Neuromechanical Control of Ballistic Contractions: Decoding Motor Unit Activity from hdEMG

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***Abstract*—The motor unit activity driving sub-maximal ballistic contractions is extracted from high density EMG using blind convolutive separation. We describe how central (e.g. recruitment and discharge rate) and peripheral (e.g. conduction velocity) factors collectively contribute towards motor output. Conduction velocity is characterized by a monotonic increase, despite a decrease in discharge rate after the initial phase of force development, and is moderately correlated with motor output ($R^2 = 0.62 \pm 0.28$). The dynamics of CV are in agreement with the transience of ionic gradient changes and may contribute to explain the late ballistic rise in motor output through a multiplicative relationship between peripheral and central control factors. Individual motor units exhibit short term synchronization over long step-and-hold contractions, and the motor output is highly correlated with both individual ($R^2 = 0.70 \pm 0.09$) and total ($R^2 = 0.78 \pm 0.07$) discharge patterns. The amplitude of the interference EMG signal is strongly correlated with discharge rate ($R^2 = 0.84 \pm 0.06$) and moderately correlated with the proportion of active motor units ($R^2 = 0.61 \pm 0.13$).**

***Index Terms*—ballistic contraction, motor output, high density surface EMG, blind source separation, motor neuron discharge rate, muscle fiber conduction velocity**

I. Introduction

BALLISTIC contractions consist in voluntary generation of muscle tension to achieve a target force as fast as possible, with undefined Rate of Force Development (RFD). The ability to execute rapid and forceful movements can be related to aging and neuro-muscular disorders and is crucial for sports performance. In daily living, explosive contractions may be important to ensure safety when movement stability is disturbed [1][2], and explosive voluntary force has been associated with balance ability in aged populations [3].

A. State of the Art

1) Ballistic Contractions: The mechanisms of neural control of ballistic contractions were investigated and compared against ramp contractions by Desmedt et al. [4], who described the fundamental features of neural activity in the Tibialis Anterior (TA) during ballistic isometric dorsiflexion. One of their main findings was the almost simultaneous recruitment of several motor units (MUs) at the onset of contraction, starting at a high firing frequency [4]. This strategy is markedly different from the gradual recruitment and discharge rate (DR) increase occurring in fast but tracked contractions [5]. In both gradual and explosive contractions, higher peak forces can be achieved by increasing the number of discharges over the initial sudden burst, and this becomes the primary mechanism for achieving greater maximal force above a certain level (however, a given ballistic contraction requires the activation of more motor units than a ramp contraction with the same maximal force) [4]. Additionally, maximal RFD capacity has also been related to maximal discharge rate in rapid contractions [6]. This is in accordance with the fact that, for different ramp contractions, the recruitment threshold of a given MU varies proportionally with the rate of force development [7], and supports a generalized spinal organization of motor units, dependent on the goal force.

Ballistic capacity may be affected by the maximal voluntary contraction, fiber type composition, stiffness of the muscle-tendon unit twitch profile and the strength of neural signals [8][2]. Ballistic performance is typically assessed in isometric contractions, by measuring force and rate of force development (either absolute or normalized to Maximal Voluntary Contraction, MVC) in standardized intervals [7][9][2]. When evaluating central control from surface electromyogram (sEMG) amplitude, or innate muscle contractile properties from evoked contractions, the most significant factor underlying differences in ballistic motor output between athletes and un-trained individuals is the intensity of the neural drive to muscles [1]. However, Folland et al. [2] found that the correlation between agonist muscle EMG activity and force changes throughout the contraction, within a range from 0.45 to 0.71, and EMG correlation with RFD ranges from 0.16 to 0.71. While EMG amplitude yields an overall estimate of neural activity, it provides limited insight into specific neural control strategies (e.g. recruitment and rate coding). For instance, Del Vecchio et al. [10] have recently reported the limited predictive value of EMG amplitude and spectral features with regard to neural factors, in particular recruitment, but is not known whether similar results should be expected in ballistic contractions.

The existing report on motor unit activity in ballistic contractions [4] is based on relatively limited populations of neurons, and does not investigate peripheral mechanisms (e.g. conduction velocity, CV). Up to date, apart from the characterization of ballistic contractions in terms of a time-independent ballistic-threshold, no other relationships between control variables (e.g. recruitment, discharge rate and conduction velocity) and the motor output have been investigated. Folland et al. [2] reported changes in relative contribution of neural and morphological factors throughout ballistic contractions, but seldom physiological interpretations can be drawn from their results. Moreover, a strong linear relationship between neural drive estimated from EMG amplitude and motor output has only been confirmed in the first 40 ms of ballistic contractions [1].

Until recently, accurate assessment of motor neuron ac-

tivity could only be performed invasively [5][4], which limited and possibly biased the sampled neurons [11]. To our knowledge, there are no studies of the interactions between neural and peripheral control of ballistic contractions using individual motor neuron (MU) discharge rate information from significantly large MN populations. On the other hand, the recent investigations on the relative contribution of neural and contractile properties towards performance of ballistic contractions were based on elementary assessment of neural activation, whose accuracy is less than state-of-the-art EMG decoding. Furthermore, ballistic contractions have not been characterized with regards to the changes in muscular electrical properties throughout the contraction, namely CV, which has been included in the parameters associated with the size principle [12].

Conduction Velocity: The spinal output is propagated through peripheral axons and across the neuro-muscular junctions (the synapses between MNs and muscle fibers, NMJs). In non-pathological cases, a NMJ is an extremely reliable synapse, where the transduction of MN action potentials (APs) into chemical signals (mediated by the neurotransmitter acetylcholine) inexorably originates action potentials in the motor plates of the corresponding muscle units. These action potentials are in turn propagated along the muscle fiber membranes to generate contraction across their length.

Analysis of stimulated tibialis anterior fibers shows that muscle fiber CV is closely related with functional and structural features included in the size principle: CV is higher in muscle fibers with larger diameter, greater twitch force, and shorter rise time [12]. CV has been reported to decrease with fatigue, either throughout maximal contractions [13] or contractions sustained for long periods [14][15][16][17]. As described in Chapter 1, the literature explores the relationships between muscle fiber properties and CV, describes fatigue-related changes in CV (correlating them with EMG), and evaluates motor performance with respect to general CV properties. Most published studies focus on CV changes in the course of very long contractions (e.g. 40 to 300 s [18][19]) and have poor time resolution (e.g. 0.5 or 1 second [19][20]). On the other hand, the progression of CV at the beginning of contraction has received minor attention. The existing reports confirm that CV follows the size principle in voluntary contractions [11][21]. Particularly, Masuda and De Luca [22] found a correlation between CV and recruitment threshold in gradual contractions. In their study, the rise in CV followed the rise in force, and was mostly attributed to the activation of larger fibers, according to the size principle; in this study, even though the individual motor unit discharges were extracted, the relationship between measures of rate coding and CV was not investigated. More recently, Del Vecchio et al. [11] corroborated the strong correlation between CV and recruitment threshold in larger populations of neurons, whose activity was decoded with convolutive blind source separation method in ramp contractions. They also reported the unimodality (i.e. absence of distinct classes) of motor units with regards to CV and fiber diameter.

In agreement with the contribution of MU synchrony and DR towards CV development [14][23], there is experimental evidence of an increase in CV with increased rate of muscle stimulation (i.e. induced discharges) [24]. Yet, it still is not clear how this translates into voluntary contractions with natural neural firing, since most studies considering the effects of DR on CV employ a fixed discharge rate for each single contraction [15][25]. Nevertheless, the velocity recovery function (VRF), relating the inter-spike intervals to the excitability and conductivity of muscle fibers, reveals both subnormal and and supranormal regions [26][27]. Using computational simulations of muscle excitation at different rates, Fortune and Lowery [26] have obtained an estimate of the variation in muscle fiber CV with respect to the instantaneous firing rate, which is consistent with experimental data. Noticeably, K^+ channels and T-tubular system were central parameters in this VRF derivation, supporting their strong impact on conduction velocity development. Finally, it is not known how CV progresses in the case of ballistic contractions, where recruitment and DR have a markedly different behaviour.

