroke Survivors with Upper-Limb Hemiparesis

Danielle R. Lopez, *Student Member, IEEE,* Caleb J. Thomson, *Student Member, IEEE,* Fredi R. Mino, Steven R. Edgely, Patrick P. Maitre, Marta M. Iversen, and Jacob A. George, *Member, IEEE*

***Abstract*— Stroke is the leading cause of disability worldwide, and nearly 80% of stroke survivors suffer from upper-limb hemiparesis. Myoelectric exoskeletons can restore dexterity and independence to stroke survivors with upper-limb hemiparesis. However, the ability of patients to dexterously control myoelectric exoskeletons is limited by an incomplete understanding of the electromyographic (EMG) hallmarks of hemiparesis, such as muscle weakness and spasticity. Here we show that stroke survivors with upper-limb hemiparesis suffer from delayed voluntary muscle contraction and delayed muscle relaxation. We quantified the time constants of EMG activity associated with initiating and terminating voluntary hand grasps and extensions for both the paretic and non-paretic hands of stroke survivors. We found that the initiation and termination time constants were greater on the paretic side for both hand grasps and hand extensions. Notably, the initiation time constant during hand extension was approximately three times longer for the paretic hand than for the contralateral non-paretic hand (0.618 vs 0.189 s). We also show a positive correlation between the initiation and termination time constants and clinical scores on the Modified Ashworth Scale. The difficulty stroke survivors have in efficiently modulating their EMG presents a challenge for appropriate control of assistive myoelectric devices, such as exoskeletons. This work constitutes an important step towards understanding EMG differences after stroke and how to accommodate these EMG differences in assistive myoelectric devices. Real-time quantitative biofeedback of EMG time constants may also have broad implications for guiding rehabilitation and monitoring patient recovery.**

***Clinical Relevance*— After a stroke, muscle activity changes, and these changes make it difficult to use muscle activity to drive assistive and rehabilitative technologies. We identified slower muscle contraction and muscle relaxation as a key difference in muscle activity after a stroke. This quantifiable difference in muscle activity can be used to develop better assistive technologies, guide rehabilitation, and monitor patient recovery.**

I. Introduction

Over 795,000 people in the United States suffer from a stroke each year, and upper-limb hemiparesis comprises 80% of motor deficits in stroke survivors [1]. Hemiparesis presents as one-sided weakness or paralysis and is caused by damage to the central nervous system. This damage to the central nervous system interrupts descending motor control, dissociates motor responses and sensory inputs, and leads to hyperexcitability [2]. Hyperexcitability of motor fibers can lead to consistently contracted muscle and increased muscle tone [2]. This can range from slight increase in muscle tone, to complete rigidity that severely limits functional use of the upper limb. Complete recovery for stroke patients with hemiparesis is unlikely given current rehabilitation approaches and our limited understanding of how to promote neuroplasticity to repair impaired motor pathways following a stroke [3].

Powered exoskeletons can assist stroke survivors with upper-limb activities by physically supporting and moving the limb [4]. Myoelectric exoskeletons, such as the MyoPro® (Fig. 1), are intuitively controlled by electromyographic (EMG) signals recorded from the arm muscles as the user attempts to move their arm [5]. Although intuitive, the control afforded by EMG signals is limited to binary actions of just a few motorized joints [6]– far from the capabilities of the natural human hand. It is particularly difficult to use EMG signals for more dexterous control of exoskeletons due to substantial changes in muscle activity that occur after a stroke [4], [7]. A clearer understanding of the EMG hallmarks of a stroke is necessary before more dexterous EMG control can be developed for assistive myoelectric devices like the MyoPro.

The objective of this study was to quantify EMG changes in stroke survivors with upper-limb hemiparesis. Specifically, we looked at EMG signals in the paretic and non-paretic arms of stroke patients as they performed brief voluntary muscle contractions, as would typically be used to control an assistive myoelectric device. We found that stroke survivors took longer to initiate voluntary contractions and took longer to relax their muscles following a voluntary contraction. We quantified the time constants of muscle contraction and relaxation, and showed a positive correlation with the Modified Ashworth Scale (MAS) score—a clinically used scale that technically measures resistance to passive stretch though it is often referred to as a spasticity assessment. These results constitute an important step towards a more holistic understanding of the EMG hallmarks of a stroke, and can be used to develop new EMG control strategies, guide rehabilitation, or monitor patient recovery.

D. R. Lopez is with the Interdepartmental Neuroscience Program at the University of Utah, Salt Lake City, UT 84132 USA (e-mail: Danielle.Lopez@utah.edu).

C.J. Thomson is with the Department of Biomedical Engineering at the University of Utah.

