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Decreased neural drive affects the early rate of force development after repeated burst-like isometric contractions

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Abstract

The neural drive to the muscle is the primary determinant of the rate of force development (RFD) in the first 50 ms of a rapid contraction. It is still unproven if repetitive rapid contractions specifically impair the net neural drive to the muscles. To isolate the fatiguing effect of contraction rapidity, 17 male adult volunteers performed 100 burst-like (i.e., brief force pulses) isometric contractions of the knee extensors. The response to electrically-evoked single and octet femoral nerve stimulation was measured with high-density surface electromyography (HD-sEMG) from the vastus lateralis and medialis muscles. Root mean square (RMS) of each channel of HD-sEMG was normalized to the corresponding M-wave peak-to-peak amplitude, while muscle fiber conduction velocity (MFCV) was normalized to M-wave conduction velocity to compensate for changes in sarcolemma properties. Voluntary RFD 0–50 ms decreased ($d = -0.56$, $p < 0.001$) while time to peak force ($d = 0.90$, $p < 0.001$) and time to RFD_{peak} increased ($d = 0.56$, $p = 0.034$). Relative RMS ($d = -1.10$, $p = 0.006$) and MFCV ($d = -0.53$, $p = 0.007$) also decreased in the first 50 ms of voluntary contractions. Evoked octet RFD 0–50 ms ($d = 0.60$, $p = 0.020$), M-wave amplitude ($d = 0.77$, $p = 0.009$) and conduction velocity ($d = 1.75$, $p < 0.001$) all increased. Neural efficacy, i.e., voluntary/octet force ratio, largely decreased ($d = -1.50$, $p < 0.001$). We isolated the fatiguing impact of contraction rapidity and found that the decrement in RFD, particularly when calculated in the first 50 ms of muscle contraction, can mainly be explained by a decrease in the net neural drive.

KEYWORDS

explosive contraction, muscle fiber conduction velocity, M-wave, neural drive, neuromuscular fatigue, octets

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1 | INTRODUCTION

Human locomotion is generated by a coordinated succession of impulsive burst-like excitation of muscle groups.[1] The muscle excitation profiles are brief (i.e., around 200 ms or less) Gaussian-shaped curves.[2–4] Evaluating ballistic burst-like contractions under isometric conditions is a viable strategy to mimic such muscle excitation in laboratory settings. The isometric protocols based on such burst-like contractions (i.e., without any holding phase) have been proven sensitive to neuromuscular training,[5] ageing,[6,7] neuromotor pathologies,[8] muscle asymmetries,[9] and fatigability.[10] However, that kind of contraction has never been adopted to induce fatigue under isometric conditions and study the underlying mechanisms.

The previous protocols that included rapid isometric contractions to induce fatigue were not based on short burst-like contractions but on longer (1–5 s) contractions.[11–14] Those contractions consist of an initial rapid force followed by a visual plateau phase where individuals reach their maximal voluntary force (MVF).[11–14] With the instruction to contract "as fast and as hard as possible" and to continue contracting for up to 5 s, these contractions are convenient because they allow the measurement of both rate of force development (RFD) and MVF. RFD, or yank,[15] is calculated as the time derivative of force and is obtained from the rising part of the force-time curve.[16] Muscle fatigability can thus be monitored during each contraction in terms of RFD and MVF decline.[17] The issue is that most effort put into these contractions focuses on achieving the maximal force (1–5 s), but only the first ≈200 ms are dedicated to quickly increasing force.[18] Therefore, the fatigue induced by those protocols could mostly depend on the steady maximal contraction rather than the rapid rising phase. The neuromuscular mechanisms underlying the rising and plateau phases are fundamentally different: broadly speaking early rising phase depends on motor unit recruitment and discharge rate, while the maximal plateau phase depends more on contractile characteristics.[19,20] Therefore, the fatiguing effect of contraction rapidity per se, beyond the influence of any plateau phase, is unknown.

In the present study, we adopted a set of 100 burst-like contractions with the knee extensors, without any plateau phase, to specifically address the fatiguing effect of contraction rapidity. Rapid muscle contractions are characterized by specific rapid motor unit recruitment and discharge rate.[21,22] When the motor unit firing frequency is high, i.e., in the first 50 ms from contraction onset,[21,22] the absolute force production is still relatively low. Therefore, reaching maximal forces is unnecessary to stress the mechanisms of contraction rapidity if at least 70%–80% of MVF is targeted.[19] Adopting burst-like contractions, we avoided any fatigue development due to maximal force production, thus reducing the influence of other factors such as blood flow occlusion or impairments in cross-bridge generation and excitation-contraction coupling.[23] To test evidence of contractile impairments, we adopted evoked octets (eight stimuli of the femoral nerve delivered at 300 Hz) as they are considered the first choice to evaluate the maximal evoked RFD since they are able to activate the whole muscle rapidly.[19,24] We mainly focused on early-phase RFD parameters because those are the most relevant to quantifying the rapid contractile capacities. To test any decrement in sarcolemma excitability,[25,26] we measured the M-wave amplitude and conduction velocity in response to single electrical stimuli using high-density surface electromyography (HD-sEMG). Furthermore, to obtain an indirect biomarker of motor unit recruitment, we measured the average muscle fiber conduction velocity (MFCV) during voluntary contractions as it can be used as MFCV is related to the dimensions of activated muscle fibers.[27,28] MFCV is a relevant physiological variable in this context also because it is related to strength loss due to muscle fatigability[29] and it has been proven to be related to RFD capacity.[27,30] Lastly, to test any reduction in neural drive to the muscle during the rapid force production, we calculated the so-called neural efficacy, which consists of the ratio between voluntary and octets-evoked RFD in the first 50 ms of contractions.[31,32] As the force production in the first 50 ms of contraction primarily depends on the neural drive to the muscles,[21,22] we hypothesized that repeated rapid contractions mostly induced a decrement in the net neural drive with the consequence of lowering the early phase RFD (i.e., the first 50 ms of force production).

