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Original Article

Muscle fibre conduction velocity varies

in opposite directions after short- vs. long-duration muscle contractions

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Abstract

Introduction

The effects of muscle contractions on muscle fibre conduction velocity have normally been investigated for contractions of a given duration and intensity, with most studies being focused on the decline on conduction velocity during/after prolonged contractions. Herein, we perform a systematic analysis of the changes in conduction velocity after voluntary contractions of different durations and intensities.

AQ1

AQ2

Methods

Conduction velocity was estimated in the *vastus lateralis* before and after knee extensor isometric maximal voluntary contractions (MVCs) of 1, 3, 6, 10, 30 and 60 s, and after brief (3 s) contractions at 10, 30, 50, 70, and 90% of MVC force. Measurements were made during the 10-min period following each

contraction.

Results

(1) Conduction velocity was increased immediately after (1 s) the MVCs of brief (≤ 10 s) duration ($12 \pm 2\%$, $P < 0.05$), and then returned rapidly (within 15 s) to control levels; (2) the extent of the increase in conduction velocity was similar after the 3-s, 6-s, and 10-s MVCs ($P > 0.05$); (3) the magnitude of the increase in conduction velocity after a brief contraction augmented with the intensity of the contraction (increases of 4.6, 7.7, 11.4, 14.8, and 15.2% for contractions at 10, 30, 50, 70, and 90% of MVC force, respectively); (4) conduction velocity was not decreased immediately after the 30-s MVC ($P > 0.05$); and (5) conduction velocity did not reach its minimum 1 s after the long (≥ 30 s) MVCs.

Conclusions

Brief (≤ 10 s) muscle contractions induce a short-term increase in conduction velocity, lasting 15 s, while long (≥ 30 s) contractions produce a long-term decrease in conduction velocity, lasting more than 2 min.

AQ3

Keywords

M-wave
Femoral nerve stimulation
Conduction velocity
Muscle fibre diameter
Muscle shortening
Brief contractions

Abbreviations

Dur_{PP}	Peak-to-peak duration of the M-wave
Fmedian	Median frequency of the M-wave
EMG	Electromyographic
MVC	Maximal voluntary contraction

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Supplementary Information

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Introduction

Muscle fibre conduction velocity depends on the polarisation state of the sarcolemmal membrane, which is influenced by numerous factors, including the intracellular and extracellular Na^+ and K^+ concentrations (Juel 1988), membrane resistance and capacitance (Homma et al. 1983; Fortune and Lowery 2012), temperature (Schneider et al. 1988; Farina et al. 2005a), and blood supply (Farina et al. 2005b). In addition, conduction velocity is affected by the geometry of the muscle fibre, as fibres shorten in length during a contraction, and expands in thickness (Hakansson 1956). During a sustained forceful contraction, the slowing of impulse conduction has been mainly attributed to the accumulation of K^+ in the extracellular space (Juel 1988; Kössler et al. 1991), although other factors, such as increases in intracellular Na^+ concentration (Overgaard et al. 1997), inactivation of Na^+ channels (Fortune and Lowery 2009) and decreases in intracellular pH (Brody et al 1991; Schmitz et al 2012) have been also suggested. Therefore, extensive research has been conducted to unravel the mechanisms underlying the changes in conduction velocity during/after sustained fatiguing contractions. In contrast, the changes in conduction velocity after short-duration muscle contractions have received much less attention. However, even a brief muscle contraction alters the architectural properties of the muscle, such as fascicle length and pennation angle (Hodges et al. 2003), and thus could potentially influence conduction velocity. Therefore, it seems necessary to investigate the changes in conduction velocity after contractions of different durations and intensities.

