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EMG synchrony to assess impaired corticomotor control of locomotion after stroke

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Abstract

Adapting one's gait pattern requires a contribution from cortical motor commands. Evidence suggests that frequency-based analysis of electromyography (EMG) can be used to detect this cortical contribution. Specifically, increased EMG synchrony between synergistic muscles in the Piper frequency band has been linked to heightened corticomotor contribution to EMG. Stroke-related damage to cerebral motor pathways would be expected to diminish EMG Piper synchrony. The objective of this study is therefore to test the hypothesis that EMG Piper synchrony is diminished in the paretic leg relative to nonparetic and control legs, particularly during a long-step task of walking adaptability. Twenty adults with post-stroke hemiparesis and seventeen healthy controls participated in this study. EMG Piper synchrony increased more for the control legs compare to the paretic legs when taking a non-paretic long step ($5.02 \pm 3.22\%$ versus $0.86 \pm 2.62\%$), $p < 0.01$) and when taking a paretic long step ($2.04 \pm 1.98\%$ versus $0.70 \pm 2.34\%$, $p < 0.05$). A similar but non-significant trend was evident when comparing non-paretic and paretic

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The authors have no conflicts of interest to report

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legs. No statistically significant differences in EMG Piper synchrony were found between legs for typical walking. EMG Piper synchrony was positively associated with walking speed and step length within the stroke group. These findings support the assertion that EMG Piper synchrony indicates corticomotor contribution to walking.

Keywords

electromyography; walking; motor control; nervous system

Introduction

Safe and successful ambulation in the home and community requires the ability to adapt one's gait pattern to meet task objectives and environmental demands [Balasubramanian et al, 2014]. Descending motor control from the cerebral cortex is crucial for integrating volitional goal-directed motor commands with the ongoing locomotor pattern generated from brainstem and spinal circuits of locomotor control [Amos et al, 1990, Beloozerova et al, 2010, Drew, 1988, Schubert et al, 1999]. The integrity of descending motor pathways including corticospinal and corticoreticular tracts are often compromised after stroke, leading to motor impairments [Kunimatsu et al, 2003, Lie et al, 2004]. There is an important need to develop physiological assessments that can be used to gauge descending corticomotor function within the context of walking. Such assessments will facilitate measurement of impairment and recovery of locomotor control after stroke.

Compelling evidence supports the use of frequency-based assessments of electromyography (EMG) to gauge the functional integrity of corticomotor tracts during motor tasks including walking [Clark et al, 2013, de Miranda Marzullo et al, 2010, Hansen et al, 2005, Nielsen et al, 2008, Norton et al, 2006, Petersen et al, 2010, Petersen et al, 2012]. Specifically, synchronous EMG activity between synergistic muscles in the Piper frequency range (30–60 Hz) may convey the cortical contribution to muscle contraction, particularly during dynamic tasks [Brown, 2000, Brown et al, 1998, Omlor et al, 2007]. EMG synchrony is thought to be driven by rhythmic activity in the motor cortex [Baker et al, 1997, Grosse et al, 2002, Kilner et al, 1999, Neto et al, 2010]. Piper band activity in the cortex has been attributed to interactions among cortical regions that are involved in movement execution under variable conditions that require attentional resources, such as to integrate visual and somatosensory information [Brown, 2000, Omlor, Patino, 2007]. This is generally consistent with the prior finding that walking adaptability tasks invoke increased cortical activity in regions important to attention and motor planning [Clark, 2015, Clark et al, 2014]. Furthermore, prior work has shown that EMG synchrony in the Piper range exhibits task-dependent changes that are consistent with the attentional and physical demands of various walking adaptability tasks [Clark, Kautz, 2013]. Importantly, EMG-based assessment provides insight into corticomotor activity that is actually reaching the muscle. This is in contrast to brain neuroimaging assessments, which can detect brain activity but cannot alone determine if that activity manifests as a command at the neuromuscular level.

In people who have experienced a stroke, damage to descending corticomotor pathways contributes to hemiparesis. Accordingly, people with post-stroke hemiparesis could be expected to have diminished EMG Piper synchrony during motor tasks including walking. The objective of this study is therefore test the hypothesis that EMG Piper synchrony will be diminished in the paretic leg of individuals with post-stroke hemiparesis relative to nonparetic and healthy control legs, particularly during a long-step task of walking adaptability. The long step task was chosen because it is a common task of walking adaptability that requires a distinct voluntary (i.e., cortical) motor contribution. A secondary objective is to explore associations between EMG Piper synchrony and walking performance in order to gauge the clinical significance of this measure.