2 | METHODS

2.1 | Participants

A convenient sample of 17 physically active healthy male adults (mean ± SD: 26 ± 2 years; 177 ± 10 cm; 74 ± 7 kg) was recruited for the study. None had any previous history of neuromuscular disorders. All the participants were informed about the testing procedure and provided written informed consent before they participated in this study. The study was approved by the Ethical Advisory Committee (University of Verona–approval no 13.R1/2021) and performed following the Helsinki Declaration except for registration in a database. Participants visited the laboratory once for ≈60 min. Participants were instructed to avoid strenuous exercise for 24 h and caffeine for 4 h before they visited the laboratory.

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2.2 | Experimental setup

2.2.1 | Force measurements

Participants were seated and firmly secured with a seat belt on a custom-made chair that allowed the assessment of isometric force for the right knee extensors. The participants' knee and hip were flexed at 90° from full extension. An ankle strap was placed 2 cm above the malleolus consistent with the strain gauge load cell (546QD- 220 kg; DSEurope, Milan, Italy) positioned perpendicular to the tibial alignment. To avoid pain and maintain stiffness a hard shin protector was placed between the thrust surface and the tibia.[32] Force and HD-sEMG signal were sampled at 2048 Hz with an external analog-to-digital (A/D) converter (Quattrocento; OT Bioelettronica, Turin, Italy). The force signal was displayed for visual feedback during the tests.

2.2.2 | High-density surface electromyography

Two bidimensional HD-sEMG matrices of 64 electrodes each (13 rows × 5 columns, 8 mm inter-electrode distance, gold-coated; model: GR08MM1305, OT Bioelettronica, Turin, Italy) were placed over the right limb. The first was placed over the vastus lateralis (VL) and the second over the vastus medialis (VM). The reference electrode (24 mm, model: CDE-S. OT Bioelettronica, Turin, Italy) was placed on the patella of the same limb, a strap ground electrode, dampened with water, was placed around the ankle. Before the array application, the skin was prepared to remove any body hair, slightly abraded with an abrasive paste, and finally cleaned with water.[33]

To ensure proper electrode-skin contact, the electrode cavities of the matrices were filled with 20–30 L of conductive paste (Spes-Medica, Battipaglia, Italy). The electrode arrays were fixed with an extensible dressing. The EMG signals were amplified (gain 150), sampled at 2048 Hz, bandpass filtered (20–450 Hz, Butterworth fourth order) and converted to digital data with a 16-bit A/D converter (Quattrocento; OT Bioelettronica, Turin, Italy). Signals, in single-differential configuration, were visualized during acquisition and then stored on a personal computer using OT BioLab+ software version 1.5.5.0 (OT Bioelettronica, Turin, Italy) for further analysis.

2.2.3 | Evoked contractions

The femoral nerve was electrically stimulated (via a constant-current, variable-voltage stimulator; DS7AH; Digitimer Ltd, Welwyn Garden City, UK) with square-wave pulses (0.2 ms in duration)[34] with maximal voltage of 400 V to elicit either singlet or octet contractions (eight pulses at 300 Hz triggered by an external train generator) to determine the maximal capacity of the muscle–tendon unit for rapid force production.[24] The anode (50 × 90 mm) was placed over the greater trochanter and the cathode (Ø = 32 mm) was placed within the femoral triangle, above the femoral nerve. During all stimulations, the experimenter pressed his hand on the anode to bring it closer to the cathode to obtain a better response to the stimulation.

The stimulation procedure consisted of a series of incremental (starting from 20 mA and increasing by 20 mA at each step) single stimuli delivered (≥15 s apart) until there was a plateau in the M-wave amplitude response, which was visually evaluated. The electrical current was then reduced and octet stimulation was delivered at progressive currents (≥15 s apart) until a plateau in peak octet force was reached. Real-time inspection of peak octet force confirmed a plateau with incremental stimulation. A representative example of evoked octet force is reported in Figure 1B in gray, and a representative example of HD-EMG response to single stimuli is reported in Figure 1C.

2.2.4 | Protocol

Following skin preparation, electrodes placement, and chair setting, the volunteers performed an isometric warm-up consisting of four contractions at 50%, four at ~75%, and one submaximal ~90% contraction of their perceived MVF. The last contraction was recorded to allow to set up real-time visual feedback on a computer screen and proceed with task familiarization. Familiarization continued until the participant was able to perform five consecutive purely explosive contractions without countermovement and/or holding phase (Figure 1A). After familiarization, participants received the ramp simulation procedures to obtain the maximal stimulus intensities for singlet and octet evoked contractions.