When a maximal contraction is sustained for a long time (≥ 30 s), extracellular K^+ concentration increases greatly, reaching values as high as ~ 10–15 mM (Vyskocil et al. 1983; Saltin et al. 1987). After cessation of muscle activity, two main mechanisms, diffusion into the capillaries and Na^+–K^+ pumping, begin to restore gradually the extracellular K^+ concentration to resting levels (Juel 1986). Thus, after cessation of a long (≥ 30 s) sustained MVC, the above mechanisms

may take a few minutes to completely clear the accumulated K^+. More specifically, Vyskocil et al. (1983) showed that extracellular K^+ concentration remained elevated for 2 min after a 45-s MVC of the elbow flexors. Consistent with these observations on the extracellular K^+ recovery, it was found that conduction velocity remained depressed for about 3–4 min after a 1-min MVC of the same muscles (Van der Hoeven et al. 1993). Therefore, it appears that the time course of recovery of conduction velocity after a long sustained MVC could be associated to the restoration of extracellular K^+ concentration to resting levels.

In contrast to long contractions, conduction velocity may not decrease after a brief (≤ 10 s) MVC contraction. The reason is that, on one hand, at cessation of a brief MVC, the level of extracellular K^+ concentration is relatively low (Vyskocil et al. 1983), and thus it could only have a small decreasing effect on conduction velocity. On the other hand, alterations in architectural features of the muscle (fascicle length) could contribute to increase conduction velocity after a muscle contraction. Indeed, it is known that the length of muscle fascicles reduces during a sustained isometric contraction (Mademli and Arampatzis 2005). It might occur that this shortening persists for a few seconds after a brief MVC, as recently suggested (Rodriguez-Falces and Place 2017). If this is the case, then the shortened muscle fibres would show higher conduction velocities transiently after a brief MVC due to their increased fibre diameter (Hakansson 1956; Aidley, 1998). Therefore, after an MVC of short duration, the positive effect of increased fibre diameter on conduction velocity may be stronger than the negative effect due to accumulation of extracellular K^+.

AQ4

The objectives of the present study were: (1) to examine whether or not conduction velocity changes significantly after a brief muscle contraction and, if so, in which direction; (2) to compare the changes in conduction velocity after muscle contractions of brief (≤ 10 s) and long (≥ 30 s) duration. We discussed the possible involvement of various mechanisms in the changes in conduction velocity after a voluntary muscle contraction. Moreover, it was tentatively suggested that there are at least two groups of mechanisms acting in opposite directions on conduction velocity during/after a contraction: a model of the possible effects of these competing mechanisms has been proposed.

Materials and methods

Participants

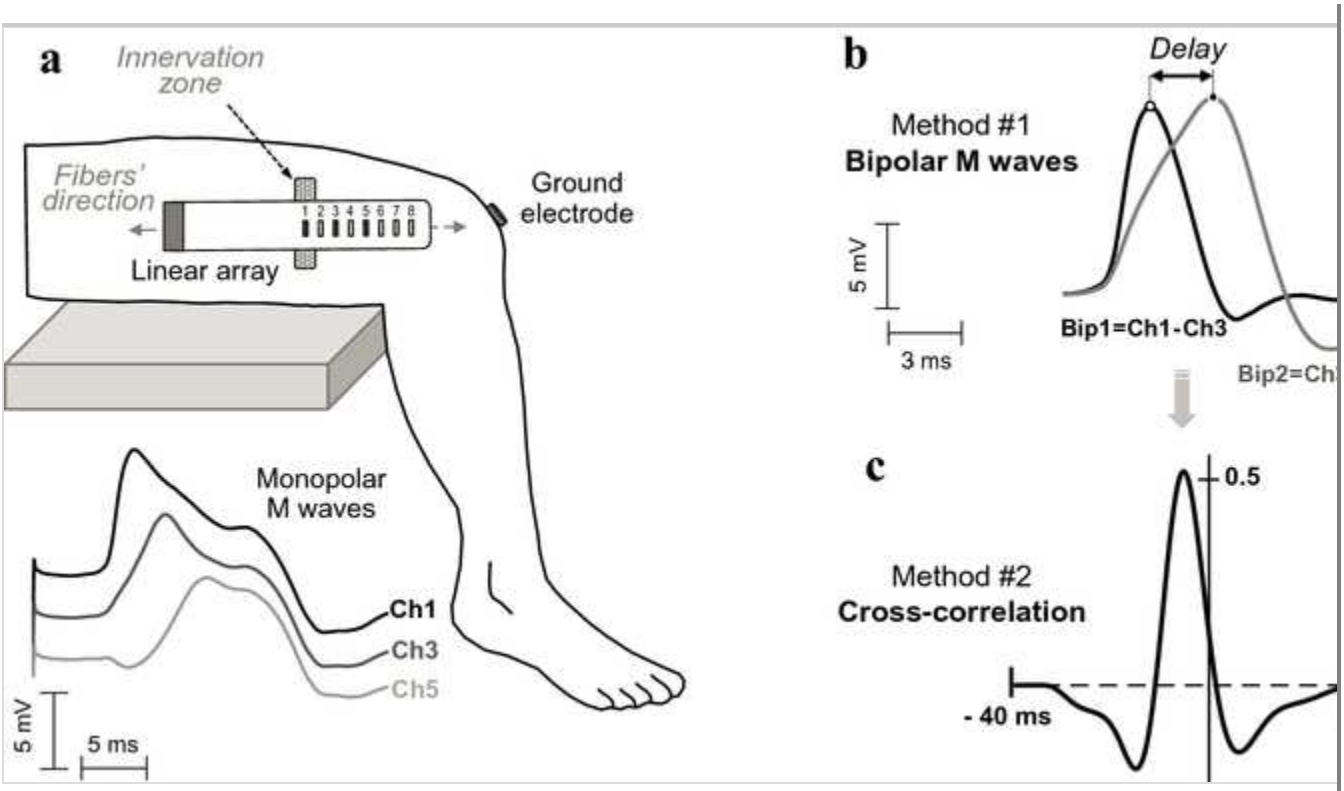
Fifteen healthy male participants aged between 22 and 30 years (mean ± SD: 25 ± 3 years) with no known history of neuromuscular or musculoskeletal disorders participated in this study. Their average height and body mass were 176 ± 5 cm and 72 ± 7 kg, respectively. Participants gave written informed consent before enrolment. The study was approved by the Ethics Committee of the Public University of Navarra, and all procedures conformed to the Declaration of Helsinki.