Materials and Methods

Participants

Twenty adults with post-stroke hemiparesis and seventeen healthy adults participated in this study. Post-stroke participants were recruited using the following inclusion criteria: hemiparesis due to a single unilateral stroke; at least 6 months since the date of stroke; and ability to ambulate independently (with or without an assistive device) for 10m on a level surface. Healthy older adults were recruited using the following inclusion criteria: age between 65 and 85 years, preferred walking speed >1.0 m/s, Berg Balance Scale score >50 (out of 56). Exclusion criteria for all participants included the presence of significant non-motor impairments that could independently affect walking function including lower extremity joint pain, bone fracture within the past year, major sensory deficits, and cardiovascular or respiratory symptoms contraindicated to exercise. All participants provided written informed consent. All study procedures were approved by the University of Florida Institutional Review Board and by the Malcom Randall VA Medical Center Human Research Protection Program.

Procedures

Demographic and clinical assessments were conducted for all post-stroke participants, including gender, time since stroke, preferred overground walking speed (from 10 meter walk test), and Fugl-Meyer assessment.

All EMG-based assessments were conducted while participants walked on a treadmill (Bertec, Columbus OH). Disposable surface gel electrode with 10mm round Ag/AgCl recording sensor (Versa-Trode, Vermed, Bellow Falls VT) were placed on the skin over the triceps surae muscles: soleus and medial gastrocnemius. For each muscle, the inter-electrode distance was approximately 2 cm. These muscles were chosen because of the important role of the triceps surae for support and forward propulsion during walking and because EMG Piper synchrony is most readily detected between synergistic muscles. The electrode sites for soleus were distal to the belly of the gastrocnemius, medial and anterior to the Achilles tendon [Perroto, 1994]. The sites for medial gastrocnemius were one hand breadth below the popliteal crease on the medial mass of the calf [Perroto, 1994]. Each site was shaved and firmly rubbed with a sterile alcohol wipe prior to electrode placement. The electrodes were attached to an EMG preamplifier (Motion Lab Systems MA-420, Baton Rouge LA) and data

were sampled at 1000Hz using a commercially available EMG system (Motion Lab Systems MA300-28, Baton Rouge LA). Kinematic data were also acquired by attaching reflective markers to anatomical landmarks on the head, trunk, arms, legs and feet using the Helen Hayes configuration [Kadaba et al, 1990] modified with additional marker triads attached to rigid plates located on each foot, shank, and thigh segment [Allen et al, 2011]. The 3-dimensional location of each marker was sampled at 200 Hz using a 12 camera motion capture system and data acquisition system (VICON, Colorado, USA).

Data collection sessions began with a familiarization period in which the participants walked for 3–5 minutes on the treadmill. The speed was initially set very slowly and gradually increased until the participants reported that the speed was faster than their typical walking. The speed was then lowered to what the participants reported was their preferred typical walking speed. The experimental assessment included two distinct walking tasks performed during separate trials on the treadmill: 1) steady state walking, and 2) a walking adaptability task in which a longer step was taken on approximately every sixth gait cycle in response to a verbal cue. Both tasks were performed at each individual's own preferred typical walking speed. Control participants performed a right long step only, while post-stroke participants performed both a paretic long step task and non-paretic long step in separate walking trials. All participants were specifically told not to take the longest possible step, but rather to take a moderately longer step. Our objective was to have participants perform the step lengthening in a manner that would be typical of daily ambulatory activities. We chose not to impose a specific goal for step lengthening (such as targeting a fixed distance or percentage over normal step length) because pilot testing showed that it is challenging to define a value that is both attainable and comparable in difficulty across participants with differing levels of stroke severity. Furthermore, targeted stepping (e.g., to a specific line or point on the ground) is a less natural and more demanding motor control task.

Data were recorded for 60 seconds for each task. To ensure safety, all participants wore a harness secured to an overhead support. Participants did not hold onto a railing or use any other support devices or walking aids during the assessment.

Data Analysis

EMG signals were detrended then high-pass filtered at 5 Hz with a fourth-order, zero-lag, Butterworth filter. EMG synchrony was calculated using a frequency domain analysis involving *Morlet* wavelet transform:

$$\Psi_0(t) = \pi^{-\frac{1}{4}} e^{i\omega_0\eta} e^{-\frac{\eta^2}{2}}$$

Where η is dimensionless time and ω_0 is dimensionless frequency (in this study we used $\omega_0 = 6$, as suggested by Grinsted and colleagues [Grinsted et al, 2004]).