To assess MVF, two maximal voluntary contractions were performed interspersed by 2 min of rest. The participants were instructed to progressively increase their force production and to keep the maximal contraction for 5 s. Standardized verbal encouragement was provided to the participants during the execution of maximal contractions. Of note, no measure of RFD was performed during those contractions as they were only intended to measure MVF. After 1 min of rest, subjects received two single stimulations and two octets interspaced by 15 s.

The fatiguing protocol comprised 100 burst-like contractions, each lasting ≈200 ms (in terms of time-to-peak force) and separated by 2 s of rest with a visual rhythmic

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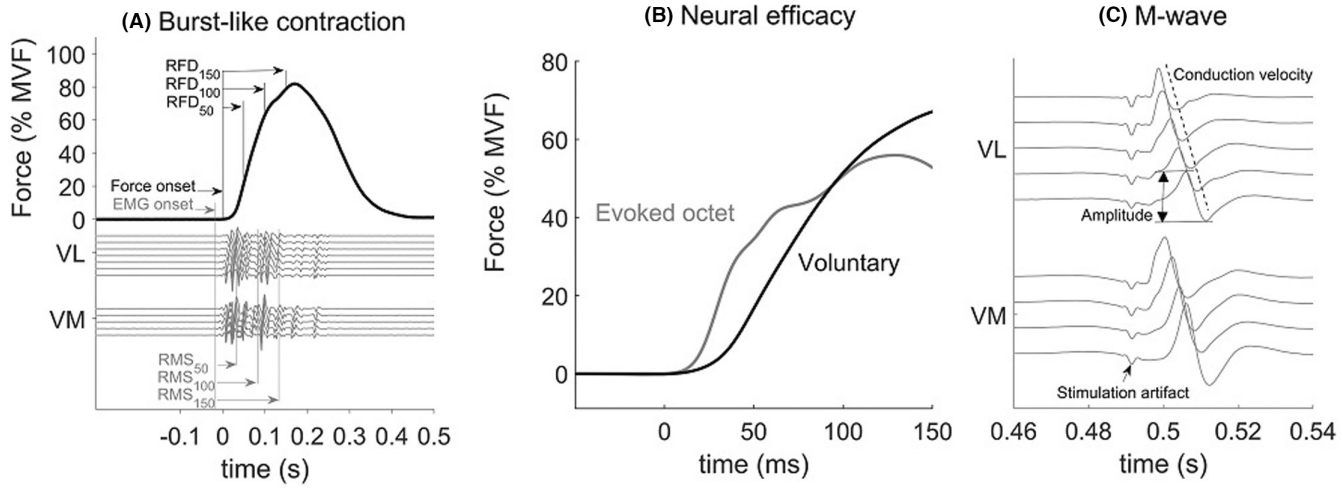

FIGURE 1 (A) Representative example of the force (black) and high-density surface electromyographic (HD-sEMG) signals (gray) recorded from one representative column of electrodes placed over the vastus lateralis (VL) and vastus medialis (VM) muscles during a burst-like contraction. The contraction is a brief pulse characterized by the fact that the active phase lasts ~200 ms and there is no holding phase. Rate of force (RFD) time-windows were calculated from force onset. EMG time-windows, which were adopted for root mean square (RMS) and muscle fiber conduction velocity (MFCV) estimates, were calculated from EMG onset. From HD-sEMG the average action potential velocity propagation was normalized to the M-wave propagation velocity (not shown here). (B) Representative example of the first 150 ms of voluntary burst-like (black) and electrically evoked octet (gray) force signals. The neural efficacy has been calculated as the ratio between the evoked and voluntary force at 50 ms from contraction onset. (C) Example of M-wave propagation recorded from one representative column of electrodes placed over VL and VM muscles. Peak-to-peak amplitude was calculated over all available electrodes and then averaged. CV was estimated with multichannel maximum-likelihood technique reported in Pozzo et al 2004.[35] MVF, maximal voluntary force.

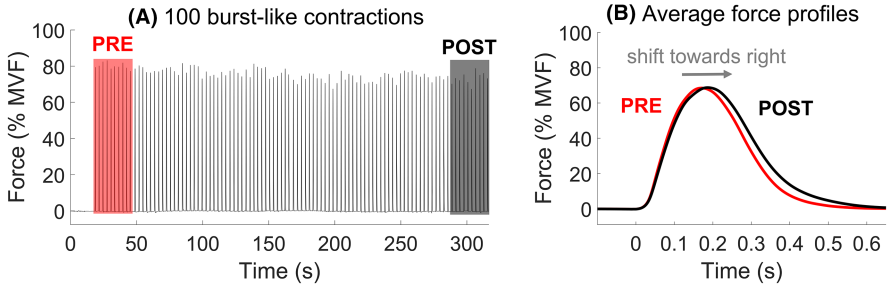

FIGURE 2 (A) Representative example of the protocol composed of 100 burst-like contractions. As can be seen, the peak force reached in each contraction was kept around 70%–80% of maximal voluntary force (MVF). (B) The figure represents the force signals of the average first 10 (PRE) and the average last 10 (POST) burst-like contractions averaged across 17 participants. It can be seen that the force-time curve is shifted towards the right as there was an overall slowing of the voluntary force production.

cadence feedback on a screen (Figure 2A). The participants were encouraged to "push fast and hard as possible",[36] to reach at least 70% (i.e., to overshoot the 70%) of their MVF, and to relax quickly after each pulse. When subjects did not reach 70% of MVF, verbal solicitations were provided. Throughout the protocol, the subjects were encouraged with strong verbal feedback to avoid loss of attention and to encourage to give their best in the "fastest and strongest" way possible and demanded to avoid countermovement or pre-tension. Immediately after the 100 contractions (which lasted 5 min), subjects received two octets and two single twitches (interspaced by 5 s) and concluded with a single 5-s maximal voluntary contraction.