F. R. Mino is with the Department of Electrical and Computer Engineering at the University of Utah.

M. M. Iversen is with the Department of Physical Medicine and Rehabilitation at the University of Utah.

J. A. George is with the Departments of Electrical and Computer Engineering and Physical Medicine and Rehabilitation at the University of Utah (e-mail: Jacob.George@utah.edu).

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Figure 1. MyoPro® exoskeleton. The MyoPro is a powered upper-limb orthosis that is controlled using EMG from the paretic arm. Stoke survivors use the MyoPro to assist them with activities of daily living.

II. Methods

A. Participants

We recorded EMG from seven stroke participants (four females, three males) that had upper-limb spastic hemiparesis, ages 22 – 57. Informed consent and experimental protocols were carried out in accordance with the University of Utah Institutional Review Board (Protocol No. 00098851 "Improving myoelectric prosthetic and orthotic limb control using predictive regression algorithms and high-count surface electrodes"). For each participant, we recorded their score on the Modified Ashworth Scale (MAS) for their wrists and fingers of their paretic side, as described in [8]. The MAS is a 6-point scale of resistance to passive stretch. A score of 0 indicates no increase in muscle tone; a score of 1 indicates slight increase in muscle tone, manifested by catch and release; a score of 1+ indicates slight increase in muscle tone, manifested by catch and followed by minimal resistance; a score of 2 indicates marked increase in muscle tone; a score of 3 indicates considerable increase in muscle tone; and a score of 4 indicates complete rigidity in flexion or extension.

B. Signal Acquisition

We recorded EMG on the forearm using a custom sleeve with 32 surface EMG (sEMG) electrodes [9]. We sampled EMG data at 1 kHz using the Summit Neural Interface processor (Ripple Neuro Med LLC) [10].EMG data was bandpass filtered between 15-375 Hz. The mean absolute value (MAV) of the EMG was calculated using a 300-ms window updated at 30 Hz, as described in [10].The EMG MAV was calculated for all electrodes. sEMG electrodes that were not conductively active (e.g., due to broken wires or poor skin contact), were removed prior to data analysis.

C. Experimental Task

We tasked participants to perform a series of 20 instantaneous maximum voluntary contractions (10 grasping, 10 extending) with their non-paretic hand, with at least 3 seconds between contractions to avoid muscle fatigue. We then had the participant repeat the task with the paretic hand. For each condition, non-paretic and paretic, we averaged the EMG MAV over the 10 trials for each channel, and then used the average EMG waveform to calculate the time constants from each channel (Fig. 2).

D. EMG-Based Performance Metrics

We calculated one initiation time constant and one termination time constant per channel (each channel averaged over 10 trials). Then, we took the median time constant (τ) of all channels for each participant in each category: initiation and termination of hand grasp and extension.

To calculate the time constants, τ, we first defined a

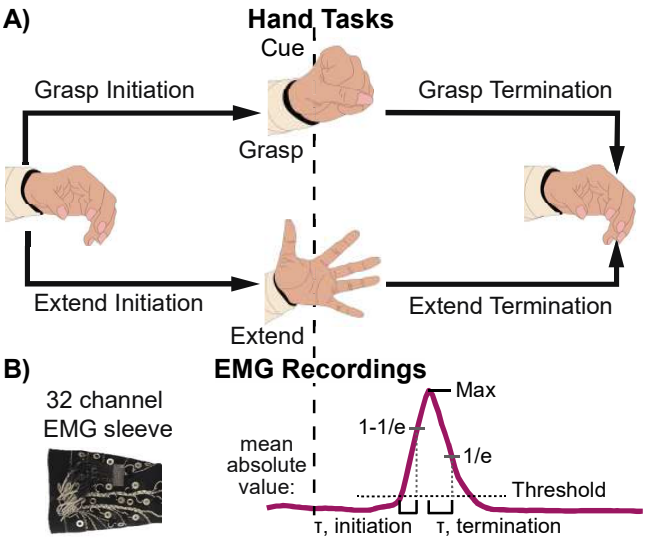

Figure 2. Experimental task. A) Stroke survivors performed 20 instantaneous maximum voluntary contractions (10 grasping, 10 extending). B) EMG was recorded from their extrinsic hand muscles using a 32-electrode sleeve placed on their forearm. An example trace of hand extension shows the mean EMG MAV for one electrode across the 10 trials and the method used to calculate the initiation and termination time constants, τ.

baseline threshold of ten percent of the EMG MAV range within the event window. We defined the start of EMG activity and end of EMG activity as the time at which the EMG MAV crossed the threshold. The initiation time constant was calculated as the duration from start of EMG activity to when the EMG MAV reached $(1-1/e)*Maximum$. The termination time constant was calculated as the duration from the maximum EMG MAV to when the EMG MAV fell to $(1/e)*Maximum$.

