Surface EMG amplitude does not identify differences in neural drive to synergistic muscles

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Running Head:

Motor unit size and EMG of synergistic muscles

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Key words

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ABSTRACT

Surface electromyographic (EMG) signal amplitude is typically used to compare the neural drive to muscles. We directly compared the neural drive sent to the vastus medialis (VM) and vastus lateralis (VL) during knee extension by identifying motor units (MU) in the two muscles with the same torque recruitment threshold. Eighteen participants performed isometric knee extensions at 10, 30, 50 and 70% of maximum torque (MVC) while high-density EMG signals were recorded. MU discharge rate, conduction velocity (MUCV), and amplitude [root mean square (MURMS)] of the MU action potentials (MUAPs) were compared between muscles after matching recruitment thresholds. The linear regression slope of the difference between mean discharge rate and discharge rate at recruitment and its relation with the difference between target torque (10, 30, 50 and 70% MVC) and recruitment threshold was used as an estimate of the neural drive to VM and VL. Amplitudes of the interference EMG of the two muscles were analyzed as absolute and normalized root mean square values. Although the two muscles received similar neural drive, the absolute EMG amplitude and the size of the MUAPs were greater for VM than VL ($p<0.001$). Moreover, the size of the MUAPs explained most of the difference in EMG amplitude between VM and VL (~63% of explained variance). Normalized EMG amplitude was higher for VL than VM ($p<0.04$). These results indicate that EMG amplitude, even following normalization, does not reflect the neural drive to synergistic muscles. Moreover, absolute EMG amplitude is mainly explained by the size of MUAPs.

New and Noteworthy

EMG amplitude is widely used to indirectly compare the strength of neural drive received by synergistic muscles. However, there are no studies validating this approach with motor unit data. Here, we compared between-muscles differences in surface EMG amplitude and motor unit behavior. The results clarify the limitations of surface EMG to interpret differences in neural drive between muscles.

INTRODUCTION

Changes in the relative activations of synergistic muscles are believed to be associated to the development of musculoskeletal disorders (18). For example, researchers argue that pathologies such as patellofemoral joint pain and Achilles tendinopathy might occur due to misbalanced activation of the vasti and calf muscles, respectively (16, 18). For patellofemoral joint pain, it is assumed that a greater activation of the vastus lateralis (VL) compared to the vastus medialis (VM) muscle induces a lateral shift of the patella, leading to misalignment of the patellofemoral joint (16, 18). Although these explanations seem plausible, there is still no consensus in the literature (6, 29), mainly because of limitations of surface electromyography (EMG) amplitude in assessing muscle activation. While normalization of EMG amplitude with respect to its value during a maximal voluntary contraction (MVC) may increase reliability when comparing between subjects (4), normalization may cancel out changes in muscle activation following, e.g., training interventions. It has recently been shown that high-density EMG (HDEMG) systems allow more reliable estimates of signal amplitude without the need for normalization (13, 32). This is possible due to the large number of observation sites (tens of electrodes) over the muscle belly that compensate for the variability of EMG with electrode location. However, the use of several electrodes does not solve the problem of comparison between muscles and subjects.

In addition to the neural drive to the muscle, EMG amplitude estimates are also influenced by several other factors, such as muscle architecture, geometry, EMG crosstalk, and subcutaneous tissue thickness (10). Although normalization could help to improve between-muscle amplitude estimates, it is still not known if such measures really reflect differences in neural drive to the muscles. The direct way to measure the neural drive to muscles is by motor unit recordings. Recent research has shown the possibility to identify large populations of motor units with HDEMG (23, 25). However, even sampling relatively large number of motor units, it is not possible to directly compare the strength of the neural drive to different muscles since the decomposition cannot identify the entire pool of active motor units. In this study, we used an approach to compare the neural drives to synergistic muscles from HDEMG decomposition and we discuss its relations with EMG amplitude. For this purpose, we analyzed, across the decomposed motor unit populations, the relation between the increase in discharge rate from discharge rate at recruitment and the difference in torque with respect to the individual motor unit torque recruitment thresholds. We hypothesized that differences in EMG amplitude between VM and VL muscles would be largely determined by

the size of the motor unit action potentials (MUAPs) rather than differences in neural drive to the two muscles, and that normalization would not completely compensate for this influence.

MATERIALS AND METHODS

Participants

Eighteen healthy and physically active men (mean (SD) age: 29 (3) years, height: 178 (6) cm, mass: 79 (9) kg) were recruited. None of the participants reported any history of neuromuscular disorders or previous lower limb surgery. Subjects were asked to avoid any strenuous activity 24 h prior to the measurements. The ethics committee of the Universität Potsdam approved the study (approval number 26/2015), in accordance with the declaration of Helsinki (2004). All participants gave written, informed consent.

Experimental protocol

All participants performed submaximal and maximal knee extension contractions on an isokinetic dynamometer (CON-TREX MJ, PHYSIOMED, Regensdorf, Switzerland). All isometric knee extensions were exerted with the knee flexed to 90°. After placement of the surface EMG electrodes (see Data acquisition), subjects performed three maximal voluntary contractions (MVC) of knee extension each over a period of 5 s. Each of these trials was separated by 2 min of rest. The highest MVC value served as a reference to define the submaximal torque levels. After 5 minutes of rest, and following familiarization trials at low torque levels (10 and 30% MVC), subjects performed submaximal isometric knee extension contractions at 10, 30, 50 and 70% MVC in random order. Contractions at 10 and 30% MVC were maintained for 20 s, while the contractions at 50 and 70% MVC were sustained for 15 and 10 s respectively. In each trial, the participants received visual feedback of the torque applied by the leg to the dynamometer, which was displayed as a trapezoid (5 s ramps with hold-phase durations as specified above). Each contraction level was performed twice with a rest of 2 min following each contraction.