2.3 | Data analysis

2.3.1 | Onset determination and time-windows

The same researcher visually selected the force and EMG onsets[37,38] through a hand-customized MATLAB code for voluntary and evoked contractions. Force and EMG signals were initially viewed on an *x*-axis scale of 300 ms before the contraction and *y*-axis scales of 2 N (force) or 0.05 mV (HD-EMG) before viewing signals on a more sensitive scale to determine the instant trough before the signal deflected away from the baseline noise. In case of

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contractions presented countermovement or pretension, they were removed from the analysis. On average, no more than one or two contractions were removed from voluntary contraction at each time point (i.e., PRE and POST) for each subject.

As can be seen in Figure 1A, the EMG time-windows were calculated from EMG onset, whilst force time-windows were calculated from force onset. Therefore, EMG and force time-windows (i.e., the windows of 50, 100, and 150 ms) were shifted by the time difference between EMG and force onsets. In our study, this difference, known as electromechanical delay, was, on average, ≈8 ms for evoked contractions and ≈18 ms for voluntary contractions.

2.3.2 | Force signals

The force signals were low-pass-filtered at 100 Hz using a fourth-order zero-lag Butterworth. MVF was the only variable measured from the 5-s maximal voluntary contractions performed before (PRE) and after (POST) the 100 burst-like contractions protocol. MVF was defined as the highest force over the two attempts performed at PRE and the single attempt performed at POST. All voluntary RFD parameters were calculated from the 100 burst-like contractions of the protocol. RFD (ΔForce/ΔTime) was estimated at 50, 100, and 150 ms (defined as RFD_{50}, RFD_{100}, and RFD_{150}). The maximum RFD (RFD_{peak}) and maximum rate of force relaxation (RFR_{peak}) were calculated, respectively, as the maximum and minimum first derivative of force signal from the onset of the contraction, adopting a moving average window of 20 ms.[18] Relative RFD, i.e., RFD/MVF were also calculated. The time to peak force and time to RFD_{peak} were also measured during burst-like voluntary contractions.

Evoked octets were only used to calculate force parameters, while evoked singlets were only used to calculate EMG responses, that is, M-wave parameters. Regarding octets, beyond peak octet force, we measured RFD_{50}, RFD_{peak}, relative RFD_{peak} (i.e., RFD_{peak}/peak octet force), and time to RFD_{peak}. Furthermore, we calculated the neural efficacy (%) as the voluntary RFD_{50} (estimated during burst-like contractions) divided by the octet RFD_{50}.

2.3.3 | High-density surface electromyography

EMG channels with excessive noise or artifacts were removed after visual analysis. Then, we identified the innervation zone for each matrix of electrodes and selected the channels with propagating action potentials. Single-differential EMG signals were calculated for each column and visually inspected. Six to eight single-differential EMG channels with clear motor unit action potential propagation without shape change from the nearest innervation zone to the distal tendon were chosen for the analysis.

M-wave peak-to-peak amplitude (mV) was calculated for each available channel as the response to singlet evoked stimulation (Figure 1C). The amplitude of voluntary HD-sEMG signals was assessed as the root mean square (RMS) across all available channels, divided by the corresponding M-wave peak-to-peak amplitude calculated over each channel. RMS calculated at 50, 100, and 150 ms from EMG onset (defined as RMS_{50}, RMS_{100}, and RMS_{150}) was then averaged across channels to obtain a single value for each muscle. This procedure has been proved to produce more reliable results in voluntary and evoked contractions.[39]

MFCV was assessed using an algorithm that allows the estimation of CV from multichannel EMG signals in burst-like contractions[35] as it provides reliable estimates in intervals as short as ~25 ms.[40] The algorithm provides maximum likelihood estimation of MFCV, minimizing the mean square error (in the frequency domain) between aligned signals.[35] Voluntary MFCV was estimated at 50, 100, and 150 ms from EMG onset (defined as $MFCV_{50}$, $MFCV_{100}$, and $MFCV_{150}$) for each column of electrodes and then averaged across channels. M-wave conduction velocity was calculated by adopting the same algorithm (Figure 1C). Finally, MFCV was also normalized to the M-wave conduction velocity (defined as $MFCV_{rel\_50}$, $MFCV_{rel\_100}$, and $MFCV_{rel\_150}$).

2.4 | Statistical analysis

The first (PRE) and the last (POST) 10 contractions were compared to analyse fatigability. To calculate the most stable indices, the 10 estimates of each parameter were averaged after having removed the highest and the lowest values.[16] The two repetitions of electrically evoked contractions were averaged. VL and VM HD-sEMG estimates were merged to provide an overall absolute quadriceps EMG measurement.[41] However, the results for separate muscles are reported in the supplementary material, and they did not show any significant difference between muscles.