Experimental apparatus and mechanical recording

In the present experiments, participants performed isometric knee extension contractions of different durations and intensities. Participants were seated in an adjustable chair with the right ankle positioned in a custom apparatus that was tightly secured to the force transducer (Fig. 1). The knee angle was kept at 90º throughout the measurements, while the trunk-thigh angle was 100º. To restrain trunk and hip movements, two crossover shoulder harnesses were placed around the upper body and a belt was placed across the lower abdomen. The isometric force exerted by the *quadriceps* muscle was recorded by a strain-gauge transducer (STS, SWJ, China, linear range: 0–2452 N, sensitivity 2 mV/V and 0.0017 V/N). The signal from the force transducer was A/D sampled at 1000 Hz (MP150; BIOPAC, Goleta, CA, USA) and displayed on a monitor in front of the participant.

Fig. 1

a Experimental arrangements for the recording of the sEMG activity from the *vastus lateralis*. A linear array of 8 electrodes was oriented along the muscle fibres. The first most proximal electrode coincided with the innervation zone. Below, representative examples of monopolar M waves corresponding to the 1st, 3rd, and 5th channels are shown. In **b** and **c**, the schematic representation of the two methods for estimating muscle conduction velocity is shown. In the first method, **b**, the conduction delay was determined from the distance between the first peak of the bipolar M waves recorded at adjacent channels. In the second method, **c**, the conduction delay was determined from the lag of the peak of the cross-correlation function



Identification of the muscle fibres' direction and innervation zone

The direction of muscles fibres and the innervation zone of the *vastus lateralis* were identified using a dry linear array of 16 electrodes (5 mm inter-electrode distance) while participants performed mild isometric contractions. The 16 surface EMG signals were acquired in single-differential (bipolar) configuration using a multichannel amplifier (OT Bioelettronica, Torino; bandwidth 10–500 Hz). The direction of the muscle fibres was determined by noticing the orientation of the array that resulted in optimal propagation of the action potentials between the innervation zone and tendon regions (Farina et al. 2002). The position of the innervation zone was identified by observing the channel of the array showing minimum amplitude or phase reversal (Masuda et al. 1985).