The Morlet wavelet (with $\omega_0 = 6$) is appropriate when performing wavelet and cross-wavelet analysis, because it provides a reasonable balance between time and frequency localization. The wavelet transform applies the wavelet function as a band pass filter to the time-series:

$$W(s, \tau)^X = \int X(t) \Psi_{s, \tau}^*(t) dt$$

Where s represents the dilation parameter (scale shifting), τ represents the location parameter (time shifting), and the basic function $\Psi_{s, \tau}^*$ is obtained by dilating and translating the mother wavelet $\Psi_0(t)$.^{0.125} Wavelet and cross-wavelet transforms were calculated using a base algorithm in Matlab (The Mathworks, Natick MA) developed by Torrence and Compo [Torrence et al, 1998] and available at URL: <http://paos.colorado.edu/research/wavelets>. From the wavelet transform of both EMG signals, the cross-wavelet transform was calculated:

$$W(s, \tau)^{XY} = W(s, \tau)^X W(s, \tau)^{Y*}$$

Where $W(s, \tau)^{XY}$ is the cross-wavelet transform of signals $X(t)$ and $Y(t)$, $W(s, \tau)^X$ is the wavelet transform of signal $X(t)$, and $W(s, \tau)^{Y*}$ is the complex conjugate of the wavelet transform of signal $Y(t)$.

Upon completing the cross-wavelet analysis, the distribution of cross wavelet power spectra across different frequency bands for each walking task was calculated. The cross-wavelet spectra were divided into the following six frequency bands: 5–13Hz, 13–30 Hz, 30–60 Hz, 60–100 Hz, 100–200 Hz, and 200–300 Hz. Normalized power was calculated by dividing the power within each band to the total power over the entire 5–300 Hz range. We report normalized power because it considers the relative strength of synchronous activity in this frequency band compared to all other frequency bands. In this manuscript, normalized cross wavelet power in the 30–60Hz band is referred to as EMG Piper synchrony. Representative data showing raw data and EMG Piper synchrony for one participant are shown in Figure 1.

Kinematic data were low pass filtered at 10 Hz using a fourth order Butterworth filter with zero phase lag. Step length was calculated in the sagittal plane as the maximal distance between the left and right heel markers during the double-limb stance phase of gait.

For each long step walking task, the gait cycles containing the long steps were separated from the rest of the walking trial after performing the cross-wavelet analysis. There were about 7–10 long step gait cycles per participant, and only data from those gait cycles were used for analysis. For the long step task, we use the term ‘contralateral leg’ to refer to the leg that served in the stance role (e.g., the paretic leg when taking a non-paretic long step). We use the term ‘ipsilateral leg’ to refer to the long stepping leg (e.g., paretic leg when taking a paretic long step).

Statistics

Due to the different neuromechanical roles of the contralateral and ipsilateral limbs during a long step, EMG from each limb was assessed using separate statistical models. Differences in EMG Piper synchrony were examined using a 3 Leg $\times$ 2 Task mixed-model repeated measures ANOVA. A post hoc analysis was conducted when significant main effects or interactions were identified. Furthermore, we examined the association between walking

performance and EMG Piper synchrony by conducting Pearson correlation tests. Statistical analyses were conducted with IBM SPSS Statistics 21.0.

Results

Participant characteristics are provided in Table 1. Group mean data for step length, ankle power, and RMS EMG amplitude for each task are provided in Table 2. On average, both groups/sides demonstrated similar changes in the percent increase in the step length during the long step task. Further, both groups exhibited similar strategies for taking a longer step as indicated by increased ankle power production and plantarflexor muscle activation.

EMG Piper synchrony was averaged over the full gait cycle, and also averaged within the late stance phase of gait where most triceps surae activity occurs. The data from each calculation were found to be nearly equivalent within each group and task, with correlation coefficients ranging from 0.90 to 0.95 ($p < 0.001$). Therefore, only the data averaged over the full gait cycle was used for subsequent analysis.