Statistical analysis and descriptive graphs were performed in JASP (JASP Team 2023, version 0.17.2.1). Repeated measure ANOVA was performed to detect the changes in RFD, RMS, MFCV, and relative MFCV calculated over three-time windows (50, 100, and 150 ms) and two times (PRE, POST). Post hoc analysis were adjusted with Bonferroni corrections. Paired, two-tailed Student's *t*-tests were used to compare the other parameters between

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PRE vs POST. Kolmogorov–Smirnov normality test was used to assess distributions normality. The level of statistical significance was set to $p < 0.05$. Data are reported as mean ± standard deviation. The effect sizes in ANOVA analysis were reported as partial eta squared (η^2). The magnitude of the difference between PRE versus POST was calculated as Cohen's d effect size with 95% confidence interval (CI). Threshold values for effect size statistics were: <0.2, trivial; ≥0.2, small; >0.5, moderate; >0.8 large; >1.4, very large.

3 | RESULTS

The results of *t*-tests, post hoc analysis, descriptive statistics, and effect size differences with 95% CI between PRE and POST are reported in Table 1. The results are reported according to effect size interpretation. Figure 3 represents the PRE–POST individual values, box plot, and distribution of the most relevant parameters. The EMG results separated for VL and VM are reported in the supplementary material: see RMS results in Figure S1; MFCV results in Figure S2; relative MFCV results in Figure S3; M-wave results in Figure S4.

3.1 | Voluntary force

MVF moderately decreased ($d = -0.7$), nevertheless, the peak force reached burst-like contractions did not vary with time ($p = 0.753$), meaning participants maintained the capacity of reaching ≈77% of MVF measured at PRE.

The rapidity of force production decreased as evidenced by the large decrease in RFD_{peak} ($d = -0.88$), the large increase in time to peak force ($d = 0.90$), and the moderate increase in time to RFD_{peak} ($d = 0.56$), see Figure 2B. The cumulative RFD showed a time × interval interaction ($F [2, 32] = 9.7$, $p < 0.001$, $\eta^2 = 0.379$). Indeed, RFD_{50} showed a moderate decrement ($d = -0.56$), RFD_{100} showed a small decrement ($d = -0.41$), while RFD_{150} remained stable ($p = 0.226$). The cumulative relative RFD (i.e., RFD/MVF) showed a time × interval interaction ($F [2, 32] = 8.6$, $p = 0.001$, $\eta^2 = 0.350$). Indeed, relative RFD_{50} showed a moderate decrement ($d = -0.45$), relative RFD_{100} and RFD_{150} remained stable. The RFR moderately decreased ($d = -0.60$).

3.2 | EMG in voluntary contractions

Relative RMS decreased ($F [1, 16] = 20.0$, $p < 0.001$, $\eta^2 = 0.556$) without any time × interval interaction ($F [2, 32] = 1.5$, $p = 0.227$, $\eta^2 = 0.089$). In particular, RMS_{50}, RMS_{100}, and RMS_{150} decreased with similar very large effect sizes (d ranged from −1.2 to −1.4).

Absolute MFCV increased ($F [1, 16] = 21.1$, $p < 0.001$, $\eta^2 = 0.569$) without any time × interval interaction ($F [2, 32] = 1.0$, $p = 0.346$, $\eta^2 = 0.064$). In particular, $MFCV_{50}$, $MFCV_{100}$, and $MFCV_{150}$ increased with similar small or trivial effect sizes (d range from 0.16 to 0.21). Conversely, relative MFCV decreased ($F [1, 16] = 28.3$, $p < 0.001$, $\eta^2 = 0.639$) without any time × interval interaction ($F [2, 32] = 0.9$, $p = 0.397$, $\eta^2 = 0.056$). Indeed, $MFCV_{rel\_50}$, $MFCV_{rel\_100}$, and $MFCV_{rel\_150}$ decreased with similar moderate effect sizes (d range from −0.53 to −0.71).

3.3 | Evoked contractions

All quickness-related octet parameters increased after the protocol. Indeed, RFD_{50}, moderately increased ($d = 0.62$) and time to RFD_{peak} moderately decreased ($d = -0.96$). Furthermore, RFD_{peak} and relative RFD_{peak} increased with a large effect size (d range from 2.33 to 2.53). Neural efficacy largely decreased after the protocol ($d = -1.53$). Only octet peak force decreased with time with a large effect size ($d = -1.09$). Regarding singlet stimulation, M-wave CV ($d = 1.75$) and peak-to-peak amplitude ($d = 0.77$) both increased with time.

4 | DISCUSSION

To isolate the fatiguing effect of contraction rapidity, we adopted a protocol constituted by 100 burst-like contractions, i.e., brief force pulses without any plateau phase. We found an overall slowing in voluntary force production evidenced by a decrease in RFD_{peak}, and an increase in time to peak force and time to RFD_{peak}. Early RFD (0–50 ms) showed the largest decline in the time-locked analysis compared to late RFD (100 ms). The decline in voluntary RFD was likely driven by neural drive impairment, as suggested by the large decline in neural efficacy, which was accompanied by the increase in the quickness-related parameters of evoked octet force.

The nervous system can produce rapid muscle contraction by compressing the motor unit recruitment and abruptly increasing the discharge rate.[21] These mechanisms are associated with the neural drive to the muscle, which is the primary determinant of early RFD.[22] As the activation of the central nervous system is crucial in performing rapid contractions, it is possible to hypothesize that repeated rapid contractions would impair the net neural drive to the muscles. Previous studies failed to isolate the specific role of contraction rapidity on muscle fatigability because they adopted rapid muscle contractions

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TABLE 1 Descriptive statistics of parameters recorded during voluntary (burst-like and maximal voluntary contraction) and evoked (singlet and octet) contractions.