Data Acquisition

The surface EMG signals of VM and VL were recorded in monopolar derivation with a two-dimensional adhesive grid (SPES Medica, Salerno, Italy) of 13 × 5 equally spaced electrodes (1 mm diameter, inter-electrode distance of 8 mm). EMG signals were initially recorded during a brief voluntary contraction during which a linear non-adhesive dry electrode array of 8 silver-bar electrodes (1-mm diameter, 5-mm length, 5 mm interelectrode distance; SA 8/5, OT Bioelettronica, Torino, Italy) was moved over the skin to detect the location of the innervation zone and tendon regions (21). After the skin was shaved and cleansed with

abrasion and water, the electrode cavities of the grids were filled with conductive paste (SPES Medica, Salerno, Italy). Grids were positioned between the proximal and distal tendons of the VM and VL muscles with the electrode columns (comprising 13 electrodes) oriented along the muscle fibers. Therefore, the VM grid was positioned ~50º with respect to a line between the anterior superior iliac spine and the medial side of the patella while the VL grid was positioned ~30º with respect to a line between the anterior superior iliac spine and the lateral side of the patella ((1, 20, 22, 23) (Figure 1). Reference electrodes were positioned over the malleoli and patella of the dominant leg.

EMG and torque signals were sampled at 2048 Hz and converted to digital data by a 12-bit analogue to digital converter (EMG-USB 2, 256-channel EMG amplifier, OT Bioelettronica, Torino, Italy, 3dB, bandwidth 10-500 Hz). EMG signals were amplified by a factor of 2000, 1000, 500, 500 and 500 for the 10, 30, 50, 70 and 100% MVC contractions, respectively. Data were analysed offline using Matlab (The Mathworks Inc., Natick, Massachusetts, USA). The 64-monopolar EMG channels were re-referenced offline to form 59 bipolar channels as the differences between adjacent electrodes in the direction of the muscle fibers.

Signal analysis

Motor unit analysis. The EMG signals recorded during the submaximal isometric contractions (from 10 to 70% MVC) were decomposed offline with a method that has undergone extensive validation (26). The accuracy of the decomposition was tested with the silhouette measure, which was set to ≥0.90 (26). The signals were decomposed throughout the whole duration of the submaximal contractions and the discharge times of the identified motor units were converted in binary spike trains. The mean discharge rate and discharge rate variability (coefficient of variation of the inter-spike-interval, CoVisi), were calculated during the stable plateau torque region. Discharge rate at recruitment was calculated using the first six discharges of the motor units (8). The motor unit recruitment threshold was defined as the knee extension torque (%MVC) at the time when the motor unit began discharging action potentials. Discharges that were separated from the next by <33.3 ms or >200 ms (30 and 5 Hz, respectively) were discarded from the mean discharge rate and CoVisi calculation since such discharges are usually considered decomposition errors (22). Motor unit conduction velocity (MUCV) was measured from a minimum of three to a maximum of nine double-differential channels (manual selection) (23). Channels that had the clearest propagation of MUAPs, with the highest amplitude in the columns of the grid and a coefficient of correlation between channels ≥0.9, were selected for further analysis. Finally, the amplitude of the MUAPs was calculated as the MUAP RMS averaged over all channels of the grid

(MURMS). VM and VL motor units were matched by their recruitment threshold with a tolerance of ±0.5% MVC. The matched motor units were then grouped in four classes, according to their recruitment thresholds ([0-10] % MVC, [10-30] % MVC, [30-50] % MVC, [50-70] % MVC).

The discharge rate of motor units with the same recruitment thresholds in the two muscles was used as a measure to compare the neural drive to muscles. This measure corresponds to the rate of change of discharge rate (average discharge rate during the stable force region – discharge rate at recruitment) as a function of the increase in torque from the recruitment threshold [target torque (10, 30, 50 and 70% MVC) – recruitment threshold torque]. A difference in this association between the two muscles across the populations of decomposed motor units indicates differences in synaptic input received by the motor neuron pools of the two muscles and therefore differences in neural drive to the muscles.

Interference EMG. The root mean square values (RMS) obtained from submaximal and maximal contractions, were averaged over all channels of the electrode grid (20). During the submaximal isometric contractions, the RMS was computed from the HDEMG signals in intervals of 1 s. These values were extracted from the stable-torque region of the contractions (e.g., hold-phase of 15 seconds at 50% MVC). RMSs of the maximal (MVC) contractions were analyzed in a time window of 250 ms centered at the peak EMG activity (20). Global conduction velocity (muscle fiber conduction velocity) was calculated from double differential signals obtained along the fiber direction (columns of the grid). In order to maximize the accuracy of global conduction velocity estimates, three contiguous columns with four to six channels with the highest cross-correlation in propagation were selected (9). Muscle fiber conduction velocity estimation was obtained with a multichannel maximum-likelihood algorithm that was previously shown to provide accurate estimates (standard deviation <0.1 ms) (12).

Amplitude normalization. Both absolute RMS and MURMS were normalized to the RMS value obtained during the MVC in order to analyze the effects of normalization on submaximal RMS amplitude of the interference EMG (absolute RMS) as well as on MURMS between muscles.

Statistical Analysis

The Shapiro-Wilk test was used to check the normality of all variables. Sphericity was checked by Mauchley's test and if violated, the Greenhouse-Geisser correction was made to

the degrees of freedom. Statistical significance was set at $p < 0.05$. Results are expressed as mean and standard deviation (SD).