Figure 2 shows the relative power distribution of EMG synchrony across each frequency band. The 30–60Hz band exhibited the largest and most consistent increase in synchrony during the long step task in both groups. Figure 3 presents a more focused view of EMG synchrony in the 30–60Hz band for each task and leg. For the contralateral leg, EMG Piper synchrony exhibited a significant main effect of Task, [$F(1,54) = 69.93$; $p < 0.001$; $\eta^2 = 0.56$], with long step greater than steady state walking ($p < 0.05$). A significant Leg $\times$ Task interaction was also found [$F(2,54) = 11.93$; $p < 0.001$; $\eta^2 = 0.31$; Fig 1A]. Post hoc analysis of the interaction effect revealed a greater increase in EMG Piper synchrony in the control legs ($5.02 \pm 3.22\%$) versus the paretic legs ($0.86 \pm 2.62\%$) for the long step task ($p < 0.01$).

For the ipsilateral leg, EMG Piper synchrony exhibited a significant main effect of Task, [$F(1,54) = 32.52$; $p < 0.001$; $\eta^2 = 0.38$], with long step greater than steady state walking ($p < 0.05$). A significant Leg $\times$ Task interaction was also found [$F(2,54) = 3.74$; $p < 0.05$; $\eta^2 = 0.12$; Fig 1B]. Post hoc analysis of the interaction effect revealed a greater increase in EMG Piper synchrony in the control legs ($2.04 \pm 1.98\%$) versus the paretic legs ($0.70 \pm 2.34\%$) during the long step task ($p < 0.05$). A similar trend was evident for EMG Piper synchrony in the non-paretic leg (2.26 ± 2.00) compared to the paretic leg during the long step task ($p = 0.06$). No statistically significant differences were found between control, paretic or nonparetic legs during typical walking ($p > 0.05$).

To assess the relationship between functional integrity of the corticomotor tracts and walking performance, the percent change in EMG Piper synchrony between the self-selected and long step condition was calculated for the paretic triceps surae, because performance by the paretic leg generally dictates functional outcomes. For this analysis, the non-paretic long step task was used because this task demands increased biomechanical and corticomotor output from the paretic plantarflexors [Clark et al, 2015]. This measure of corticomotor functional capacity was strongly positively associated with preferred overground walking speed ($R^2 = 0.48$, $p = 0.006$), preferred nonparetic step length during steady state walking

($R^2=0.41$, $p=0.019$) and maximal nonparetic step length during the nonparetic long step task ($R^2=0.43$, $p=0.015$).

Discussion

This study assessed whether EMG Piper synchrony in the triceps surae muscle group is sensitive to differences in the corticomotor contribution to different walking tasks for adults post-stroke and healthy controls. To our knowledge, this is the first study to use an EMG synchrony analysis to examine impaired descending voluntary control of leg muscle activity during walking adaptability post-stroke. The assessment of adults post-stroke is a crucial aspect of this study, because differences in EMG Piper synchrony between impaired and healthy legs can be attributed in part to the impaired descending corticomotor command that is characteristic of post-stroke hemiparesis. We examined this in the context of a long-step walking task, which is an acute gait adaptation involving a voluntary command transmitted from the motor cortex through descending motor pathways [Amos, Armstrong, 1990, Beloozerova, Farrell, 2010, Drew, 1988]. A common strategy for step lengthening is to increase plantarflexion force from the triceps surae during the stance phase of the leg contralateral to the long stepping leg [Clark, Neptune, 2015, Varraine et al, 2000]. To a lesser extent, triceps surae activity is also increased for the ipsilateral long stepping leg during the stance phase immediately preceding the long step [Clark, Neptune, 2015, Varraine, Bonnard, 2000].