E. Statistical Analyses

All data were screened for normality using the Anderson-Darling test. For each participant (n = 7), we calculated a single median τ for the paretic and non-paretic hand for each of the four conditions: 1) initiation τ for grasp, 2) termination τ for grasp, 3) initiation τ for extension, 4) termination τ for extension. Multiple comparisons were not performed between these four conditions. Only one comparison (paretic vs non-paretic) was determined to be relevant *a priori.* Because the data from the seven participants were normally distributed, we used paired t-tests to compare the difference between the paretic and non-paretic hands. To evaluate the correlation between the time constants and participant MAS score, we calculated the Pearson's correlation coefficient (r).

III. Results

A. Time Constants are Longer on the Paretic Side

We compared the initiation and termination time constants of hand grasps and extensions between the paretic and non-paretic side of stroke survivors (Fig. 3). The paretic side had significantly longer initiation time constants for hand extensions: 0.618 ± 0.144 s vs. 0.189 ± 0.018 s (p = 0.029, paired t-test). The paretic side also trended towards having longer initiation time constants for hand grasps: 0.409 ± 0.082 s vs. 0.210 ± 0.023 s (p = 0.054, paired t-test). Termination time constants were also significantly longer on the paretic side for both extensions (0.561 ± 0.029 s vs. 0.345 ± 0.044 s;

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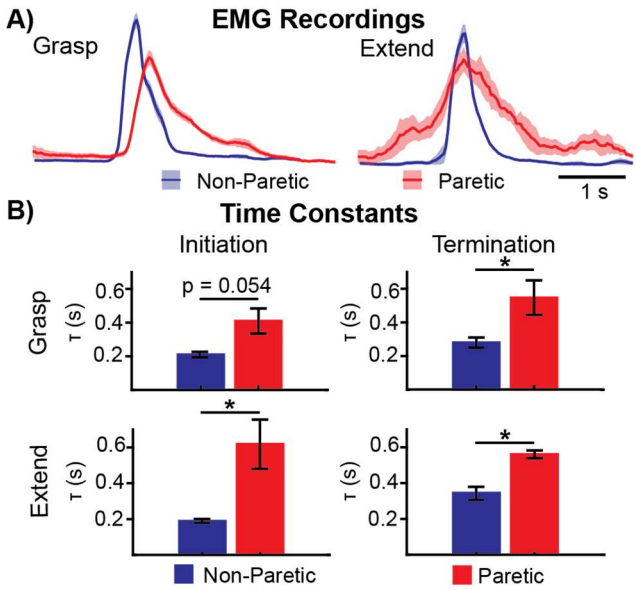

Figure 3. Delayed muscle activity on the paretic side of stroke survivors. A) EMG traces (mean ± standard error of the mean) from the paretic (red) and non-paretic (blue) sides of one participant during a hand grasp or extension. B) Initiation and termination time constants for hand grasping and extension for the paretic and non-paretic sides. Bars shown mean ± standard error of the mean. Asterisk (*) indicates $p < 0.05$ for a paired t-test. N = 7 participants.

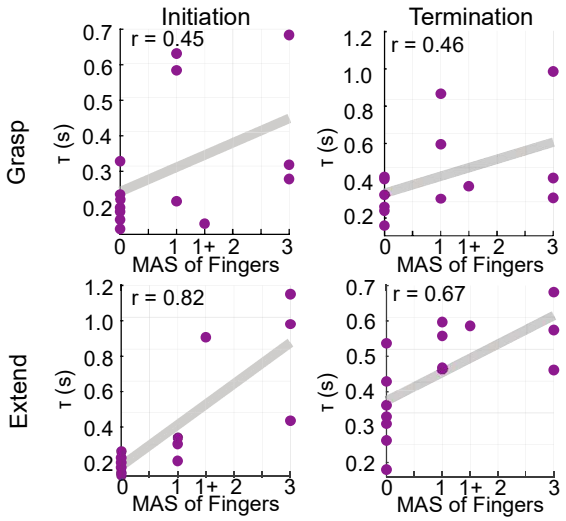

Figure 4. Correlation of time constants of muscle activity with Modified Ashworth Scale (MAS). Initiation and termination time constants of grasping had moderate correlations with MAS (r = 0.45 and r = 0.46, respectively). Initiation time constant of extension had a high positive correlation with MAS (r = 0. 82). Termination time constant of extension had a moderate positive correlation with MAS (r = 0.67).

$p = 0.012$, paired t-test) and grasps (0.547 ± 0.110 s vs 0.280 ± 0.038 s; $p = 0.046$, paired t-test).