EMG (absolute RMS, normalized RMS and muscle fiber conduction velocity) and motor unit variables (MURMS, discharge rate, CoVisi, motor unit conduction velocity and normalized MURMS) were compared between muscles at each torque level with a two-way repeated measures analysis of variance (ANOVA) with factors muscle (VM and VL) and torque (10, 30, 50 and 70% MVC). When repeated measures ANOVA was significant, pairwise comparisons were made with a Student-Newman-Keuls (SNK) post-hoc test. The difference between VM and VL mean discharge rate and discharge rate at recruitment and its relation with the difference between target torque and recruitment threshold were analyzed by linear regression. The slopes of the linear regression were compared between the two muscles by analysis of covariance (ANCOVA) (33). The same analysis was applied to VM and VL MURMS vs. recruitment threshold.

Finally, a multiple linear regression (stepwise) analysis was performed on EMG/motor unit parameters to identify the variables that predicted the differences between VM and VL absolute RMS. Therefore, the percent (%) difference in absolute RMS between VM and VL was used as the predictor variable and the % differences in MU behaviour/properties were regarded as independent variables. Each torque level was analysed independently (e.g. absolute RMS % difference between VM and VL at 30% MVC was compared with motor unit variables obtained at the same torque level). The partial eta-squared (ηp^2) for ANOVA was used to examine the effect size of the differences between EMG and motor unit parameters between muscles. A ηp^2 less than 0.06 was classified as “small”, 0.07-0.14 as “moderate”, and greater than 0.14 as “large” (5).

RESULTS

Interference EMG

Absolute RMS (Figure 2a) was significantly higher for VM than VL at 30, 50 and 70% MVC (interaction: muscle-torque, $p<0.0001$, $\eta p^2=0.79$). However, muscle fiber conduction velocity (Figure 2b) was similar for the two muscles (interaction: muscle-torque, $p=0.96$, $\eta p^2=0.019$).

238 Decomposed motor unit populations
239 A total of 641 and 583 motor units (with a SIL≥0.90) were identified in VM and VL,
240 respectively (considering all torque levels). The average number of motor units accurately
241 identified per subject at each torque level was 8 (0.7) and 7 (1.2) in VM and VL, respectively.
242 According to their recruitment threshold, 348 motor units were matched between VM and
243 VL. Per subject, an average of 6.2 (3.0), 5.0 (2.5), 5.7 (2.8) and 3.3 (2.0) motor units were
244 matched between VM and VL at 10, 30, 50 and 70% MVC, respectively. The average
245 recruitment threshold of the matched motor units at 10, 30, 50 and 70% MVC was 7.5, 23.3,
246 38.2 and 56.2% MVC, respectively.
247
248 Discharge rate and discharge rate variability
249 The discharge rate of VM was greater than for VL motor units as revealed by a significant
250 effect of muscle ($p=0.009$, $\eta p^2=0.38$) (Figure 3a). However, the regression lines of delta
251 discharge rate [discharge rate – discharge rate at recruitment] vs. delta torque [target torque –
252 recruitment threshold] were not different between muscles (slope of the regression lines,
253 $p=0.12$, intercept, $p=0.74$) (Figure 3b). Finally, There was no difference in discharge rate
254 variability between muscles as CoVisi (Figure 4) remained similar at all torque levels
255 (interaction: muscle-torque, $p=0.4$, $\eta p^2=0.07$).
256
257 Size and conduction velocity of MUAPs
258 MURMS (Figure 5a) was significantly greater for VM than VL at 30, 50 and 70% MVC
259 (interaction: muscle-torque, $p<0.0001$, $\eta p^2=0.57$). Moreover, MURMS increased at a greater
260 rate with recruitment threshold for VM than for VL ($p<0.0001$, Figure 5b). Motor unit
261 conduction velocity (Figure 6) showed significantly higher greater at 70% MVC for VM
262 (interaction: muscle-torque, $p=0.023$, $\eta p^2=0.46$).
263
264 Multiple linear regression
265 Motor unit variables that significantly differed between muscles were entered into the
266 multiple linear regression analysis to explain the differences in absolute EMG amplitude
267 between muscles. Therefore, the difference (%) in VM-VL MURMS, discharge rate, and
268 motor unit conduction velocity were regarded as independent variables. Table 1 reports the
269 results of the multiple regression. At 10% MVC only MURMS was entered in the model,
270 explaining 71% of the variance for the difference (%) in VM-VL absolute RMS. At 30%,
271 both MURMS and discharge rate entered in the model, however MURMS explained most of

the variance (53% MURMS vs. 13.2% for discharge rate). Similar results were obtained at 50% MVC where MURMS explained 72% of the difference between VM-VL absolute RMS, with discharge rate just explaining 7.7% of the variance. Finally, at 70% MVC, only MURMS was entered in the model, explaining 57% of the %difference in VM-VL absolute RMS.

Normalized amplitude

Normalized RMS (Figure 7) showed systematically higher values for VL across all torque levels (effect: muscle, $p=0.039$, $\eta p^2=0.23$). Conversely, normalized MURMS did not show any difference between muscles at any torque level (effect: muscle, $p=0.46$, $\eta p^2=0.04$, interaction: torque-muscle, $p=0.12$, $\eta p^2=0.11$).

DISCUSSION

This study shows that differences in EMG amplitude between synergistic muscles are mostly explained by differences in MUAP size (MURMS), with little influence of other motor unit properties. Moreover, EMG normalization does not provide clear explanation of differences in muscle activation between the vasti. Taken together, the results suggest that amplitude parameters (in absolute values or normalized) should not be used to infer differences in neural drive between synergistic muscles.