While the literature on CV variation with fatigue is relatively vast, to the author's knowledge, there is only one account of the relationships between neural control and changes in CV at the onset of gradual voluntary muscle tension, which considers recruitment and disregards DR [22]. Furthermore, no literature on the impact of physiological discharge rates on CV or on the variation of CV during ballistic contractions.

B. Motivation

Our investigation addresses the gap in the literature on neural and peripheral control of ballistic contractions, using hdEMG decomposition to extract the individual MU spike trains from significantly large MN populations. Over the last decade, the development of new EMG decomposition methods [28], particularly the blind convolutive source separation of high density multi-channel recordings [29], introduced the possibility of non-invasive assessment of the complete discharge profiles of large populations of neurons [10]. In the current study the activity of 188 motor neurons is assessed during ballistic contractions of the tibialis anterior and its association with motor output (e.g. force and rate of force development) or muscle conductivity.

The following research intends to contribute to clarify the basic neural strategies and electro-physiological features in fast production of a target force, complementing the scant literature on neuro-motor control of ballistic contractions. Its main goal is to analyze motor neuron excitation patterns during ballistic contractions and to quantify how they relate to both motor output and global surface EMG variables, aiming to provide new insights into performance mechanisms for power-oriented tasks. This investigation involves interpreting signals transmitted from the central nervous system (CNS) and their relationship with muscle physiology, and estimating how motor output depends on conduction velocity, recruitment and discharge rate.

Despite being widely employed in research and clinical practice (e.g. to infer the quality of therapeutic or resistance exercises [30]), global surface EMG interpretation is not trivial and has recently been questioned [31]. Nevertheless, surface

EMG remains an extremely versatile technology, with a much wider application range than high density EMG decomposition and allowing real-time processing. Besides describing the evolution of control parameters during ballistic contractions, our investigation analyzes the predictive value of the surface EMG signal for the assessment of neuro-muscular variables, contributing for the open discussion on whether EMG is over-exploited for inference of neuro-mechanical variables, such as motor unit recruitment and the type of recruited muscle fibers, or prediction of neuro-muscular adaptations, such as hypertrophy.

II. Methods

A. Experimental Protocol

Fifteen healthy subjects performed isometric dorsiflexions at sub-maximal and maximal contraction levels. While sitting on a chair, the subjects extended their dominant leg and dorsiflexed the angle to 30° from neutral position. The ankle position was strongly fixed with Velcro tapes placed around the foot and the ankle. A strain gauge load cell was attached in series with the tape on the ankle, perpendicularly to the lateral malleolus.

With the aid of a dry electrode array, the proximal and distal innervation zones of the TA were determined; in each zone, the direction of the fibers was identified by detecting the position with greatest consistency in AP shape propagation when moving the array over the skin surface. A 64-electrode high density EMG grid, with 5 columns and 13 rows of 1 mm wide gold-coated electrodes, with a 8 mm interelectrode distance d_G. was placed on the skin, after shaving, abraising and cleaning it with 70% ethanol, so that 4 electrode rows were over the innervation, and the columns were parallel to fiber direction. The electrodes were individually covered with a conductive ointment before placement of the grid, which was attached to the skin with adhesives. The monopolar multi-channel EMG signals were acquired with and amplifier (bandwidth 3 dB, 10-500 Hz).

Force and EMG signals were sampled simultaneously at 2048 Hz and 12 bits per sample. The experimental sessions started with three MVC trials, separated by at least 30 s, where subjects received auditory incentive to contract as forcefully as possible for at least 3 s. The force transducer signal was used as a standardizing factor and feedback signal for performance of contractions by the subjects. The greatest force achieved in the course of the three trials was selected as the MVC. Subsequently, the subjects performed at least four ballistic contractions to 70% of their MVC. The subjects had to reach the goal force as fast as possible, guided by a feedback monitor showing the desired force level and the transducer signal in real time. Each ballistic contraction was followed by a plateau, in which the subject tried to hold the tension at the goal level (this type of contraction has been referred to as *step-and-hold* [6]). Motor output (i.e. force transducer signal) was normalized to peak trial force.

The proximal electrode grid signals were used for MU source separation, whereas the distal ones were used to determine CV.

B. Signal Processing

The following signal analysis was performed with custom MatLab programs, using the Signal Processing and Statistic and Machine Learning toolboxes.

Force signals were corrected for the gravitational force and converted to Newton. The recording was segmented into separate trials and force onset detection was performed with the AGLR method (see the accompanying paper "Neuromechanical Control of Ballistic Contractions: Detection of Ballistic Action Onset").

The multi-channel surface EMG was decomposed with blind source separation [32], yielding the individual motor unit spike trains:

$$ST_m(t) = \begin{cases} 1, & if\ t \in T_m^{AP} \\ 0, & otherwise \end{cases} \tag{1}$$

where T_m^{AP} are the times of AP arrival to the m^{th} unit's motor end plate. High density EMG signals were spatially filtered by computing the double-differential. Root Mean Square (RMS), CV, Average Spike Density (ASD), RFD and the percentage of firing units were determined in 75 sample long intervals plus a 5 sample overlap ($L = 75$, $o = 5$), corresponding to ca. 40 ms. When comparing the above variables by superimposing plots and calculating correlations, the remaining variables (e.g. DR) were averaged over the same intervals. Furthermore, RFD, RMS, mean DR and CV were also determined as a function of force (in intervals corresponding to 10% increases in trial peak force).

In each discrete time interval i, RMS was averaged across all the double differential signals.

$$RMS_i^{dd} = \frac{1}{K}\sum_{k=1}^{K}\sqrt{\frac{1}{L}\sum_{n=Li-o+1}^{L(i+1)+o}|EMG_k^{dd}(n)|^2} \tag{2}$$

For each subject, the double differential signals were visually inspected. The observer selected four to six consecutive channels, with clear AP propagation in the same direction from the innervation zone and the least changes of shape and scale. For each subject, the same channels were used in conduction velocity computation of each (discrete) time interval in all trials. After a first estimation of the signal delay θ between multiple detection points in consecutive channels, using cross-correlation, θ was approximated with Maximum Likelihood, using Newton's criteria [33], and CV was determined from:

$$CV = \frac{d_G \times f_{samp}}{\theta} \tag{3}$$

C. Discharge Rate

Average Spike Density: When estimating variables over 75 sample windows, ASD is determined in each discrete time interval w by counting the number of decoded AP's among all neurons and dividing it by both the interval length and the number of decoded motor neurons M:

$$ASD = \frac{f_s \times \sum_{m=1}^{M}\sum_{t=w_i}^{w_f} ST_m(t)}{M(w_f - w_i)} \tag{4}$$

where w_i and w_f are the first and last indexes of the time window, f_s is the sampling frequency and $ST_m(t)$ is given by equation 1. While ASD is a commonly employed measure of neural activity, it requires a relatively large time resolution, given the sparseness of action potentials with respect to the sampling time-scale.

Instantaneous Motor Neuron Firing Rate: As the measurement intervals become smaller, ASD becomes less accurate. We employ an alternative method for firing frequency estimation, which approximates the real instantaneous discharge rate of each motor unit $\mathrm{DR_m}$. Let us consider that the time difference between any two consecutive spikes of the $\mathrm{m^{th}}$ motor neuron, i.e at times $T_m^{AP}(n)$ and $T_m^{AP}(n+1)$ (see Section II-B) is the instantaneous firing period. Then, since firing frequency is the inverse of firing period:

$$DR_m = \frac{f_s}{T_m^{AP}(n+1) - T_m^{AP}(n)}, \quad T_m^{AP}(n) \leq t < T_m^{AP}(n+1) \tag{5}$$

The total discharge rate (DR) is the average over all motor unit instantaneous discharge rates. This measure allows overall DR to be estimated with the same time-resolution as the sampling frequency, which in turn allows determination of Short Term Synchronization and Firing to Performance Phase (FPP, see Section II-D). When comparing DR to variables measured in larger time-scales (e.g. EMG amplitude and conduction velocity), the total DR is averaged through time in the same intervals.