Electromyographic recordings

Surface M-wave signals were obtained in response to femoral nerve stimulation applied before and after isometric conditioning contractions. The signals were recorded in monopolar derivation using linear adhesive electrode arrays (OT Bioelettronica, Torino, Italy) consisting of 8 electrodes separated by an inter-electrode distance of 5 mm. The array was placed so that its first, most proximal,

electrode was lying just above the innervation zone (Fig. 1), thus ensuring that unidirectional propagation of action potentials was detected. The array was oriented along the muscle fibres direction (see previous section). Reference electrodes were placed over the malleoli and patella of the dominant leg. To ensure appropriate electrode–skin contact, the electrode hollows of the array were filled with conductive paste. The surface EMG signals were amplified, sampled at 4096 Hz, band-pass filtered (3 dB bandwidth, 10–1000 Hz), and converted to digital data by a 12-bit A/D converter (EMG-USB, OT Bioelettronica, Torino, Italy). The surface EMG signals were stored in a personal computer using OT BioLab software (version 1.8, OT Bioelettronica, Torino, Italy) for further analysis.

Stimulation procedure

The femoral nerve was stimulated using single rectangular pulses (1-ms duration) delivered by a high-voltage constant current stimulator (DS7AH; Digitimer, Hertfordshire, UK). A circular (5-cm diameter) self-adhesive electrode (Dermatrode, American Imex, Irvine, CA) served as the cathode and was placed 3–5 cm below the inguinal ligament. The anode was located over the gluteal fold and consisted of a large (5 × 10 cm) rectangular self-adhesive electrode (Compex, Ecublens, Switzerland). Stimulus intensity was determined with the muscle fully relaxed by progressively increasing the current in 10 mA increments until a plateau in the M-wave amplitude of the *vastus lateralis* was observed. This intensity was then further increased by 20% to ensure that the stimulation remained supramaximal throughout the experiments (Rodriguez-Falces and Place 2016).

Experimental procedures

The experiments consisted of two parts performed sequentially in a single session in 1 day. In the first part, we investigated the changes in conduction velocity after contractions of varying intensities and the same (brief) duration. To this end, the participants were required to perform, in a random order, isometric contractions of 3 s at five different contraction levels: 10, 30, 50, 70, and 90% of MVC. One contraction per condition was performed. A resting period of at least 10 min was left between conditions (contractions). Before each conditioning contraction, three supramaximal single shocks (control responses), separated by 5-s intervals, were evoked while the muscle was at rest. Ten seconds after the last

control response, the conditioning contraction was performed and, subsequently, single electrical stimuli were delivered to the femoral nerve at 1 s, 5 s, 10 s, 15 s, 30 s, 1 min, 2 min, 5 min, and 10 min after the contraction. The duration of this session was ~ 60 min.

In the second part of the experiment, we examined the changes in conduction velocity after contractions of varying durations and the same (maximal) intensity. To do this, the participants were asked to perform conditioning MVCs of four different durations (1, 3, 6, and 10 s) in a randomized order. After these "brief" ($\leq$ 10 s) MVCs, participants also performed two long MVCs: the first of 30 s and the second of 60 s. A resting interval of at least 10 min was allowed between successive conditions (contractions). At the end of this resting period, we checked that the amplitude of the M-wave did not differ from the control values obtained at the beginning of the session. We found that M-wave amplitude remained unchanged throughout the experimental session (one-way ANOVA, $P = 0.67$). In addition, the MVC force was measured during a 1-s epoch around the peak force, and compared across the different maximal contractions. MVC forces did not differ (one-way ANOVA, $P = 0.56$), indicating that the 10-min resting interval was long enough to ensure complete recovery. Before each conditioning MVC, three supramaximal single shocks (control responses), with 5-s intervals in between, were evoked with the muscle fully relaxed. Subsequently, the conditioning MVC was performed, after which femoral nerve stimulation was applied at 1 s, 5 s, 10 s, 15 s, 30 s, 1 min, 2 min, 5 min, and 10 min after the contraction. The duration of this session was ~ 60 min.