Neural drive to VM and VL muscles

Due to current limitations in EMG decomposition, it is not possible to identify the full populations of active motor units. For this reason, the neural drives cannot be directly compared between muscles. We compensated for this limitation by an indirect assessment of the strength of the neural drive. Matching synergistic muscles motor units by recruitment threshold allows a direct comparison of motor unit parameters across muscles since these units should contribute similarly to the exerted joint torque. In the present study, we used motor unit discharge rate as a measure to compare the drive between muscles. Because the discharge rate depends on the torque relative to the recruitment threshold, we focused on the rate of change of discharge rate (mean discharge rate – discharge rate at recruitment) with respect to the difference between exerted torque and recruitment threshold across the decomposed motor unit populations. This analysis provides an estimate of the synaptic input received by the motor neuron pools of VM and VL. This approach indicated a similar change

in motor unit discharge rate with torque (figure 3b) despite a difference in absolute discharge rates that can be due to the random sampling of motor units in the two muscles. This suggests that the net excitatory synaptic input to the pool of motor neurons of the vasti was similar, with a similar drive from motoneurons to muscle units. This conclusion is in agreement with a study showing that VM and VL share most of their synaptic input (19). We also did not observe differences in discharge rate variability (CoVisi) between the two muscles (Figure 4), in agreement with previous results (34). The present results show that, despite a difference in mean absolute discharge rates between motor units of the VM and VL, the two muscles did receive similar strengths of neural drives. Differences in VM and VL surface EMG amplitude therefore do not reflect differences in the neural drive between the vasti, as also confirmed by the multiple regression analysis.

EMG amplitude and muscle fiber conduction velocity

Surface EMG amplitude is usually used to infer the magnitude of the neural drive to muscles. However, EMG amplitude depends on both motor unit behavior (recruitment, discharge rate and discharge rate variability) and muscle fiber properties (MUAP size and conduction velocity) (10, 11). In the present study, despite similar neural drives to the VM and VL, the EMG amplitude for VM was significantly greater than for VL for torques in the range 30%-70% MVC. These results are consistent with other reports on absolute EMG amplitude for these two muscles (14, 20, 32). EMG amplitude is influenced by muscle's geometry, architecture, crosstalk and subcutaneous tissue thickness (10, 27). Since the observed differences in EMG amplitude between muscles did not correspond to differences in neural drive, they are mainly explained by these anatomical factors. Although previous research has reported similar subcutaneous tissue thickness for the distal VM and VL (3), it has also been shown that the distal VM has a larger cross sectional area and greater fascicle angle compared to the distal VL (2). Indeed, recent research has shown that differences in muscle architecture can influence EMG amplitude, even when the muscle is activated at a similar intensity (30).

Muscle fiber conduction velocity estimated from the interference EMG was similar between the vasti, in agreement with previous studies (3). However, motor unit conduction velocity differed between muscles. Muscle fiber conduction velocity is associated to fiber diameter (15) but also depends on the level of muscle acidosis (28), temperature (7), muscle fatigability (21), subcutaneous tissue thickness (31), exercise training (23, 31), discharge rate

(24). Because of these factors of influence, the relation between average and motor unit muscle fiber conduction velocity is not exactly linear.

EMG amplitude and MUAP size

As for absolute EMG amplitude, the size of MUAPs was significantly higher for VM in the range of torques above or equal to 30% MVC. Moreover, MURMS increased at a faster rate with recruitment threshold for VM than VL (Figure 5b). This is consistent with a recent report comparing VM and VL MUAP peak-to-peak amplitude (22). As for EMG amplitude, MURMS is also influenced by muscle's geometry, architecture and subcutaneous tissue thickness (10, 27); therefore it is not surprising to find similar results for absolute RMS and MURMS. Accordingly, results from the multiple linear regression (Table 1) showed that most of the variance of the difference between absolute RMS of VM and VL was explained by MURMS. This result directly indicates that that the neural drive has a relatively small influence on EMG amplitude with respect to the MUAP waveforms.

Amplitude normalization

Since a vast number of studies apply normalization of the surface EMG prior to comparing levels of muscle activations (4, 16), we analyzed the effect of normalization of both EMG amplitude and MUAP size with respect to MVC. Even though normalization decreased the VM/VL activation ratio and cancelled out the differences in MUAP size between muscles, normalized EMG amplitude was greater for VL compared to VM that is contrary to the result without normalization. This result does not correspond to the estimated similar neural drive to the two muscles (figure 3b) and explains the divergent results across studies on normalized activations of the VM and VL in healthy subjects (29) and patients with musculoskeletal disorders (e.g. patellofemoral pain syndrome) (17). Taken together, our findings suggest that neither absolute nor normalized EMG amplitude (even when recorded from HDEMG electrodes) are appropriate for inferring differences in neural drive between muscles.

Conclusion

The difference in surface EMG amplitude between VM and VL muscles was mostly explained by differences in MUAP size, with little effect of motor unit properties associated to the neural drive to muscles. EMG amplitude levels are therefore determined by peripheral properties rather than by the neural activation. Normalization of the EMG compensates for the differences in MUAP sizes but is still a poor determinant of neural activation.

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Figure captions

Figure 1. Placement of the HDEMG electrodes. Vastus medialis (VM) electrode grid was placed ~50º with respect to a line between the anterior superior iliac spine and the medial side of the patella (dashed lines, left) while the VL grid was positioned ~30º with respect to a line between the anterior superior iliac spine and the lateral side of the patella (dashed lines, right).