Recruitment: Recruitment order is determined from the rank of the time of each unit's first spike. At each time interval, recruitment is quantified as the proportion of Active Motor Units (AMU): the percentage of units whose neurons are activated (e.g. transmit an AP) within the time window.

D. Short Term Synchronization

The level of synchronization is assessed from the statistics of the coefficients of correlation between the instantaneous discharge rate profiles $\mathrm{DR_m}$ of all motor units [34].

Firing to Performance Phase: In each trial, two measures of neuro-mechanical lag are determined: both at the onset and within the contraction. EMD is the difference between the onsets of force and EMG activity. FPP is the time-shift that maximizes the cross correlation between the instantaneous total DR and the force transducer signal.

E. Statistical Analysis

To evaluate the linear relationships between different factors across time, the correlation coefficient between variables was obtained with Pearson Statistics. The correlations among discharge rates or between discharge rate and force were determined from the profiles at the sampling rate (Table I). The correlations between motor output, neuro-muscular activity variables and EMG amplitude were determined over the full length of contraction, with the profiles determined in 75 sample intervals, and averaged over all subjects. The data were pooled and plotted as a function of time (Figures 3 and 4). The natural logarithms of the data in Figure 4 (up to 650 ms after tension onset) were fitted to a multiple linear regression model, with CV, DR and recruitment as predictor variables and force as the dependent variable. All variables were re-scaled, by subtracting the value at the onset (and then normalized) before determining their natural logarithm. The multiple linear regression was generated with the Statistics and MatLab Machine Learning Toolbox. Results are presented as mean $\pm$ standard deviation.

III. Results

A. Recruitment

In total, 188 motor units were decoded. In general, recruitment and rate coding obtained with high density sEMG decomposition were in agreement with the invasive studies on TA in ballistic contractions [4]. We observe the same discharge burst described in previous research [4], with full recruitment generally being completed before 21% of the peak force is reached, after which the motor units remained active while firing rate decreased noticeably (Figure 2). The proportion of motor units firing before force onset was less than the one suggested in the literature [4], which is coherent with the use of a more accurate force detection method in the present study.

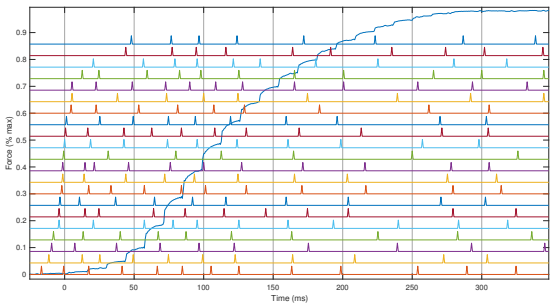

Fig. 1: Decoded motor unit spike trains and motor output during ballistic contractions.

B. Discharge Rate

Besides being recruited within a short period and before the gross of force development, the instantaneous firing rates of different motor units are highly correlated throughout the whole contraction (e.g. 90% of the inter-unit correlations were between 0.6 and 0.95 in subject 12). Individual neural profiles are strongly correlated with each other. When aligned to eliminate FPP, the electro-mechanical time-offset within the contraction period determined from cross correlation, both the averaged instantaneous discharge rate and the discharge rate from individual units were highly correlated with total force (Figure 2). This feature of the derived variables denotes the reliability of the methods employed, validating hdEMG decomposition as an instrument for neural control assessment during ballistic contractions. When comparing individual MUs with motor output, the mode of the distribution of correlation coefficients was above 0.85. Additionally, there was no correlation between ASD and motor output (Table III). When averaging force and total DR profiles, either across trials or over time windows, small fluctuations during the plateau phase

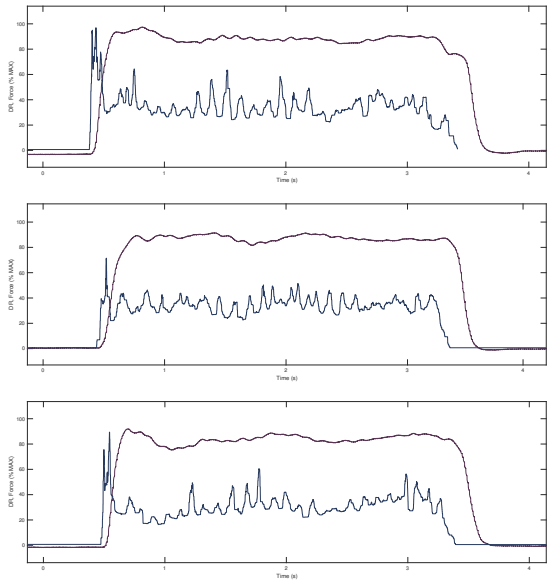

Fig. 2: Normalized profiles of force and instantaneous discharge rate (mean over motor units) in step-and-hold trials.

are eliminated and there is no significant correlation between motor output and DR (Table III).

TABLE I: Correlation between measures of neural activity and motor output.

	Correlation	
Subject	Mean DR	MU DR
1	0.75 ± 0.05	0.70 ± 0.09
2	0.77 ± 0.05	0.71 ± 0.09
3	0.80 ± 0.05	0.80 ± 0.05
4	0.82 ± 0.06	0.72 ± 0.11
5	0.88 ± 0.01	0.81 ± 0.10
6	0.89 ± 0.02	0.80 ± 0.11
7	0.88 ± 0.02	0.82 ± 0.06
8	0.74 ± 0.00	0.63 ± 0.11
9	0.82 ± 0.04	0.72 ± 0.11
10	0.75 ± 0.21	0.66 ± 0.21
11	0.64 ± 0.24	0.54 ± 0.21
12	0.73 ± 0.41	0.70 ± 0.35
13	0.76 ± 0.21	0.74 ± 0.19
14	0.66 ± 0.10	0.52 ± 0.22
15	0.78 ± 0.09	0.62 ± 0.18
Mean	0.78 ± 0.07	$0{,}70 \pm 0{,}09$

Pearson correlation coefficient between DR (average and individual MU) and strain gauge signal at the sampling frequency (2048 Hz) resolution.

C. Conduction Velocity

Conduction velocity was collectively analyzed for the twelve subjects with the best quality CV measurements in ca. 40 ms intervals. The average MFCV increased from 3.5 to 4.5 m/s over ballistic contractions, which is within the range of velocities in elicited TA twitch contractions reported by Andreassen et al. [12] . The change in conduction velocity in a 400 ms long contraction is consistent with the measurements in submaximal contractions of the vastus lateralis [35] and with slow ramp contractions of the TA up to 80% MVC [22]; the

TABLE II: Neural delays: EMD and FPP.

	EMD	FPP
Subject	(ms)	(ms)
1	55.05 ± 2.59	103.76 ± 5.00
2	55.34 ± 11.78	78.21 ± 6.47
3	48.44 ± 14.59	54.59 ± 30.76
4	51.35 ± 10.51	77.96 ± 6.01
5	38.49 ± 19.31	55.91 ± 24.85
6	42.97 ± 13.60	89.01 ± 9.47
7	48.58 ± 5.83	88.79 ± 3.92
8	71.78 ± 0.00	51.27 ± 0.00
9	46.55 ± 2.94	55.01 ± 8.60
10	38.09 ± 7.92	63.31 ± 33.91
11	58.76 ± 14.32	38.25 ± 28.63
12	42.24 ± 7.96	72.75 ± 28.42
13	55.80 ± 7.99	81.89 ± 29.41
14	59.49 ± 9.97	94.73 ± 49.63
15	51.62 ± 15.30	92.35 ± 25.22
Mean	50.97 ± 8.94	73.19 ± 19.19

EMD – absolute difference between EMG onset and motor output onset; phase between DR and motor output profiles, determined from cross-correlation.

average CV is consistent with the rise in conduction velocity from 3 to 4 m/s as full recruitment occurs [22], and a standard deviation under 0.5 m/s is agreement with the accuracy of the Maximum Likelihood method [33]. Assuming a mean TA