Data analysis

To check the appropriateness of the recorded monopolar M waves (low traces in Fig. 1a), we verified that these signals fulfilled two shape requirements. First, the monopolar M-wave detected over the innervation zone (first electrode in the array) had the steepest rising phase, as demonstrated in Lateva et al. (1996). Second, the monopolar M-wave detected over the innervation zone exhibited a single well-defined positive peak, and this peak was well separated in time from the stationary "shoulder" component, as shown by Rodriguez-Falces and Place (2018). The methods employed to calculate the conduction velocity and the M-wave parameters were implemented using custom-written scripts implemented in Matlab (Mathworks, Natick, MA) and are described below. Conduction velocity was estimated from the bipolar M waves, and thus the monopolar

signals were first converted to single differential by software.

Two different methods were employed to estimate conduction velocity (Fig. 1). Both approaches consisted of detecting the M-wave at two different locations along the fibre direction and then measuring the time separation between these two bipolar signals. In the first method (Fig. 1b), the conduction delay was determined as the time separation between the peaks of the M-wave first phase, as described by Andreassen and Arendt-Nielsen (1987) and Christova et al. (1999). In the second method (Fig. 1c), the conduction delay was determined by first locating the maximum of the cross-correlation function of the bipolar M waves, and then measuring the time lag of this maximum, as proposed by Naeije and Zorn (1983) and Broman et al. (1985). Conduction velocity was estimated by dividing the spatial distance between the recording electrodes (see next sentence) by the time delay between the bipolar M waves as calculated above. In both methods, the bipolar M waves utilised for the calculation of conduction velocity were computed by subtracting channels 1 and 3 of the array (Bip1) and channels 3 and 5 of the array (Bip2). Thus, the inter-electrode distance was 10 mm. There are two reasons for choosing these channels. First, to ensure that the M waves exhibited clear signs of action potential propagation, a separation of at least 10 mm between electrodes was needed. Second, in the *vastus lateralis*, it has been found that the greatest changes in M-wave profile occurred along the 20 mm length starting from the centre of innervation zone in the distal direction (Rodriguez-Falces and Place 2018).

Changes in conduction velocity are reflected in alterations in the time and frequency parameters of the M-wave, such as its peak-to-peak duration and median frequency (Rodriguez-Falces et al. 2014). Therefore, the changes in these parameters after a contraction should be largely determined by (and thus be informative of) the changes in conduction velocity. The peak-to-peak duration (Dur_{PP}) was computed as the time interval between the first and second peaks of the M-wave, whereas the median frequency (Fmedian), defined as the frequency at which the EMG power spectrum is divided into two regions with equal amplitude, was computed as in Stulen and DeLuca (1981). These parameters were measured from the monopolar M-wave recorded over the innervation zone (first electrode, see Fig. 1).

Statistics

Kolmogorov–Smirnov tests confirmed that conduction velocity and all M-wave parameters calculated were normally distributed ($P > 0.05$). Differences in the control values of conduction velocity and M-wave parameters between the successive conditioning contractions were examined using a one-way repeated-measures ANOVA. The changes in muscle conduction velocity and M-wave parameters during the 10-min recovery following the conditioning MVCs of different durations were analysed with a two-way repeated-measures ANOVA (MVC duration × time after the contraction). The changes in muscle conduction velocity and M-wave parameters during the first 10 min following the conditioning contraction of different intensities were analysed with a two-way repeated-measures ANOVA (contraction intensity × time after the contraction). Differences between the estimates of conduction velocity performed by the cross-correlation method and the delay-between-the-peaks method were examined using a Student's paired *t* test. When main effects or interactions were significant, Tukey's post hoc tests for pairwise comparisons were applied. Significance was set at $P < 0.05$. Data were presented as mean ± SD in the text and tables and as mean ± SE in the figures.