Figure 2. Interference EMG parameters [mean (SD)] for vastus medialis (VM, white dots) and vastus lateralis (VL, black dots) at 10, 30, 50 and 70% of the maximum voluntary contraction torque (MVC). A) Absolute root mean square (ABS RMS). B) Muscle fiber conduction velocity. Presented values were averaged for each subject and presented at each submaximal target torque. * $P<0.001$.

Figure 3. Motor unit (MU) discharge rate calculated from recruitment-threshold matched MUs from vastus medialis (VM, white dots) and vastus lateralis (VL, black dots) at 10, 30, 50 and 70% of the maximum voluntary contraction torque (MVC). A) MU discharge rate values [mean (SD)] were averaged for each subject and presented at each submaximal target torque (10, 30, 50 and 70% MVC), # main effect of muscle $P=0.009$. B) Linear regression analysis of the difference between VM and VL mean discharge rate and discharge rate at recruitment (Y-axis) and the difference between target torque (10, 30, 50 and 70% MVC) and MU recruitment threshold (X-axis). Linear regression equations are shown in the figure, both lines increased significantly ($P<0.0001$) and their R^2 values were 0.19 and 0.12, for VM and VL respectively.

Figure 4. Motor unit (MU) coefficient of variation of the inter-spike interval (CoVisi) calculated from recruitment-threshold matched MUs from vastus medialis (VM, white dots) and vastus lateralis (VL, black dots) at 10, 30, 50 and 70% of the maximum voluntary

contraction torque (MVC). Presented values were averaged for each subject and presented at each submaximal target torque.

Figure 5. Motor unit (MU) root mean square amplitude (MURMS) [mean (SD)] extracted from recruitment-threshold matched MUs from vastus medialis (VM, white dots) and vastus lateralis (VL, black dots) at 10, 30, 50 and 70% of the maximum voluntary contraction torque (MVC). A) MURMS values [mean (SD)] were averaged for each subject and presented at each submaximal target torque (10, 30, 50 and 70% MVC), * $P<0.01$. B) VM and VL MURMS vs. recruitment threshold regression lines. Both lines increased significantly with torque ($P<0.0001$) and displayed significantly different slopes ($P<0.0001$); R^2 values are shown in the figure.

Figure 6. Motor unit (MU) conduction velocity [mean (SD)] extracted from recruitment-threshold matched MUs from vastus medialis (VM, white dots) and vastus lateralis (VL, black dots) at 10, 30, 50 and 70% of the maximum voluntary contraction torque (MVC). Presented values were averaged for each subject and presented at each submaximal target torque. * $P<0.01$.

Figure 7. Normalized EMG and motor unit (MU) amplitude [mean (SD)] for vastus medialis (VM, white dots) and vastus lateralis (VL, black dots) at 10, 30, 50 and 70% of the maximum voluntary contraction torque (MVC). A) Normalized root mean square EMG (EMG RMS NORM), B) Normalized MU root mean square (MURMS NORM). # main effect of muscle $P=0.039$.