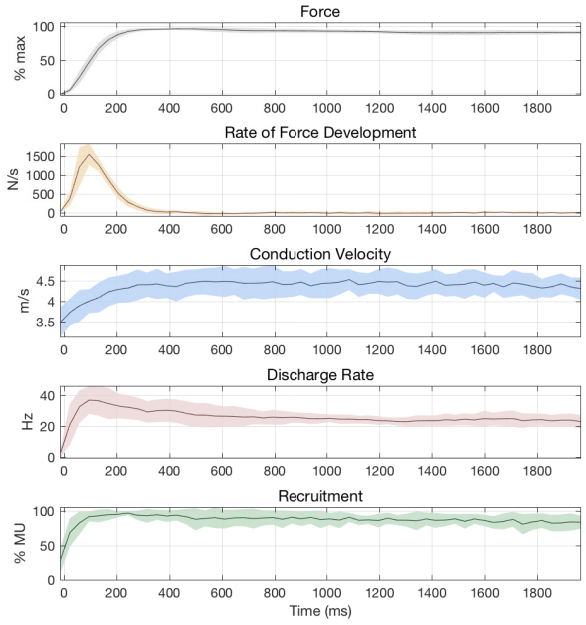

Fig. 3: Step-and-hold contraction: mean and standard deviation of force, recruitment (proportion of active motor units), discharge rate and conduction velocity determined in 40 ms windows, averaged over subjects.

fiber length of 70 mm [36], an action potential should take up to 20 ms (i.e. much less than the time resolution for CV) to travel from the motor end-plate to each end of the fiber at the beginning of contraction (i.e. when CV is minimal), up to 17.5 ms around the peak of DR, and up to 15 ms once force

is sustained (i.e. when CV is maximal). The rise time of mean CV was similar to the rise time of force. In the holding phase, the average CV was sustained at the maximal value through the force plateau (Figure 3).

Figure 3 depicts the average and standard deviation of force, RFD, CV, DR and recruitment. While the initial rise in CV is consistent with the rise in single unit DR and torque, once DR starts decreasing, both CV and torque continue rising, reaching a plateau at roughly the same time. CV was not consistently correlated with force, rate of force development and recruitment (Table III).

While Hedayatpour et al. [15] demonstrated a strong dependency between the conduction velocity of individual fibers and the average conduction velocity obtained from cross correlation of EMG signals, the influence of larger (and more conductive) fibers may be underestimated with this method.

D. Motor Control

The multiple linear regression yielded:

$$\ln(\mathrm{MO}) = 0.74\ln(\mathrm{DR}) + 0.32\ln(\mathrm{CV}) + 0.02\ln(\mathrm{AMU}) + 1.70$$
$$R^2 = 0.8, p = 0 \tag{6}$$

Where MO is the motor output (i.e. the measured force). Given that the $p - value$ is null and R^2 is close to one, there is a significant linear regression relationship between the logarithm of force and the logarithm of the peripheral and central control variables, implying a multiplicative relationship between the variables themselves. Furthermore, we found a strong correlation between the neural control variable DR and the double differential EMG signal (Table III).

TABLE III: Correlation between sEMG features, bioelectrical factors and motor output.

	Correlation
Force vs. CV	0.62 ± 0.28
Force vs. ASD	0.33 ± 0.31
Force vs. AMU	-0.08 ± 0.29
RMS vs. ASD	0.84 ± 0.11
RMS vs. DR	0.84 ± 0.06
RMS vs. AMU	0.61 ± 0.13
RMS vs. RFD	0.53 ± 0.15
RMS vs. CV	-0.02 ± 0.29
RFD vs. ASD	0.53 ± 0.19
RFD vs. DR	0.54 ± 0.22
RFD vs. AMU	0.19 ± 0.15
CV vs. ASD	0.02 ± 0.24
CV vs. DR	0.11 ± 0.27
CV vs. AMU	0.29 ± 0.32

Correlation between variables determined in 40 ms intervals.

IV. Discussion

A. Motor Unit Recruitment

As previously observed with invasive techniques [12], the ballistic neural discharge burst initiates before force onset (e.g. before visual and muscle spindle feedback can be processed), and the participating MUs are recruited early in the contraction. Throughout the force-holding phase (i.e. after the first half second of contraction) AMU remains steady (alike CV and DR) and close to its maximal value for 70% MVC, with no signs of fatigue (Figure 3). In average, full recruitment of the decoded MUs occurred between 1 and 30% of peak force, in all but one outlying subject (Table IV). The accuracy of the force onset detection method and the larger number of sampled units explain why these results differ from Desmedt et al. [4], who reported that full recruitment in ballistic contractions usually occurred before force detection.

The small differences in times of activation of different units corroborate a previous report from Dideriksen et al. [37] claiming that size-based ranking of motor unit recruitment does not yield a significant functional gain, and that its most relevant feature is the wide spectrum of innervation numbers.

B. Rate Coding

The average maximal discharge rate for single motor units was consistent with the range of 60 to 120 Hz indicated by Desmedt et al. [4] (except for a single outlying subject; Table IV), and the firing peak was followed by a drop in instantaneous DR to much lower values (e.g. less than 50 Hz) [6]. Noticeably, the overall instantaneous DR (i.e. average over all MU DRs) reached its maximum in under 40% of peak force, in all but three subjects. The force at full recruitment and force at maximal DR were less consistent than than the force at peak RFD, which was highly consistent across both trials and subjects, occurring in average at 32% of peak force. This is agreement with the strong dependency of ballistic RFD on intrinsic contractile properties, besides neural drive, reported by Folland et al. [2]. Furthermore, we observe a relatively long period of force increase following full recruitment and peak DR, which also agrees with previous findings of a greater relative contribution of the neural drive in the earlier phase of ballistic force production, and a more pronounced impact of non-neural factors in latter phases [2].

FPP (i.e. the estimated time lag between the overall discharge profile and the motor output) was generally higher than the initial EMD (Table II). FPP takes into account the delay during the force plateau phase, and is an estimate of the phase between force and instantaneous DR profiles, whereas EMD is determined from the initial difference between EMG and force onsets. Therefore, FPP reflects the *average* period between reception and execution of a particular neural instruction over the whole contraction, while EMD reflects the single period between reception of a neural signal and the production of a mensurable output. Besides the inherently different measuring methods, the mechanisms underlying the difference between the two delays likely include the rise time of fiber twitches and the time of AP propagation to the fiber ends. Besides excitability (e.g. CV), the contractile, viscoelastic and stiffness properties of the mucoleskeletal system may vary through different stages of contraction, and thus may also influence FPP.

Nearly all motor units were kept active throughout the force holding stage, during which the fluctuations in force follow the oscillations of the motor unit instantaneous firing rates, delayed by 50 to 100 ms (Table II, Figure 3). This similarity

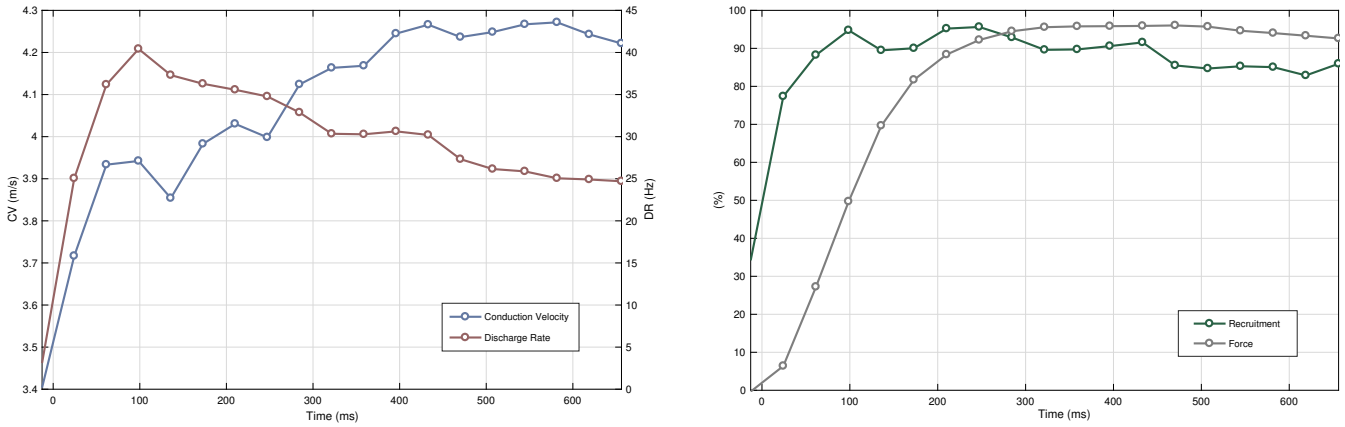

Fig. 4: The initial 650 ms of contraction: mean force, recruitment (proportion of active motor units), discharge rate and conduction velocity determined in 40 ms windows, averaged over subjects.