	Time window (ms) or peak	Pre	Post	Δ	*p*−value	Cohen's d test (95% CI)
Burst-like voluntary contractions						
Peak force (% MVF)	Peak	77.0 ± 9.8	77.3 ± 10.3	+0.4%	0.753	0.08 (−0.40, 0.53)
Time to peak force (ms)	Peak	178 ± 29	198 ± 33	+11.0%	0.002	0.90 (0.333, 1.46)
RFD_{peak} (N/s)	Peak	9684 ± 2062	8969 ± 1914	−7.4%	0.002	−0.89 (−1.44, −0.31)
Relative RFD_{peak} (MVF/s)	Peak	11.76 ± 1.71	11.61 ± 1.96	−1.3%	0.616	−0.12 (−0.59, 0.36)
Time to RFD_{peak} (ms)	Peak	55 ± 13	60 ± 21	+9.1%	0.034	0.56 (0.04, 1.07)
RFR (N/s)	Peak	6071 ± 1620	5541 ± 1726	−8.7%	0.025	−0.60 (−1.11, −0.08)
RFD (N/s)	50	3243 ± 1000	2664 ± 1071	−17.9%	<0.001	−0.57 (−1.02, −0.12)
	100	4688 ± 939	4270 ± 1171	−8.9%	0.002	−0.41 (−0.80, −0.03)
	150	3956 ± 911	3807 ± 990	−3.8%	0.266	−0.15 (−0.46, 0.17)
Relative RFD (MVF/s)	50	3.97 ± 1.29	3.49 ± 1.62	−12.1%	0.081	−0.46 (−0.10, −1.14)
	100	5.69 ± 0.62	5.48 ± 1.12	−3.7%	0.551	−0.19 (−0.32, 0.71)
	150	4.76 ± 0.43	4.84 ± 0.56	+1.7%	0.669	0.08 (−0.43, 0.58)
Relative RMS (% M-wave amplitude)	50	0.036 ± 0.013	0.027 ± 0.012	−25.0%	0.002	−1.13 (−2.24, −0.01)
	100	0.059 ± 0.009	0.045 ± 0.008	−23.7%	<0.001	−1.44 (−2.65, −0.22)
	150	0.059 ± 0.011	0.048 ± 0.009	−18.6%	0.001	−1.23 (−2.38, −0.09)
Relative MFCV (% M-wave CV)	50	1.053 ± 0.061	1.015 ± 0.063	−3.6%	0.012	−0.54 (−1.09, −0.02)
	100	1.097 ± 0.072	1.049 ± 0.070	−4.4%	<0.001	−0.68 (−1.28, −0.08)
	150	1.162 ± 0.09	1.110 ± 0.08	−4.5%	<0.001	−0.72 (−1.33, −0.10)
Absolute MFCV (m/s)	50	4.65 ± 0.62	4.80 ± 0.65	+3.2%	0.004	0.20 (0.00, 0.41)
	100	4.80 ± 0.73	4.96 ± 0.72	+3.3%	0.002	0.22 (0.01, 0.43)
	150	5.14 ± 0.85	5.26 ± 0.91	+2.3%	0.033	0.16 (0.35, 0.03)
5-s maximal voluntary contraction						
MVF (N)	Peak	831 ± 178	783 ± 168	−5.8%	0.005	−0.78 (−1.32, −0.23)
Single evoked contraction						
M-wave CV (m/s)	~	4.43 ± 0.63	4.75 ± 0.75	+7.2%	<0.001	1.75 (0.98, 2.48)
M-wave amplitude (mV)	~	4.01 ± 0.95	4.34 ± 1.03	+8.2%	0.009	0.78 (0.23, 1.31)
Octet evoked contraction						
RFD Octet (N/s)	50	5657 ± 1330	5916 ± 1413	+4.6%	0.020	0.63 (0.10, 1.14)
RFD_{peak} Octet (N/s)	Peak	12509 ± 2764	14788 ± 3120	+18%	<0.001	2.53 (3.51, 1.53)
Time to RFD_{peak} Octet (ms)	Peak	36 ± 12	26 ± 4	−27.8%	0.001	−0.96 (−1.53, −0.37)
Relative Octet RFD_{peak} (Octet peak force)	Peak	27.1 ± 7.2	33.5 ± 9.0	+23.6%	<0.001	2.33 (3.25, 1.39)
Octet peak force (N)	Peak	478 ± 123	458 ± 117	−4.2%	<0.001	−1.10 (−1.70, −0.48)
Neural efficacy	50	59.5 ± 21.8	46.5 ± 21.3	−21.8%	<0.001	−1.54 (−2.23, −0.81)

Abbreviations: CV, conduction velocity; MFCV, muscle fiber conduction velocity; MVF, maximal voluntary force; RFD, rate of force development; RFR rate of force relaxation; RMS, root mean square.

followed by a plateau phase to reach maximal force. To isolate the impact of rapidity, we required participants to perform burst-like contractions, i.e., brief and fast pulses (≈200 ms of active phase). As such, they never produced forces higher than 80% of MVF, which would have confounded the results by inducing peripheral changes typical of maximal force production.[42] Moreover, such motor task mimics, in isometric conditions, the brief muscle excitation profiles typically observed in locomotion.[2,3] We found a shifting of the force-time curve towards the right resulting in a wider Gaussian-shaped force curve (Figure 2B). Indeed, the time to peak force ($d = 0.90$) and the time to RFD_{peak} ($d = 0.56$) shifted towards the right, and those changes were accompanied by a large decrease