Results

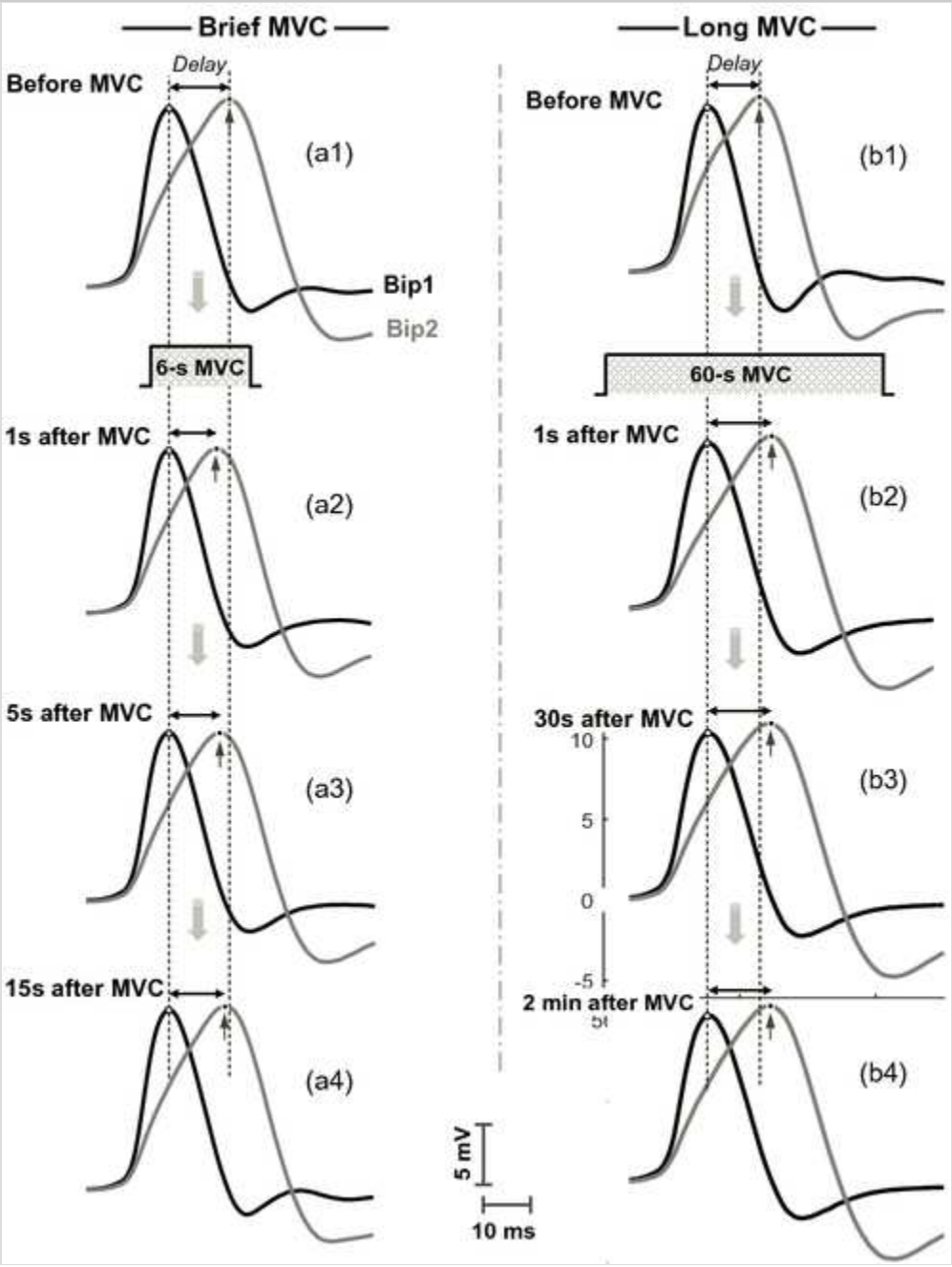
Representative M waves before and after maximal contractions of different durations

Figure 2 provides representative examples of pairs of bipolar M waves obtained before (control) and at various times after a brief MVC (6 s, left panel), and after a long MVC (60 s, right panel). As can be seen, the time separation between the positive peaks of the Bip1 and Bip2 M waves changed in a different manner after the 6-s MVC, as compared to after the 60-s MVC. Specifically, the delay between the peaks decreased immediately (1 s) after the 6-s MVC (Fig. 2a2), and subsequently this delay recovered quickly, returning to pre-contraction values within 15 s (Fig. 2a3, a4). In contrast, immediately after (1 s) the 60-s MVC, the delay between the peaks was lengthened (Fig. 2b2), and this delay remained increased even 2 min after the cessation of the contraction (Fig. 2b4).