TABLE IV: Motor output and neural activity.

Subject	FFR (% max)	FP_{DR} (% max)	FP_{RFD} (% max)	$MUDR_{max}$ (Hz)
1	16.28 ± 16.17	46.44 ± 3.07	27.02 ± 2.38	114.12 ± 14.27
2	3.25 ± 2.36	34.88 ± 25.75	27.99 ± 1.92	89.06 ± 18.40
3	10.55 ± 7.70	11.26 ± 7.67	36.42 ± 2.55	81.85 ± 18.75
4	19.80 ± 24.18	13.22 ± 13.16	30.85 ± 2.87	57.67 ± 8.42
5	5.20 ± 4.90	4.13 ± 8.45	36.23 ± 4.97	98.98 ± 25.24
6	10.44 ± 11.47	3.21 ± 8.38	32.22 ± 5.53	48.04 ± 13.20
7	30.14 ± 8.10	34.28 ± 8.10	29.49 ± 4.04	65.18 ± 7.46
8	4.97 ± 0.00	57.07 ± 0.00	30.36 ± 0.00	124.72 ± 43.97
9	20.43 ± 17.77	45.66 ± 8.36	29.20 ± 1.66	77.74 ± 12.03
10	1.21 ± 1.02	7.94 ± 15.20	33.66 ± 2.57	92.09 ± 15.10
11	12.35 ± 10.28	22.78 ± 20.65	46.10 ± 0.75	89.32 ± 20.15
12	68.22 ± 18.21	38.26 ± 5.85	22.50 ± 3.39	38.72 ± 6.23
13	6.60 ± 3.97	26.19 ± 21.45	31.33 ± 3.75	82.71 ± 11.90
14	7.65 ± 9.54	6.22 ± 7.65	36.07 ± 7.32	88.57 ± 23.81
15	24.27 ± 31.27	19.90 ± 22.12	29.83 ± 2.75	80.35 ± 31.81
Mean	$16.09 \pm 16{:}64$	24.76 ± 17.29	31.95 ± 5.41	75.90 ± 29.84

FFR – force at full recruitment, percentage of trial peak force; FP_{DR} – force at peak instantaneous cumulative DR (mean DR across all motor units); FP_{RFD} – force at peak rate of force development; $MUDR_{max}$ – maximal motor unit discharge rate, mean across all individual instantaneous motor unit discharge rates, over all trials.

is a strong indicator of the decomposition accuracy. Given the high short term synchronization (Table I), these oscillations corroborate the finding of increased force fluctuations in simulations with motor unit synchronization [38].

The findings of strong correlation between the activities of different MUs and close times of recruitment suggest that the motor units active in a step-and-hold contraction are either under similar central commands (e.g. similar synaptic input to all motor neurons [34]) or prone to synchronization. There was no evidence of the existence of differentiated control strategies for neural sub-populations, which is consistent with the the literature presenting evidence of a continuous distribution of fiber characteristics, and suggesting that muscle units should not be classified into discrete types [39].

When comparing single MU DR to force rather than against each other, we found strong correlations, with mean R^2 equal to 0.85. The correlation between individual MU discharge rates and motor output was similar for all motor units except in punctual trials from three (Table I). This suggests that different units have somewhat similar roles in sustaining force at submaximal levels. The sum of the instantaneous DR over all units tended to have higher correlation with motor output than the average of single unit correlations (Table I). However, these correlations between neural drive and motor output are completely lost when using the more popular spike average density [10] or when averaging the values over time windows (Table III), as done for analysis of EMG RMS and CV. Therefore, the information lost when determining DR at a resolution much greater than the sampling period may be relevant to explain fine gradation of motor output, and, as such, the choice of method of *instantaneous* DR computation may be an important factor when studying fast and precise movements.

C. Conduction Velocity

CV rises monotonically for ca. 300 to 400 ms after force onset in sustained ballistic contractions. The increase in CV is concurrent with a strict increase in force, whereas DR and RFD have a non-monotonic behaviour in the same period, with mean DR decreasing to half of its peak value and

RFD returning to zero before stabilizing (Figure 3). In the initial contraction phase, the rise in CV is coherent with fast recruitment, activation of parallel fibers and increasing discharge rate as expected from the literature; however, CV and force continue rising for ca. 200 ms beyond the peak of total DR.

Despite being present in published data (e.g. Broman et al. [14] and Eberstein et al. [18]), the transient rising behaviour of CV at the onset of contraction is typically disregarded, since such studies are usually concerned with the variation of CV at much longer time scales (e.g. during fatiguing contractions). Here we discuss possible mechanisms for the rise in CV and its potential contribution towards ballistic contraction performance.

Conduction Velocity and Neural Control: A close relationship between force and CV matches Masuda and De Luca's data [22] on slow ramp contractions, especially when considering the last two seconds of the upward ramp (e.g. the contraction time scale employed in the present study). An often proposed mechanism driving the rise in CV is the activation of additional MUs as higher force levels are reached. According to the size principle, the recruited muscle units are sequentially larger and more conductive [22][15][1]. Consequently, recruitment may influence CV in a non-linear way, as different motor units do not have the same intrinsic CV. However, in ballistic contractions, the variation of CV cannot be entirely explained with the aforementioned rationale: in this case, all units are recruited within a very short time, after which CV continues rising for longer than the sum of EMD and the time of AP propagation through TA muscle fibers.

A careful analysis of Masuda and De Luca's [22] results reveals that the most pronounced increase in CV in slow ramp contractions tends to occur towards the latter phase of recruitment (e.g. after 50% MVC). On the one hand, this distinct rise in CV could be attributed to the partaking of large conductive neurons, which are recruited at later (i.e. high) thresholds in paced contractions, but closer to tension onset in ballistic contractions [4]. On the other hand, above a certain force level, the number of additionally recruited units is dramatically reduced, and thus "even though the high threshold units generate more tension, the contribution of recruitment to increases in voluntary force declines at higher force levels" (Milner-Brown et al. [5]). Since CV and force behave similarly, it is conceivable that the variation of CV at higher force levels may also be caused by factors besides additional unit recruitment. As reported here, the prolonged increase in CV, lasting much longer than the recruitment phase in ballistic contractions, supports this hypothesis.

While CV has also been reported to vary proportionally to DR [13], in the present study CV varied differently from DR and AMU (Figures 3 and 4). After the ballistic contractions, when the target force is sustained, at lower firing rates, all variables ultimately stabilize. The final period of rise in CV cannot be imputed to an increase in DR, since during this phase the mean instantaneous discharge rate is roughly halved. Moreover, the VRF based on computational models indicates that the positive dependency of CV on DR only holds in a limited interval of instantaneous frequencies, which does not include the firing rates observed in ballistic contractions. According to Fortune and Lowery's simulation [26], the linear relationship between DR and CV occurs for instantaneous firing rates up to ca. 20 Hz, above which CV is in a supra-normal region and does not vary proportionally to DR. In general, the units sampled in the present study fired within the supra-normal range during the explosive segment of contractions – hence the absence of correlation between conduction velocity and DR is consistent with the VRF simulation results [26]. These findings stress that the positive correlation between DR and CV does not hold indefinitely and may have limited application in fast natural movements. Remarkably, K^+ channel dynamics is key in Fortune and Lowery's model [26], which supports the critical role of time-dependent biochemical changes leading to the establishment of an optimal ionic environment for fast peripheral control.