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in RFD_{peak} ($d = 0.88$). The slowing of voluntary force production due to repetitive rapid contractions can profoundly affect sports and work-related tasks.[15,43]

Performing 100 rapid contractions induced a very large decline ($d = 1.50$) in neural efficacy. Neural efficacy is the most specific parameter to quantify the ability to voluntarily activate muscles in the first 50 ms of contractions.[24] Its drop showed a decrease in the net neural drive to the muscles involved in a rapid task. The reduced net neural drive can be due to decreased motoneuron excitability, lowering in descending drive from the motor cortex or other supraspinal area, and increased presynaptic

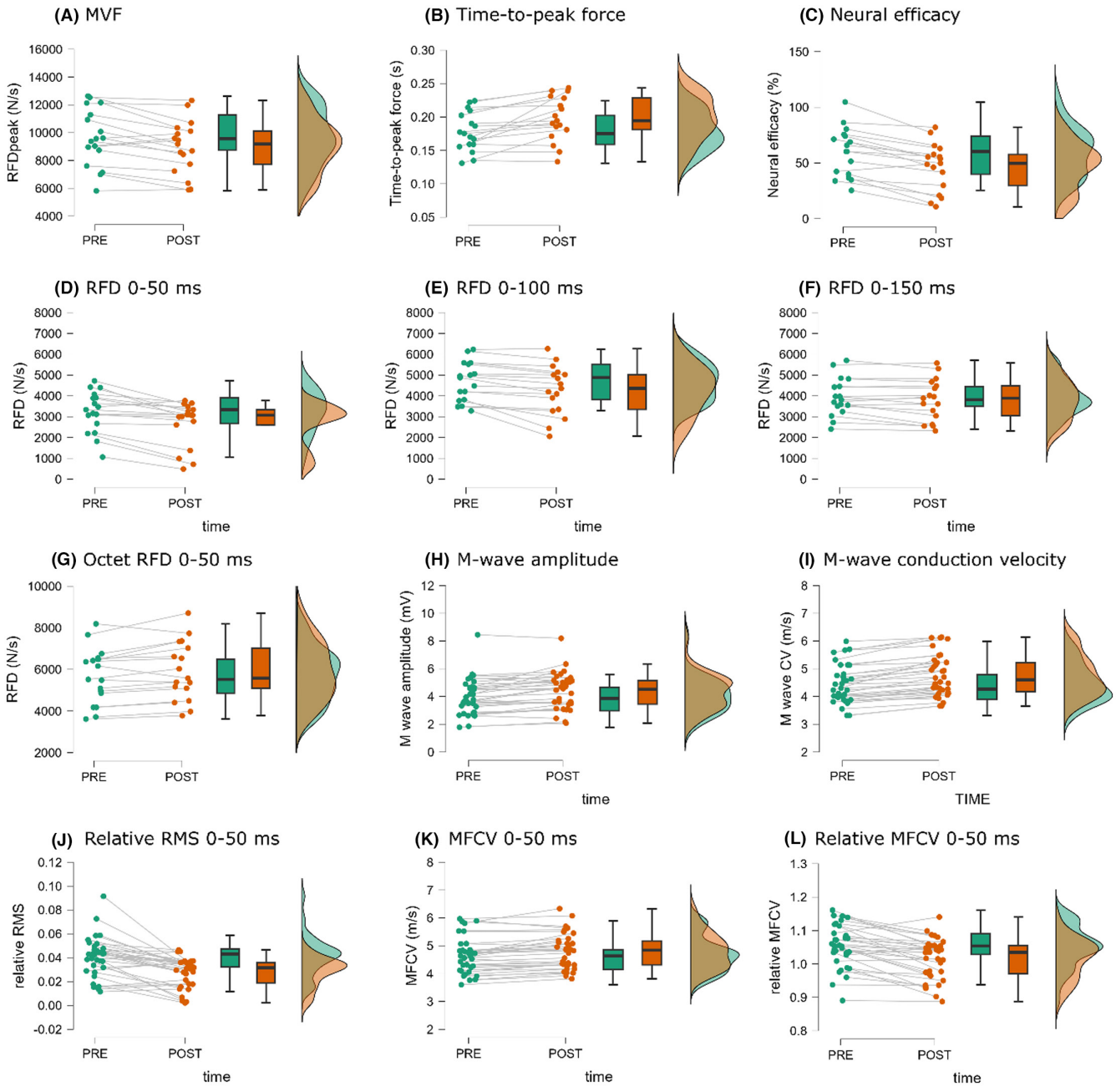

FIGURE 3 PRE–POST individual values, box plot and distribution of the most relevant parameters: (A) MVF, (B) time-to-peak force, (C) neural efficacy, (D) RFD_{50}, (E) RFD_{100}, (F) RFD_{150}, (G) Octet RFD_{50}, (H) M-wave amplitude, (I) M-wave conduction velocity, (J) relative RMS_{50} (normalized to M-wave amplitude), (K) muscle fiber conduction velocity (MFCV), and (L) relative MFCV (normalized to M-wave conduction velocity). For completeness, the results of electromyographic measures included the estimates of both the vastus lateralis and medialis muscles. The maximal voluntary force (MVF) was the only voluntary parameter measured during maximal voluntary contractions, the other voluntary parameters were measured during the first 10 (PRE) and last 10 (POST) burst-like contractions. Relative root mean square (RMS) was calculated as the RMS of each channel of HD-sEMG normalized to the corresponding M-wave peak-to-peak amplitude, while MFCV was normalized to M-wave conduction (relative MFCV). RFD, rate of force development.