Figure 1

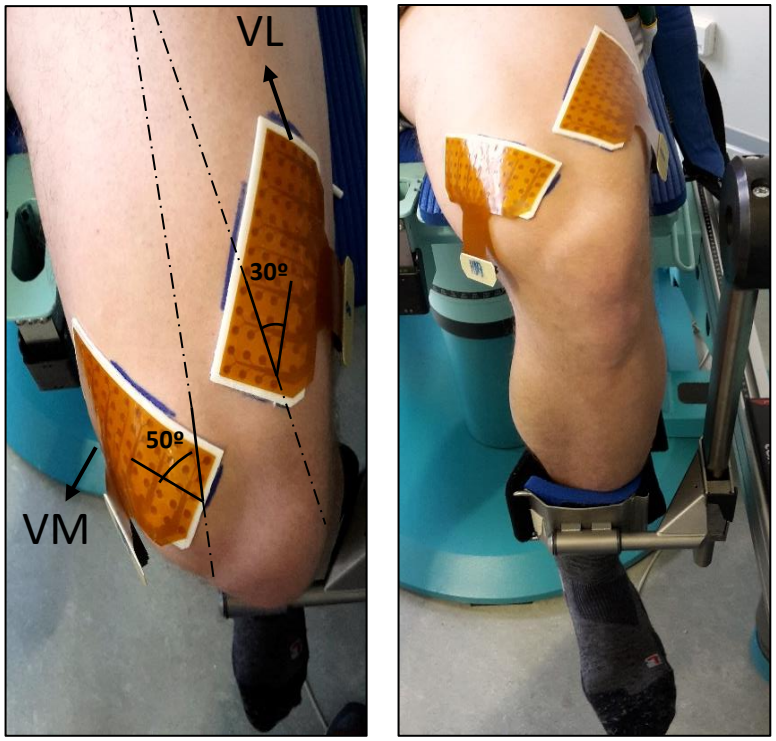

Figure 2

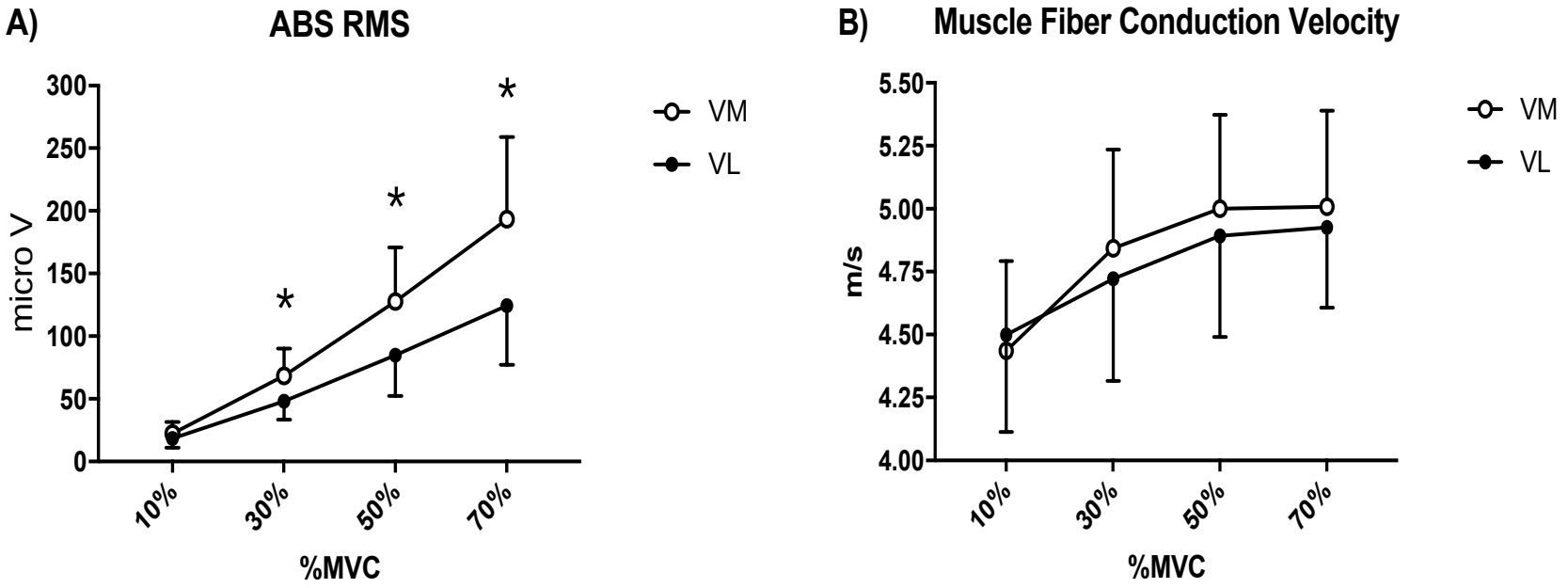
A)
ABS RMS
micro V
300
250
200
150
100
50
0
*
*
*
10%
30%
50%
70%
%MVC
VM
VL
B)
Muscle Fiber Conduction Velocity
m/s
5.50
5.25
5.00
4.75
4.50
4.25
4.00
10%
30%
50%
70%
%MVC
VM
VL

Figure 3

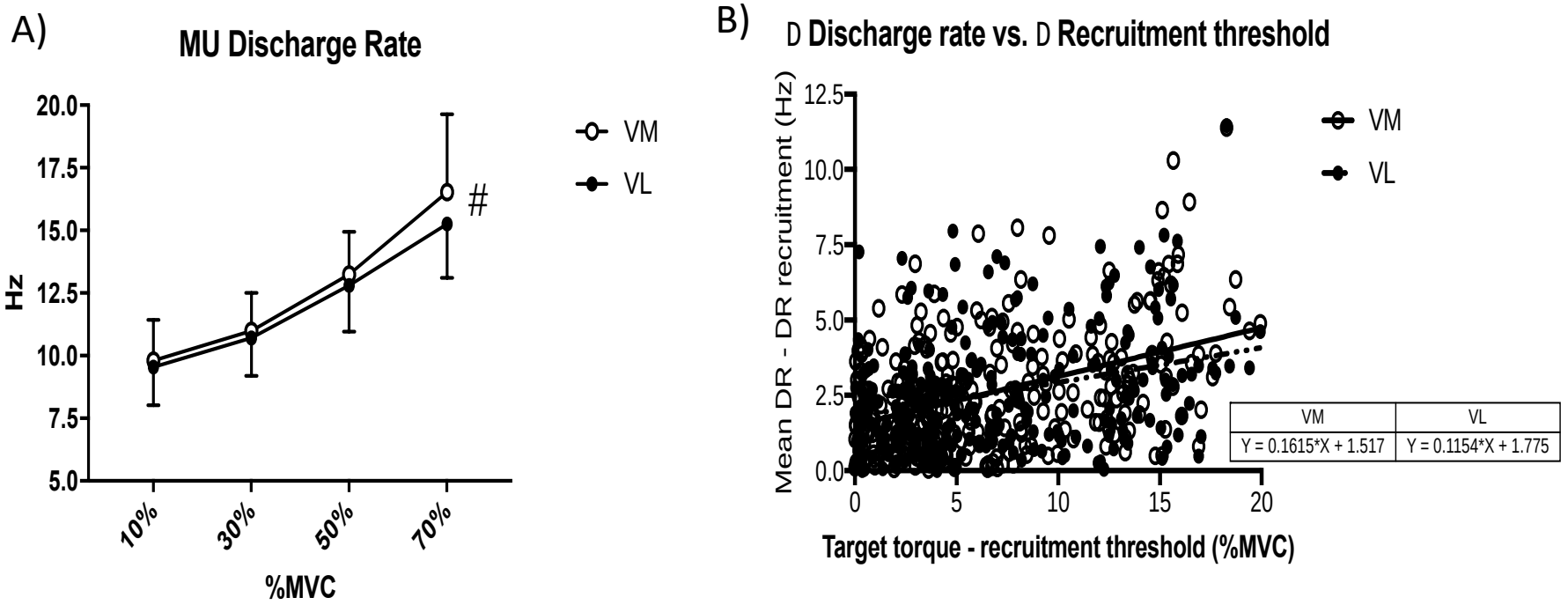
A)
MU Discharge Rate
20.0
17.5
15.0
12.5
10.0
7.5
5.0
Hz
10%
30%
50%
70%
%MVC
#
VM
VL
B)
D Discharge rate vs. D Recruitment threshold
Mean DR - DR recruitment (Hz)
12.5
10.0
7.5
5.0
2.5
0.0
0
5
10
15
20
Target torque - recruitment threshold (%MVC)
VM
VL
VM
VL
Y = 0.1615*X + 1.517
Y = 0.1154*X + 1.775