Consistent with the present findings, prior work in this area indicates that EMG Piper synchrony is increased during a long step adaptability task in healthy adults, particularly for the contralateral limb [Clark, Kautz, 2013]. The present results show that this task-appropriate increase in EMG Piper synchrony is maintained in the non-paretic leg of stroke participants, but markedly diminished in the paretic leg. These results are in agreement with the study hypothesis, and are consistent with diminished corticomotor drive from the injured hemisphere after stroke. The assertion that EMG Piper synchrony can reveal cortical drive to muscle is supported by a considerable body of prior evidence. Studies that have examined synchrony between electroencephalography (EEG) and EMG have revealed that submaximal voluntary isometric contractions exhibit synchrony at 15–30 Hz in a somatotically organized manner [Brown, Salenius, 1998, Conway et al, 1995, Halliday et al, 1998, Kilner et al, 2000, Salenius et al, 1997]. During dynamic muscle contractions, the 15–30 Hz EEG-EMG synchrony is diminished [Kilner, Baker, 1999, Omlor, Patino, 2007], and stronger synchrony becomes evident in the 30–60 Hz Piper range [Brown, 2000, Brown, Salenius, 1998, Omlor, Patino, 2007]. The amount of synchronous activity is considered to be associated with the integrity of the corticomotor output [Mima et al, 2001]. The same drive that elicits EEG-EMG synchrony can also lead to task-dependent synchronous activation among paired EMG recordings, such as synergistic muscles (as in the present study) or between different regions of a single muscle [Baker, Olivier, 1997, Grosse, Cassidy, 2002, Kilner, Baker, 1999, Neto, Baweja, 2010]. A number of prior studies have shown that central nervous system disease and functional deficits are linked to reduced EMG synchrony in gamma/Piper bands [Petersen et al, 2013, Willerslev-Olsen et al, 2015]. However, in the present study there was no significant group difference in EMG Piper synchrony during typical walking. This lack of difference despite corticomotor impairment after stroke may be

due to variability in the relative distribution of synchronous activity across the range of frequencies due to inter-individual differences and EMG signal conditions [De Luca, 1997]. In contrast, assessing task-dependent (i.e., within-subject) changes between the typical walking and long step tasks using repeated measures analysis helps to control for inter-individual variability and yielded stronger findings as discussed above. Accordingly, assessment of walking adaptability tasks can be useful for providing additional insight into the neural control of walking.

A second objective of this study was to determine whether the functional corticomotor output, as quantified by the percent change in EMG Piper synchrony between steady state and long step tasks, is associated with walking performance after stroke. Functional corticomotor output was found to be strongly positively associated with preferred overground walking speed, nonparetic step length during steady state walking, and maximal nonparetic step length during the nonparetic long step task. We focused on nonparetic step length as a performance outcome because it is highly dependent on the neuromuscular output and propulsion from paretic plantarflexors (i.e., from the contralateral stance leg) during stance. Accordingly, deficient EMG Piper synchrony on the paretic side is expected to be linked to impaired propulsion and shorter nonparetic step lengths. This evidence of an association between EMG Piper synchrony and gait performance supports the assertion that it may be a biomarker of functional corticomotor integrity.

The findings of this study support further investigation of EMG Piper synchrony as an indicator of corticomotor impairment and its effect on recovery of walking adaptability after stroke. This approach offers a relatively feasible and low cost approach for gauging the neural control of walking, and may be appropriate for use as an outcome measure in large clinical trials. For instance, this approach could be valuable for comparing the efficacy of different rehabilitation interventions. Likewise, this approach may help to identify patients who would be good candidates for post-stroke rehabilitative interventions that target corticomotor neuroplasticity.

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Biographies

Neha Lodha completed her undergraduate degree in engineering from India and a PhD degree in Kinesiology from the University of Florida. Her Ph.D. work was focused on bimanual force control in stroke. She completed postdoctoral training in Neurophysiology and stroke rehabilitation at the Malcom Randall VA Medical Center. Dr. Lodha is now an Assistant Professor at Colorado State University. The goal of her research is to develop rehabilitation interventions to improve driving, walking, and function of both hands in people who have had transient ischemic attack or stroke.

Yen-Ting Chen received his Bachelor's and Master's degree in physical therapy from the National Cheng Kung University in Taiwan. He then received his PhD degree in Biobehavioral Science from the University of Florida. He is a postdoctoral fellow at the University of Texas Health Science Center-Houston in the Department of Physical Medicine and Rehabilitation. His research interest is the motor control and learning and the neuromuscular mechanism behind it.

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Figure 1.

Representative data from a participant with post-stroke hemiparesis. The left panel shows data from the non-paretic leg during a paretic long step (PLS) task and the right panel shows data from the paretic leg during a non-paretic long step (NLS) task. In each case the leg shown is serving in the stance/contralateral role of the long step. The region of data highlighted in black shows the gait cycle in which the long step occurred. The top row shows anterior (propulsion) and posterior (braking) ground reaction forces. The second and third rows show unprocessed electromyography (EMG) from the soleus and medial gastrocnemius muscles, respectively. The fourth row shows the cross-wavelet power that is present within the 30–60Hz frequency band (EMG Piper synchrony), expressed as a percentage of total cross-wavelet power in all frequencies. There is a visible increase in Piper synchrony for the non-paretic leg during the PLS. This increase is not evident for the paretic leg during the NLS, suggesting impaired corticomotor drive.