Conduction Velocity and Membrane Potential: The time constants of biochemical reactions influence the rate of contraction within single muscle fibers. The time needed for an AP to propagate through a muscle fiber (up to 20 ms, see Section III-C) is much lower than the rise time of evoked twitches in TA fibers (between 47 and 80 ms [12]), indicating that the biochemical events occurring locally within the fibers, from the release of Ca^+ from the sarcoplasmic reticullum to the cross-bridge cycle, are determinant for the temporal progression of force. These biochemical alterations may carry on for considerable periods. For instance, the membrane potential can decrease for several minutes after muscle fibers are stimulated [40]. Given the dependency of CV on membrane potential, CV itself may also have an inherent rise time depending, for instance, on the time required for establishing chemical gradients across the sarcolemma, that would in turn enable quicker re-polarization of the muscle fiber and thus increase CV. The existence of a CV rise time cold go unnoticed in ramp contractions, particularly in Masuda and De Luca's set-up [22], where the choice of an extremely low RFD imposes slow recruitment. Specifically, the rise time of CV for the lastly recruited fibers could be greatly reduced in slow contractions, due to the prior activation of nearby fibers that is known to facilitate conduction [15] (likely by pre-establishing the optimal ionic gradient; see explanation below). The findings of the current study agree with a strong dependency of CV on transient chemical changes, resulting in a noticeable effect on its time-dependency at the scale of ballistic contractions.

Once the units are recruited, the *baseline* myocyte thermo-chemical environment (i.e. the ion concentrations and membrane channel state prior to arrival of an action potential) changes depending on factors such as the dynamics of ionic channels. The easily variable ionic gradients across the the sarcolemma determine the membrane potential and may thus influence CV. Conduction velocity may be maximized when an *optimal conducting environment* is established throughout the length of all recruited fibers, after which it is maintained

[1]Conversely, progressive fatigue of active units would contribute to decrease CV (or its rate of increase) in maximal/long contractions; however, fatigue is unlikely to play a relevant role in our study.

as long as the neurons keep firing (even though they may fire at a lower frequency than in the initial activation burst) and fatigue does not occur (Figure 3). In general, temporary changes in the peri- and intra-cellular envoronment could be caused by altered ion channel dynamics in both sarcolemma and T-tubules, temperature or pH, metabolite build up or water content in the muscle. During ballistic contractions, changes in channel dynamics and ion concentration could explain the final rise in CV.

When the K^+ gradient across the membrane increases, the membrane becomes hyperpolarized (i.e. the memebrane potential becomes more negative[2]). As a result, the repolarization time is reduced and CV increases [13]. Evidently, the gradient can be raised by increasing the intracellular ionic concentration and/or decreasing the extracellular concentration. For instance, a temperature rise may intensify the Na^+/K^+-pump activity and thus enhance the build up of K^+ inside the muscle fibers. Consequently, the extracellular K^+ concentration is reduced, and this effect can be propagated to the neighbouring fibers. Noticeably, the activity of Ca^{2+}-dependent-K^+-channels can also influence CV [26] and might be optimized with Ca^{2+} saturation, which can take up to 50 ms to be attained in ballistic contractions [2]. Conversely, a reduction of the interstitial space water content increases the extracellular K^+ concentration, with the opposite effect on CV[3]. Another proposed mechanism for the influence of temperature on CV is the increased activation of voltage-gated Na^+ channels, which would cause the AP amplitude to decrease. A smaller AP depolarization would result in a shorter innactivation time, as in the case of hyperpolarization, and thus increase CV [44].

Early activation of low threshold fibers may contribute to enhance the overall conductivity, in agreement with several published phenomena. Indeed, a *low activation* state leads to greater CV in subsequent contractions, when compared to a complete rest state [45]. Tonic activation might *prime* the chemical gradients of some units and thus enhance the overall CV at the onset of strong contractions – this has been suggested to be the cause of supranormal CV in subjects with fibromyalgia [45]. Since the fibers of different units are amalgamated [39][17] the extracellular chemical changes (initiated by low threshold units in gradual contractions) may spread to neighboring fibers that, when activated, may be closer to their *optimal conductive state*. Changes in the membrane environment may also contribute for the increase in conduction velocity that follows fatiguing contractions: this phenomenon is mostly attributed to muscle fiber swelling and hyperpolarization due to greater sodium pump activity [25].

CV and Muscle Tension: Force increases for a period longer than FPP after DR starts decreasing. From the conservative estimations of TA conduction time, the increase in conduction velocity leads to a reduction of about 2.5 ms (see Section III-C) in muscle fiber propagation time, between peak DR and peak CV. An ever faster propagation of APs from the motor end-plate through the fiber counteracts the reduction in the frequency of APs reaching the sarcomeres. As a result, the signal intensity at the ends of the fibers is not reduced as dramatically as the DR. The reduction in fiber propagation time is about half of the increase in mean firing period ($\Delta T_{DR} = 1/32 - 1/38\,Hz \approx 5\,ms$; Figure 3).

This observation suggests that the rise in CV counteracts the lowering of mean DR, mitigating the decrease in signal intensity within the end-effectors (i.e. muscle fibers). Moreover, the conduction velocity measurement from EMG may underestimate the actual individual fiber CV, especially for lastly recruited fibers in gradual contractions, which are the most conductive ones [15]. Consequently, the actual maximal CV is likely to be larger than the previous estimation, and thus CV alone may partly compensate for the drop in mean central drive at the later stage of ballistic contractions. The maintenance of the effective cellular signal contributes to explain the concurrent rise and holding of motor output.

Besides the proposed chemical-related time dependency of CV, additional changes in the viscoelastic state or the biochemical environment in the sarcoplasm (affecting contractility) could influence the variation in motor output that is not directly related to DR. For instance, Folland et al. [2] suggests that at around 100 ms the relative contribution of the mechanical state of the muscle-tendon unit out-weights neural drive. This is in agreement with the literature suggesting that, in the first 100 ms of contraction, the contractile properties are predominant, whereas the impact of the individual's maximal strength capacity is revealed later in the contraction [8]. Finally, in agreement with the analysis above, force is relatively stabilized once both central (e.g. recruitment and DR) and peripheral (e.g. CV) variables reach a steady state. From that point on, motor output fluctuations linearly reflect overall DR at a small time-resolution (Table I).

D. EMG Amplitude

The findings of the present study indicate a strong dependency of the EMG amplitude on total instantaneous DR, and no correlation with CV in submaximal step-and-hold contractions (Table III). Furthermore, despite all units initially recruited remaining active throughout the contraction, there are oscillations in AMU, which is also mildly correlated with RMS. The relationship between CV and EMG features has been described in the literature, especially in the context of muscle exhaustion. Eberstein and Beattie [18] correlated the rates of decline of CV and mean EMG frequency in the biceps braquii in isometric contractions held at 60 and 70% MVC. Although CV and EMG were strongly related over the course of long fatiguing contractions (e.g. 18 to 36 s), careful observation of the trial subset illustrated in their paper suggests the over the initial 4-8 seconds CV rises slightly (increasing in a range coherent with this study), and only then decreases linearly, along with EMG mean frequency, due

[2]Recall that the electrical equilibrium potential for a given ion is given by the Nernst Equation: $E = \frac{kT}{q} \ln\left(\frac{[\mathrm{out}]}{[\mathrm{in}]}\right)$ [41][42], where k is the Boltzmann constant, T is the temperature, q is the ion charge, $[\mathrm{out}]$ is the extra-membrane ion concentration and $[\mathrm{in}]$ is the intra-membrane ion concentration. E_{K^+} has a strong direct impact on membrane potential, which can be estimated from the Millman or the Goldman-Hodgkin-Katz voltage equations, weighted by the membrane's relative conductance to K^+ [43].