Figure 4

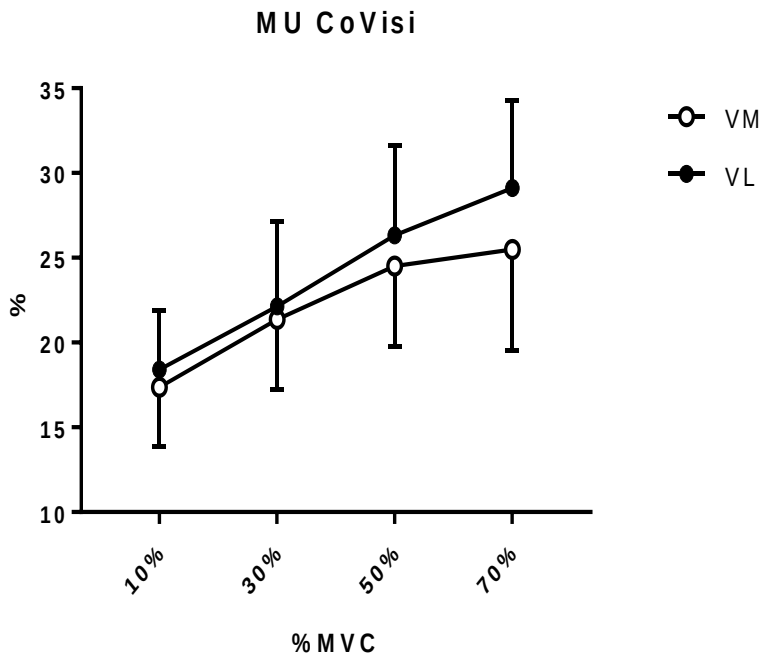

Figure 5

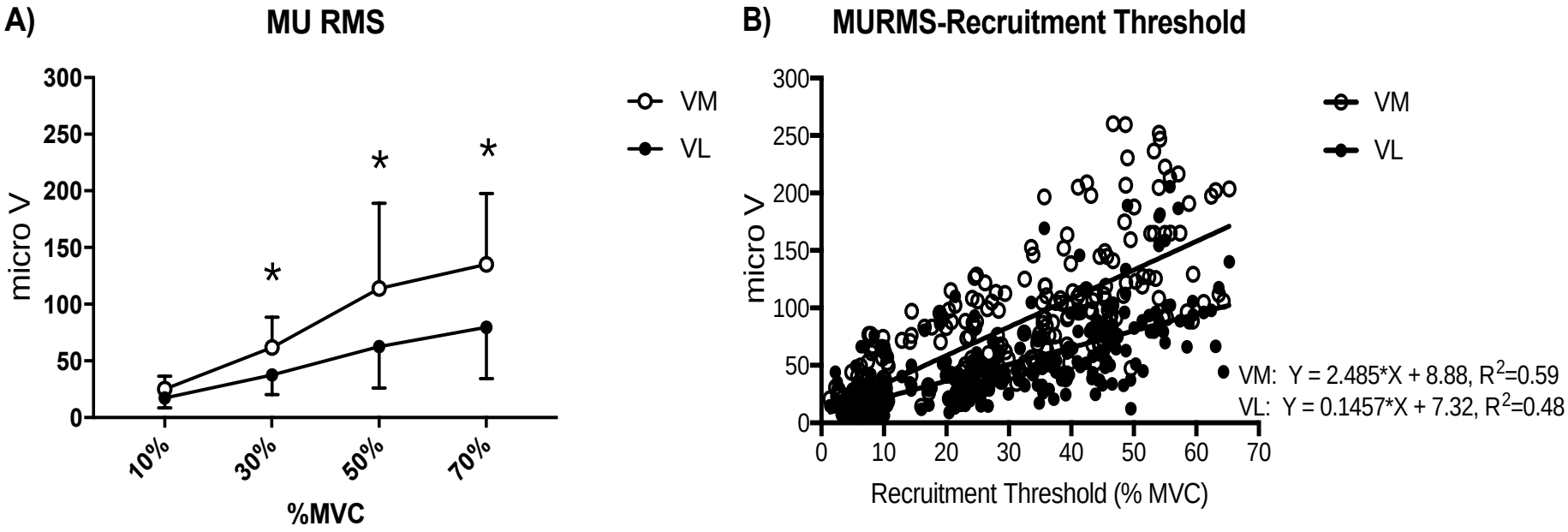
A)
MU RMS
micro V
300
250
200
150
100
50
0
10%
30%
50%
70%
%MVC
*
*
*
VM
VL
B)
MURMS-Recruitment Threshold
micro V
300
250
200
150
100
50
0
0
10
20
30
40
50
60
70
Recruitment Threshold (% MVC)
VM
VL
VM: Y = 2.485*X + 8.88, R2=0.59
VL: Y = 0.1457*X + 7.32, R2=0.48