Fig. 2

Representative examples of bipolar M waves obtained before (control) and at various times after a brief (left panel) and long (right panel) maximal voluntary contraction (MVC). The bipolar M waves (Bip1 and Bip2) were recorded by

electrodes placed along the fibre direction. Note that the delay between the positive peaks of the Bip1 and Bip2 M waves changed in a different manner after the 6-s MVC (left panel) than after the 60-s MVC (right panel). To better appreciate changes in the delay between the peaks, M waves were aligned by the positive peak of the Bip1 M-wave



Recovery of conduction velocity after maximal contractions of different durations

Figure 3 shows the changes in conduction velocity after conditioning MVCs of

different durations for the whole study group (calculations made with the delay-between-the-peaks method). It can be seen that conduction velocity increased immediately after (1 s) the MVCs of brief (≤ 10 s) duration ($P < 0.05$, Table 1), and this parameter returned very rapidly (within 15 s) to control levels. The extent of the increase in conduction velocity was similar after the 3-s, 6-s, and 10-s MVCs ($P > 0.05$), and also, the time course of recovery was similar after these contractions. The magnitude of the increase in conduction velocity after the 1-s MVC was smaller ($P < 0.05$) than that after the 3-s, 6-s, and 10-s MVCs.

Fig. 3

Time course of recovery of muscle fibre conduction velocity after conditioning maximal voluntary contractions (MVCs) of different durations. Estimations of conduction velocity were made using the delay-between-the-peak method (see text). All data are expressed in percentage of control values and reported as mean ± SE ($n = 15$). *Significant difference with control ($p < 0.05$). †Significant difference between 1-s, 3-s, 6-s, and 10-s MVC compared with the 30-s and 60-s MVCs ($p < 0.05$)