[3]Interestingly, an increase of intracellular water could lead to greater conductivity through fiber swelling despite the negative impact on membrane K^+ concentration.

to fatigue. This suggests that the contribution of CV towards EMG in non-exhausted states may be less relevant. A possible mechanism for the observed electrical changes in fatigued muscles is the accumulation of potassium ions in the extracellular space, leading to lower sarcolemma excitability (see Section IV-C) [46]. Similarly, studies in ischemic conditions propose K^+ [17] and lactate [25] accumulation as sources of reduced CV during fatigue.

E. Motor Output

Through most of the holding phase, when peak force is sustained at a submaximal plateau, recruitment, total discharge rate and conduction velocity remain at an approximately constant level, and at this stage the instantaneous fluctuations of motor output are proportional to fluctuations in DR (Table I and Figure 2). Rate coding is thus a neural control mechanism used in gradation of force at high (i.e. $> 70\%$ MVC) levels of force, which likely involve visual and haptic feedback (thus the phase between electrical and mechanical profiles becoming greater than EMD; Table II).

It is known that CV, DR and recruitment are moderately associated with force. However, the available literature is typically focused on measuring correlation, and fails to demonstrate strong linear relationships between control factors and motor output throughout the whole duration of ballistic contractions (e.g. across time); is some studies, the relationships between rough estimates of neural drive and contractile properties at different time-periods are interpreted in terms of changes in "relative contribution" of different factors across the contraction (e.g. Folland et al. [2]). Such analysis based on correlation inherently assumes the linearity and additivity of the contributions of central and peripheral factors towards motor output.

Since CV measures the continuous propagation of a discrete signal DR to the mechanical effectors, their cumulative effect on motor output should reflect their product rather than their sum. The present study introduces the investigation of multiplicative relationships between CV, DR and force. Its findings suggest that peripheral and central effects have a mensurable multiplicative effect on motor output. In order to quantify the combined effect of the different control variables over muscle tension in the time-domain, a multiple linear regression was employed on the logarithms of the different variables, up to 650 ms after force onset, resulting in a coefficient of determination $R^2 = 0.8$. The high value of R^2 indicates that ballistic force depends non-linearly on DR, AMU, as well as CV (e.g. a peripheral control factor that is not exclusively explained by neural strategies). This analysis serves as proof of principle for the recognition of their non-trivial dependencies, going beyond the linear relationships that are typically assessed in the literature [7].

V. Conclusions

The investigation here reported leads to new insights into the relationships between central and peripheral bioelectrical factors, e.g. neural activity and muscle fiber conduction velocity. To the author's knowledge, this has been the first attempt to investigate the evolution of conduction velocity and to analyze its impact on the motor output in combination with central control variables during sub-maximal fast contractions.

Conduction velocity and ballistic force were found to increase monotonically beyond the peak of DR, for longer than twice the largest estimates of neuromechanical delay (and hence longer than might be explained by the viscoelastic properties of muscle and connective tissue). The analysis here proposed shows that the dynamics of CV are in agreement with the transience of ionic gradient changes and may contribute to explain an extended rise in muscle fiber tension.

Besides regulating the excitability of the sarcolemma, and thus influencing CV, biochemical mechanisms may also contribute for prolonging tension production through direct action in the sarcomeres (e.g. enzyme activity or calcium build up). The rise in CV in the late phase of force development may counteract the concurrent decrease in discharge rate, mitigating its impact on the effective signal reaching the contractile structures. The experimental data were successfully fit to a tentative model of the motor response as a non-linear function of central and peripheral bioelectric factors (R^2=0.8), but further research has to be conducted in order to validate this relationship.

Future studies may investigate the biochemical agents (e.g. membrane channels, enzymes) that lead to a persistent increase in CV during ballistic contractions. Furthermore, although the filtering effect of passive tissue on the contractile tension is minimized in isometric contractions, in further investigations the magnitude of the the extrafascicular (e.g. connective) delaying factors should be evaluated (e.g. using electrical stimulation and an AR model of the biomechanical system similar to the one employed in the accompanying paper "Neuromechanical Control of Ballistic Contractions: Detection of Ballistic Action Onset").

The motor neuron activity decoded from non-invasive EMG matched the only existing report on spinal control of ballistic contractions[4], where motor neurons were invasively probed. However, the present protocol did not allow the study of ballistic thresholds. This may be assessed in future studies of ballistic contractions to varied target levels. Additional improvements to the protocol should include the identification of structural features of motor units, estimation of individual muscle fiber conduction velocity and further evaluation of the existence of differentiated firing patterns during fast contractions. EMG amplitude was highly correlated with DR and moderately related to AMU. Global EMG features may be useful to assess both central and peripheral control in biofeedback applications. Blind source separation of high density EMG should be adopted in future studies to continue unraveling the firing strategies that influence fast and accurate motor performance.

References

[1] N. A. Tillin, P. Jimenez-Reyes, M. T. G. Pain, and J. P. Folland, "Neuromuscular performance of explosive power athletes versus untrained individuals," *Medicine and Science in Sports and Exercise*, vol. 42, no. 4, pp. 781–790, 2010.

[2] J. P. Folland, M. W. Buckthorpe, and R. Hannah, "Human capacity for explosive force production: Neural and contractile determinants," *Scandinavian Journal of Medicine and Science in Sports*, vol. 24, pp. 894–906, 2014.

[3] R. Ema, M. Saito, S. Ohki, H. Takayama, Y. Yamada, and R. Akagi, "Association between rapid force production by the plantar flexors and balance performance in elderly men and women," *AGE*, vol. 38, no. 5, pp. 475–483, 2016.

[4] J. E. Desmedt and E. Godaux, "Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle," *The Journal of Physiology*, vol. 264, no. 3, pp. 673–693, 1977.

[5] H. Milner-Brown, R. Stein, and R. Yemm, "The orderly recruitment of human motor units during voluntary isometric contractions," *The Journal of Physiology*, vol. 230, no. 2, pp. 359–370, 1973.

[6] J. Duchateau and S. Baudry, "Maximal discharge rate of motor units determines the maximal rate of force development during ballistic contractions in human," *Frontiers in Human Neuroscience*, vol. 8, no. 4, pp. 9–11, 2014.

[7] N. A. Maffiuletti, P. Aagaard, A. J. Blazevich, J. Folland, N. Tillin, and J. Duchateau, "Rate of force development: physiological and methodological considerations," *European Journal of Applied Physiology*, vol. 116, no. 6, pp. 1091–1116, 2016.

[8] L. L. Andersen and P. Aagaard, "Influence of maximal muscle strength and intrinsic muscle contractile properties on contractile rate of force development," *European Journal of Applied Physiology*, vol. 96, no. 1, pp. 46–52, 2006.

[9] N. A. Tillin, M. T. G. Pain, and J. P. Folland, "Identification of contraction onset during explosive contractions. Response to Thompson et al. "Consistency of rapid muscle force characteristics: Influence of muscle contraction onset detection methodology"," *Journal of Electromyography and Kinesiology*, vol. 23, no. 4, pp. 991–994, 2013.

[10] A. Del Vecchio, F. Negro, F. Felici, and D. Farina, "Associations between Motor Unit Action Potential Parameters and Surface EMG Features," *Journal of Applied Physiology*, vol. 123, no. 4, pp. 835–843, 2017.

[11] A. Del Vecchio, F. Negro, F. Felici, and D. Farina, "Distribution of muscle fibre conduction velocity for representative samples of motor units in the full recruitment range of the tibialis anterior muscle," *Acta Physiologica*, vol. 222, no. 2, 2018.

[12] B. Y. S. Andreassen and L. Arendt-Nielsen, "Muscle fibre conduction velocity in motor units of the human anterior tibial muscle: a new size principle parameter," *The Journal of Physiology*, vol. 391, no. 1, pp. 561–571, 1987.

[13] S. R. Gray, G. De Vito, M. A. Nimmo, D. Farina, and R. A. Ferguson, "Skeletal muscle ATP turnover and muscle fiber conduction velocity are elevated at higher muscle temperatures during maximal power output development in humans," *American Journal of Physiology-Regulatory Integrative and Comparative Physiology*, vol. 290, no. 2, pp. 376–382, 2005.