Figure 2.

The relative power distribution of EMG synchrony across all frequency bands. For each leg and each frequency band, EMG synchrony is shown for steady state walking (black bars), ipsilateral/swing leg during a long step (gray bars), and contralateral/stance leg for a long step (white bars). An asterisk indicates that the value for the long step is difference from steady state walking at $p < 0.05$.

Figure 3.

EMG synchrony in the Piper frequency band (30–60Hz) measured by cross wavelet spectral analysis and quantified as percent of total cross wavelet power. Data are shown for typical steady state walking and long step adaptability tasks, with the contralateral leg in the left panel and the ipsilateral leg in the right panel (see Methods for further explanation). Data from paretic and nonparetic limbs of participants post-stroke are shown as black triangles and open circles, respectively. Data from control legs are shown as black circles. Error bars indicate standard error of the mean. Asterisks indicate a statistically significant interaction effect between Limb and Task, with the increase for the control leg larger than the increase for the paretic leg ($p < 0.05$).

Table 1**Participant Characteristics**

	Stroke Group	Control Group
Age (years)	62.7 ± 9.4	70.1 ± 3.9
Sex (female/male)	9F / 15M	8F / 9M
Preferred Walking Speed (m/s)	0.66 ± 0.29	1.31 ± 0.15
FMA score (out of 34)	22.8 ± 5.3	NA
Time since stroke (months)	47.1 ± 37.0	NA

Abbreviations: FMA - lower extremity Fugl-Meyer Assessment; NA - not applicable

Table 2

Kinematic, Kinetic, and EMG Magnitude Data

	Steady State	Paretic Long Step	Nonparetic Long Step (or Left long step for Controls)
Step Length: Paretic (cm)	35.0 ± 11.6	62.7 ± 14.2 (+79%, p<0.001)	25.6 ± 16.5 (-27%, p=0.008)
Step Length: Nonparetic (cm)	30.6 ± 15.1	23.0 ± 15.2 (-25%, p=0.01)	61.4 ± 17.7 (+100%, p<0.001)
Step Length: Control Right (cm)	49.6 ± 6.7	na	35.2 ± 11.7 (-29% (p=0.006)
Step Length: Control Left (cm)	49.0 ± 6.1	na	91.2 ± 16.3 (+86% (p<0.001)
Ankle Power: Paretic ([N*ml/s)	0.61 ± 0.52	0.77 ± 0.87 (+26, p=0.25)	1.02 ± 0.64 (+70%, p<0.001)
Ankle Power: Nonparetic ([N*ml/s)	1.11 ± 0.61	1.84 ± 0.85 (+66%, p<0.001)	1.52 ± 0.85 (+37%, p=0.001)
Ankle Power: Control Right ([N*ml/s)	1.66 ± 0.43	na	2.68 ± 0.87 (+61%, p<0.001)
Ankle Power: Control Left ([N*ml/s)	1.64 ± 0.40	na	1.80 ± 0.72 (+10%, p=0.31)
SO EMG: Paretic (RMS µV)	63.8 ± 41.3	69.8 ± 47.1 (+9%, p=0.09)	94.0 ± 84.2 (+47%, p=0.02)
SO EMG: Nonparetic (RMS µV)	94.8 ± 84.6	117.1 ± 103.5 (+23%, p=0.005)	87.2 ± 95.2 (+8%, p=0.31)
SO EMG: Control Right (RMS µV)	138.6 ± 61.3	na	230.1 ± 116.0 (+66%, p<0.001)
SO EMG: Control Left (RMS µV)	138 ± 76.8	na	149.7 ± 74.6 (+9%, p=0.19)
MG EMG: Paretic (RMS µV)	50.0 ± 42.8	58.9 ± 51.0 (+18%, p=0.04)	63.4 ± 50.3 (+27%, p=0.01)
MG EMG: Nonparetic (RMS µV)	109.0 ± 73.8	97.2 ± 55.0 (-11%, p=0.27)	105.3 ± 72.1 (-3%, p=0.51)
MG EMG: Control Right (RMS µV)	151.6 ± 80.0	na	201.7 ± (+33%, p=0.02)
MG EMG: Control Left (RMS µV)	161.1 ± 87.9	na	181.6 ± 132.5 (+13%, p=0.02)