Figure 6

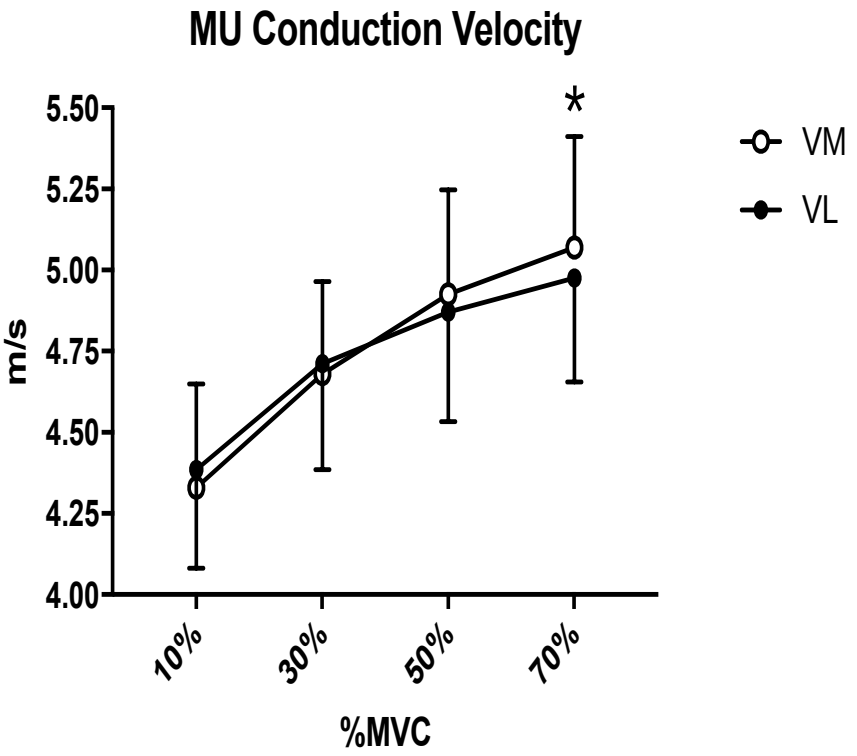
MU Conduction Velocity
5.50
5.25
5.00
4.75
4.50
4.25
4.00
m/s
*
VM
VL
10%
30%
50%
70%
%MVC

Figure 7

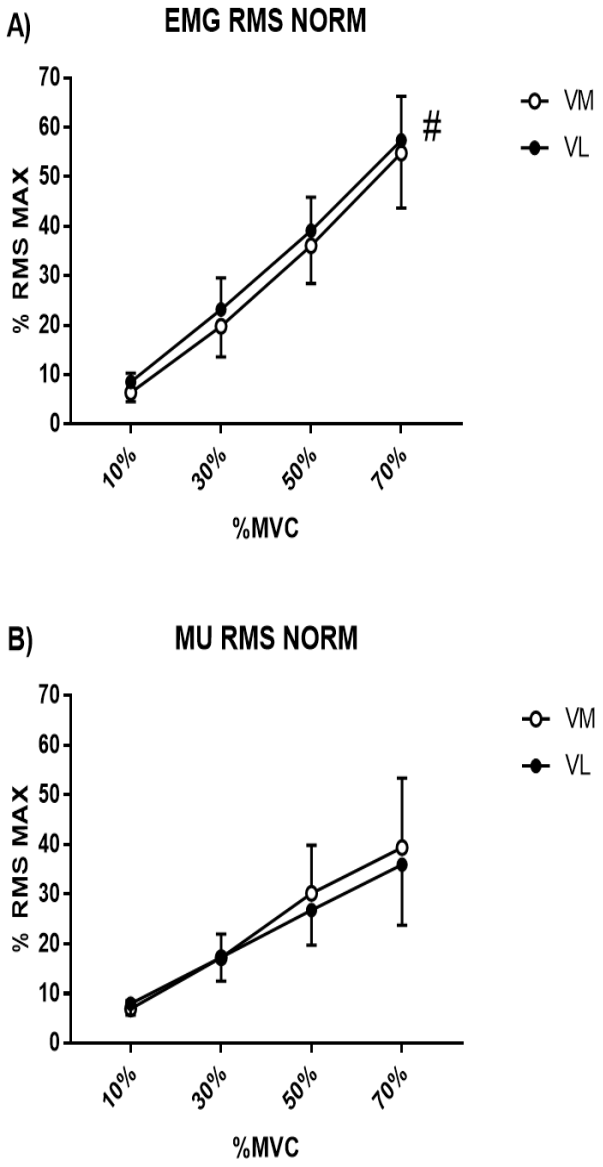
A)
EMG RMS NORM
% RMS MAX
70
60
50
40
30
20
10
0
10%
30%
50%
70%
%MVC
#
VM
VL
B)
MU RMS NORM
% RMS MAX
70
60
50
40
30
20
10
0
10%
30%
50%
70%
%MVC
VM
VL

Table 1. Percent difference [%, mean (SD)] and bivariate correlation coefficients (r) between predictor variable (% change in VM-VL EMG RMS) and independent variables: %change in VM-VL motor unit (MU) RMS, %change in VM-VL in MU discharge rate (DR) and %change in VM-VL MU conduction velocity (CV)

Torque Level (%MVC)	%Difference in EMG RMS	% Difference in MU RMS	% Difference in MU DR	% Difference in MU CV
10%	14.8 (25.3)	25.2 (34.1), r= 0.84**	2.3 (7.8), r=-0.48	-1.4(4.9), r=-0.27
30%	27.2 (19.4)	36.5 (25.4), r=0.73**	2.3 (7.8), r=0.14	-0.7 (2.5), r=0.12
50%	32.8 (12.5)	42.3 (19.6), r=0.85**	4.1 (9.5), r=0.02	1.3 (3.1), r=-0.2
70%	34.9 (15.8)	42.2 (19.1), r=0.76**	6.2 (13.3),r=0.26	1.8 (3.9), r=0.07

** Significant correlation ($p<0.0001$)