[14] H. Broman, G. Bilotto, and C. J. De Luca, "Myoelectric signal conduction velocity and spectral parameters: influence of force and time," *Journal of Applied Physiology*, vol. 58, no. 5, pp. 1428–1437, 1985.

[15] N. Hedayatpour, L. Arendt-Nielsen, and D. Farina, "Motor unit conduction velocity during sustained contraction of the vastus medialis muscle," *Experimental Brain Research*, vol. 180, no. 3, pp. 509–516, 2007.

[16] D. Farina, L. Arendt-Nielsen, R. Merletti, and T. Graven-Nielsen, "Assessment of single motor unit conduction velocity during sustained contractions of the tibialis anterior muscle with advanced spike triggered averaging," *Journal of Neuroscience Methods*, vol. 115, no. 1, pp. 1–12, 2002.

[17] M. Gazzoni, F. Camelia, and D. Farina, "Conduction velocity of quiscent muscle fibers decreases during sustained contraction," *Journal of Neurophysiology*, vol. 94, no. 1, pp. 387–394, 2005.

[18] A. Eberstein and B. Beattie, "Simultaneous measurement of muscle conduction velocity and EMG power spectrum changes during fatigue," *Muscle and Nerve*, vol. 8, no. 9, pp. 768–773, 1985.

[19] D. Farina, M. Gazzoni, and F. Camelia, "Low-threshold motor unit membrane properties vary with contraction intensity during sustained activation with surface EMG visual feedback," *Journal of Applied Physiology*, vol. 96, pp. 1505–1515, 2004.

[20] R. Merletti, M. Knaflitz, and C. J. De Luca, "Myoelectric manifestations of fatigue in voluntary and electrically elicited contractions," *Journal of Applied Physiology*, vol. 69, no. 5, pp. 1810–1820, 1990.

[21] J. Y. Hogrel, "Use of surface EMG for studying motor unit recruitment during isometric linear force ramp," *Journal of Electromyography and Kinesiology*, vol. 13, no. 5, pp. 417–423, 2003.

[22] T. Masuda and C. J. De Luca, "Recruitment threshold and muscle fiber conduction velocity of single motor units," *Journal of Electromyography and Kinesiology*, vol. 1, no. 2, pp. 116–123, 1991.

[23] D. Farina and D. Falla, "Effect of muscle-fiber velocity recovery function on motor unit action potential properties in voluntary contractions," *Muscle and Nerve*, vol. 37, no. 5, pp. 650–658, 2008.

[24] H. Nishizono, H. Kurata, and M. Miyashita, "Muscle fiber conduction velocity related to stimulation rate," *Electroencephalography and Clinical Neurophysiology*, vol. 72, no. 6, pp. 529–534, 1989.

[25] M. J. Zwarts, T. v. Weerden, and J. H. v. d. Hoeven, "Long-lasting supernormal conduction velocity after sustained maximal isometric contraction in human muscle.," *Muscle and Nerve*, vol. 16, no. 3, pp. 312–320, 1993.

[26] M. Fortune E. & Lowery, "Simulation of the Interaction Between Muscle Fiber Conduction Velocity and

Instantaneous Firing Rate," *Annals of Biomedical Engineering*, vol. 39, no. 1, pp. 96–109, 2011.

[27] M. Mihelin, J. V. Trontelj, and E. Stålberg, "Muscle fiber recovery functions studied with double pulse stimulation," *Muscle and Nerve*, vol. 14, no. 8, pp. 739–747, 1991.

[28] D. Farina, A. Holobar, R. Merletti, and R. M. Enoka, "Decoding the neural drive to muscles from the surface electromyogram," *Clinical Neurophysiology*, vol. 121, no. 10, pp. 1616–1623, 2010.

[29] D. Farina and A. Holobar, "Characterization of Human Motor Units From Surface EMG Decomposition," *Proceedings of the IEEE*, vol. 104, no. 2, pp. 353–373, 2016.

[30] L. L. Andersen, S. P. Magnusson, M. Nielsen, J. Haleem, K. Poulsen, and P. Aagaard, "Neuromuscular Activation in Conventional Therapeutic Exercises and Heavy Resistance Exercises: Implications for Rehabilitation," *Physical Therapy*, vol. 86, no. 5, pp. 683–697, 2006.

[31] R. M. Enoka and J. Duchateau, "Inappropriate interpretation of surface EMG signals and muscle fiber characteristics impedes understanding of the control of neuromuscular function," *Journal of Applied Physiology*, vol. 119, no. 12, pp. 1516–1518, 2015.

[32] F. Negro, S. Muceli, A. M. Castronovo, A. Holobar, and D. Farina, "Multi-channel intramuscular and surface EMG decomposition by convolutive blind source separation," *Journal of Neural Engineering*, vol. 13, no. 2, p. 17, 2016.

[33] D. Farina, W. Muhammad, E. Fortunato, O. Meste, R. Merletti, and E. Rix, "Estimation of single motor unit conduction velocity from surface electromyogram signals detected with linear electrode arrays," *Medical and Biological Engineering and Computing*, vol. 39, no. 2, pp. 225–236, 2001.

[34] D. Farina, F. Negro, S. Muceli, and R. M. Enoka, "Principles of Motor Unit Physiology Evolve With Advances in Technology," *Physiology*, vol. 31, no. 2, pp. 83–94, 2016.

[35] K. R. Mills, L. Arendt-Nielson, and A. Forster, "Changes in muscle fibre conduction velocity during prolonged submaximal contractions of human vastus lateralis," *Electroencephalography and Clinical Neurophysiology*, vol. 61, no. 3, p. 125, 1985.

[36] J. A. J. Friederich, "Muscle fiber architecture in the human lower limb," *Journal of Biomechanics*, vol. 23, no. 1, pp. 91–95, 1990.

[37] J. L. Dideriksen and D. Farina, "Motor unit recruitment by size does not provide functional advantages for motor performance," *The Journal of Physiology*, vol. 591, no. 24, pp. 6139–6156, 2013.

[38] W. Yao, A. J. Fuglevand, R. M. Enoka, A. J. Fuglevand, and R. M. Enoka, "Motor-unit Synchronization Increases EMG Amplitude and Decreases Force Steadiness of Simulated Contractions," *Journal of Neurophysiology*, vol. 83, no. 1, pp. 441–452, 2000.

[39] R. Merletti and D. Farina, *Surface Electromyography: Physiology, Engineering and Applications*, 1st ed., ser. IEEE Press Series on Biomedical Engineering. IEEE Press, Mar. 2016.

[40] C. Juel, "Potassium and sodium shifts during in vitro isometric muscle contraction, and the time course of the ion-gradient recovery," *Pflügers Archiv*, vol. 406, no. 5, pp. 458–463, 1986.

[41] P. Dayan and L. F. Abbott, *Theoretical Neuroscience : Computational and Mathematical Modeling of Neural Systems*. Cambridge, Mass.: MIT Press, 2014.

[42] G. Wulfram, W. M. Kistler, R. Naud, and L. Paninski, *Neuronal Dynamics: from Single Neurons to Networks and Models of Cognition*. Cambridge: Cambridge University Press, 2014.

[43] D. Junge, *Nerve and muscle excitation*, 3rd ed. Sunderland, Mass.: Sinauer Associates, 1992.

[44] S. Methenitis, N. Karandreas, K. Spengos, N. Zaras, A. N. Stasinaki, and G. Terzis, "Muscle Fiber Conduction Velocity, Muscle Fiber Composition, and Power Performance," *Medicine and Science in Sports and Exercise*, vol. 48, no. 9, pp. 1761–1771, 2016.

[45] E. G. Klaver-Krol, J. J. Rasker, N. R. Henriquez, W. G. Verheijen, and M. J. Zwarts, "Muscle fiber velocity and electromyographic signs of fatigue in fibromyalgia," *Muscle and Nerve*, vol. 46, no. 5, pp. 738–745, 2012.

[46] K. Mills and R. Edwards, "Muscle Fatigue in Myophosphorylase Deficiency: Power Spectral Analysis of the Electromyogram," *Electroencephalography and Clinical Neurophysiology*, vol. 57, no. 4, pp. 330–335, 1984.