B. Initiation Time Constant of Extension Most Strongly Correlates to Modified Ashworth Scale of Spasticity

We next sought to quantify if the time constants were correlated with the severity of resistance to passive stretch/spasticity via the Modified Ashworth Scale (MAS) (Fig. 4). We found a high positive correlation [11] with the initiation time constant of extension (r = 0.8227), and a moderate positive correlation [11] with the termination time constant of extension (r = 0.6667). Both the initiation and termination time constants for grasping showed only low correlations with MAS scores [11] (r = 0.45 and r = 0.46 respectively).

IV. Discussion

After a stroke, individuals often have abnormal patterns of muscle activation [12], [13]. This presents a problem when trying to control assistive devices, such as powered exoskeletons, with muscle activation. Here, we highlight and quantify two key differences in muscle activation after a stroke – the time to initiate and the time to terminate a contraction. We show longer initiation time constants and termination time constants in hand grasps and hand extensions on the paretic side relative to the non-paretic side. We also show correlations between the time constants and severity of hemiparesis, albeit with a relatively small sample size of seven participants.

Prior work has shown temporal delays in initiating motor activity on the paretic side for the arm [14], wrist [15], hand [16], and lower limb [17]. Delays in terminating muscle activity on the paretic side have also been shown for the wrist [15], hand [16], and lower limb [17]. We build upon these studies by quantifying delays in muscle activity during the specific muscle activation patterns used to control assistive myoelectric devices. That is, we looked at brief muscle activity (~1 second) instead of extended muscle activity (~3 second) and added a novel analysis of hand extension. Furthermore, we introduced a new methodology to quantify delays in muscle activity that can be used on any arbitrary muscle activation without the need for explicit starting and stopping cues. Importantly, this methodology is also independent of an individual's reaction time, which has been shown to be delayed in stroke survivors [14]. The use of relative EMG thresholding to determine time constants of muscle activity alone could be implemented into wearable devices and myoelectric exoskeletons to guide rehabilitation or monitor patient recovery.

Also novel in this work, we demonstrate that the delays in muscle activity observed after a stroke are correlated with the severity of resistance to passive stretch in spastic hemiparesis, as assessed by the Modified Ashworth Scale (MAS). Spastic hemiparesis in the hand after a stroke most commonly presents as a "clenched fist" resting posture that is attributed to 1) weak extensors from diminished voluntary activation secondary to corticospinal tract damage, and 2) co-activation of spastic flexors secondary to hyperexcitable reticulospinal tract activation after loss of inhibition [18]. Given that spasticity is associated with weak and inhibited extensor muscles, it is reasonable that the initiation time constant for hand extension had the strongest correlation with MAS. Future work is needed to validate the correlations between EMG time constants and MAS with a larger sample size.

The non-paretic side of the patients were all rated as an MAS score of 0 indicating no increase in muscle tone due to passive stretch. Initiation time constants on the non-paretic side were tightly clustered among patients for both hand grasps and extensions. In contrast, we observed considerable variability in the termination time constants on the non-paretic side. This difference in variability between the initiation and termination time constants is consistent with prior work [15] and may be attributed to different neurological bases for

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delayed motor initiation and termination. Delayed motor initiation may be due to increased inhibition from the non-lesioned to lesioned hemisphere [19], fewer functioning motor units, and slower motor unit firing, all of which predominately effect the paretic side [20]–[22]. Delayed motor termination may be due to spastic motor neurons [23], [24], decreased inhibition from the lesioned to non-lesioned hemisphere [25] and increased excitatory activity with reduced inhibitory activity in the non-lesioned motor cortex [26], [27]; the latter two of which would lead to greater variability in termination time constants on the non-paretic side.

V. Conclusion

This work explored the EMG hallmarks of hemiparesis associated with muscle activations used to control assistive myoelectric devices. Consistent with prior work, we showed delayed muscle initiation and delayed muscle termination on the paretic side. We also introduced a novel approach to analyzing temporal activity of arbitrary muscle activations without the need for external start and stop cues. Lastly, we showed that delays in muscle activity are correlated with the severity of hemiparesis, as assessed by the MAS for spasticity. Together, this work constitutes an important step towards a better understanding of the EMG hallmarks of hemiparesis. This work can serve as a basis for future studies incorporating EMG time constants into myoelectric devices for real-time biofeedback to enhance control, guide rehabilitation, and monitor patient recovery.

VI. Autor Contributions

DRL derived metrics, analyzed data, wrote the manuscript, and generated figures. CJT collected participant data. FRM designed the graphical-user interface that enabled metric calculation. MMI helped write the manuscript. SRE and PPM helped with patient recruitment. JAG supervised and provided guidance for the study and edited the manuscript.

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