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inhibition. In turn, spinal and supraspinal mechanisms have likely lowered the motor unit recruitment and discharge rate in the first phase of voluntary contractions. It is unsurprising that repetitive explosive contractions impair the neural drive as neural drive is the main determinant of rapid contractions.[20,21,44] Indirect evidence of decreased muscle excitation and motor unit recruitment occurrence could also be found in the significant drop of normalized RMS ($d \sim 1.0$) and normalized MFCV ($d \sim 0.6$) over the first 50–100 ms of contractions. To remove the effect of sarcolemma property changes, we normalized each RMS estimate to the corresponding M-wave peak-to-peak amplitude and the MFCV estimate by the M-wave CV. As such, those normalized RMS and MFCV estimates are robust indices as they are calculated over numerous electrodes and can be considered indirect markers of decreased motor unit firing or recruitment.

The findings regarding modifications in peripheral characteristics (i.e., mechanisms occurring distally from the neuromuscular junction on the neuromuscular pathway) were inconsistent but suggest that there was a preservation of sarcolemma membrane excitability and neuromuscular junction transmission (Table 1). Evoked octets showed an amelioration of all parameters related to quickness (RFD_{50}, RFD_{peak}, relative RFD, time to RFD_{peak}) but a decrease in peak octet force (Table 1). The speed-related measurements are crucial here as they demonstrate that the potential capacity to produce force quickly was preserved and even improved. Therefore, voluntary RFD drop could not be explained by contractile RFD drop, further reinforcing the hypothesis that the voluntary RFD drop was mainly driven by neural drive impairment. The other elements suggesting the maintenance, or even improvement, of peripheral properties were increased M-wave amplitude and conduction velocity. M-wave characteristics showed that the protocol did not deteriorate sarcolemma excitability. As mentioned above, peripheral fatigability is typically induced by prolonged high-intensity muscle contractions,[42] while for most of the contraction duration in the present protocol, the force production is not very high and is always lower than 80% of MVF. Also absolute estimates of voluntary MFCV increased after the protocol. This was not surprising as MFCV has already been demonstrated to increase after intermittent contractions in response to altered membrane properties, muscle fiber swelling, and temperature increase.[45] The increase in the above-mentioned parameters could be seen as signs of potentiation induced by repetitive high-intensity contractions. Possible explanations for the potentiating effect of combination of membrane hyperpolarization due to increased Na^+/K^+ pumping during activity[46] and muscle fiber swelling.[45] The only two parameters that showed a decrease after the protocol were: i) the peak octet force, suggesting a decrease in excitation-contraction coupling,[23] and ii) the voluntary RFR, suggesting an impairment in calcium handling in the sarcolemma.[47] However, those indices can only partially be related to the speed of the contraction during the rising force phase, which is the most relevant to explain voluntary RFD impairment.

4.1 | Limitation and perspectives

There are many ways to induce neuromuscular fatigue under isometric conditions. Here we proposed a protocol that can partially mimic the brief impulsive contractions typical of locomotion. The present approach can provide new insights on the specific role of contractions rapidity in the induction of muscle fatigability. In the present study we only recruited young male adults in good health, therefore, the findings may not apply to women and older subjects. Furthermore, we measured EMG only from VL and VM muscles. It should be considered that rectus femoris, which was not investigated, plays an essential role in knee extension force production. Furthermore, we did not evaluate the activity of the motor units through decomposition analysis. This is an approach that should be adopted to clarify better the neural mechanisms involved. Finally, future studies should consider muscle groups such as the plantar flexors because they have different morphological and biomechanical characteristics and are essential for locomotion.

5 | CONCLUSIONS

In the present study, we first isolated the fatiguing effect of contraction rapidity in isometric condition without the interference of prolonged maximal force production. Repeating 100 purely explosive contractions induces a slowing in the force production, i.e., shifts towards the right in the force-time curve. This slowing was more evident in the first 50 ms of contraction (early RFD) and was mainly due to decrease in the net neural drive. As the neural drive to the muscles is the primary determinant of early RFD, we can now clearly associate central fatigue induced by repetitive explosive contractions with their high neural drive requirements.

AUTHOR CONTRIBUTIONS

Gennaro Boccia, Samuel D'Emanuele, Paolo Riccardo Brustio conceived the study. Samuel D'Emanuele collected the data. Cantor Tarperi, Alberto Rainoldi, and Federico Schena supervised the data collection. Gennaro Boccia and Samuel D'Emanuele analyzed the data. Gennaro Boccia and Samuel D'Emanuele performed the statistical analysis. Samuel D'Emanuele and Gennaro Boccia wrote

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the draft and Federico Schena, Paolo Riccardo Brustio, Alberto Rainoldi, and Cantor Tarperi revised the draft. All Authors approved the final version of the manuscript.

CONFLICT OF INTEREST STATEMENT

No conflicts of interest, financial or otherwise, are declared by the authors.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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