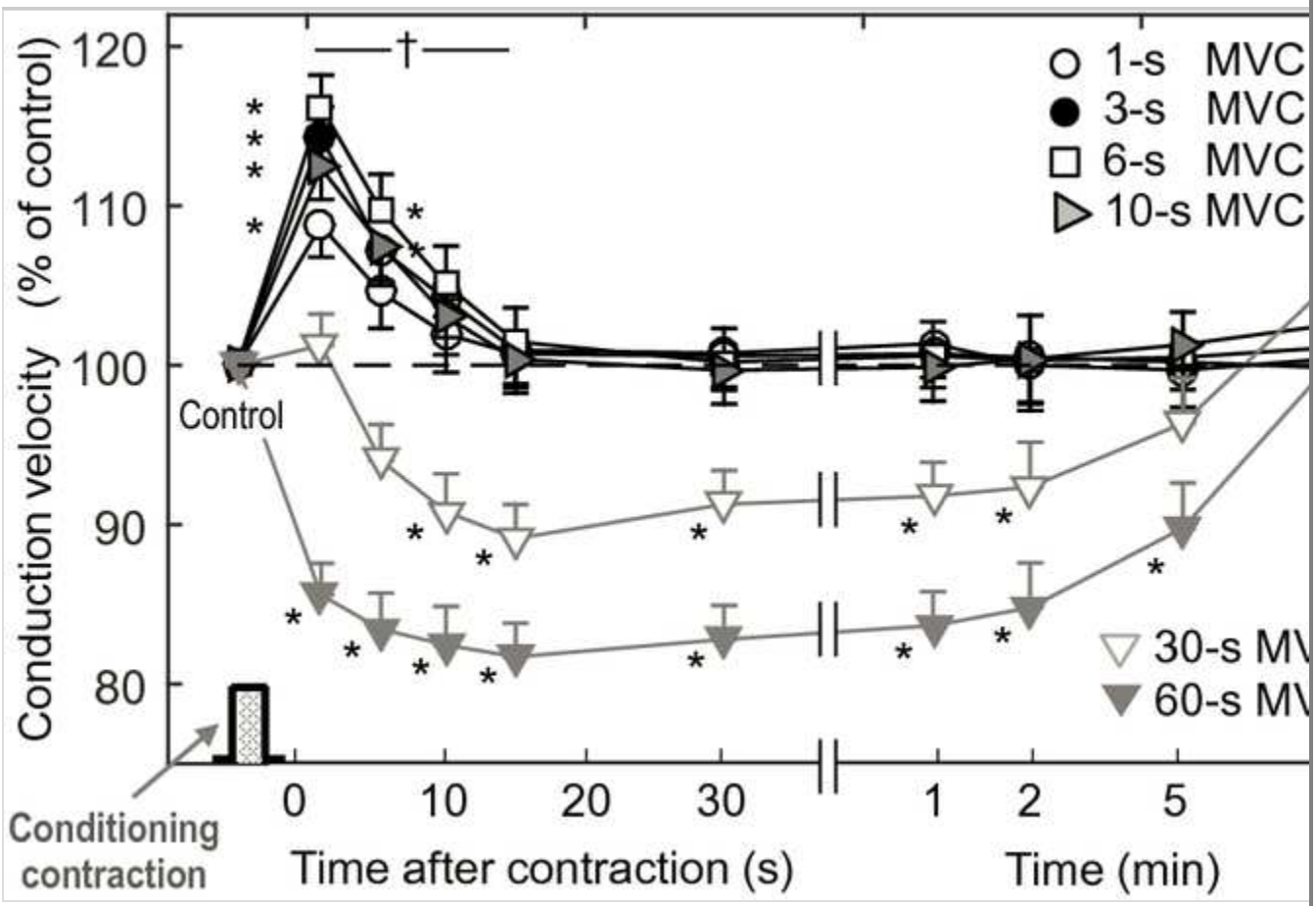


Table 1

Conduction velocity at various time points after conditioning maximal voluntary contractions (MVCs) of different durations for the *vastus lateralis*

		Conduction velocity (m/s)							
MVC duration	**Method**	**Control values**	**1 s**	**5 s**	**10 s**	**30 s**	**2 min**	**5 min**	**10 min**
3-s MVC	Delay method	4.9 ± 1.2	5.7 ± 1.3*	5.3 ± 1.2*	5.1 ± 1.1	4.9 ± 1.2	4.9 ± 1.1	4.9 ± 1.2	5.0 ± 1.2
	Cross-correlation	5.0 ± 1.3	5.8 ± 1.4*	5.3 ± 1.3*	5.2 ± 1.2	5.0 ± 1.3	4.9 ± 1.2	5.0 ± 1.3	5.0 ± 1.2
10-s MVC	Delay method	5.0 ± 1.2	5.9 ± 1.3*	5.4 ± 1.2*	5.2 ± 1.3	5.0 ± 1.2	5.0 ± 1.1	4.9 ± 1.2	5.1 ± 1.2
	Cross-correlation	5.1 ± 1.3	6.0 ± 1.5*	5.5 ± 1.3*	5.2 ± 1.4	5.1 ± 1.2	5.1 ± 1.3	5.0 ± 1.2	5.1 ± 1.3
30-s MVC	Delay method	5.0 ± 1.1	5.1 ± 1.2	4.7 ± 1.2	4.5 ± 1.2*	4.5 ± 1.2*	4.6 ± 1.2*	4.8 ± 1.1	5.3 ± 1.3*
	Cross-correlation	5.1 ± 1.3	5.0 ± 1.3	4.6 ± 1.4*	4.5 ± 1.3*	4.5 ± 1.3*	4.6 ± 1.2*	4.9 ± 1.3	5.4 ± 1.4*
60-s MVC	Delay method	5.1 ± 1.2	4.2 ± 1.3*	4.1 ± 1.1*	4.0 ± 1.2*	4.1 ± 1.2*	4.3 ± 1.1*	4.5 ± 1.2*	5.2 ± 1.3
	Cross-correlation	5.2 ± 1.3	4.3 ± 1.4*	4.2 ± 1.3*	4.1 ± 1.3*	4.1 ± 1.3*	4.3 ± 1.3*	4.5 ± 1.3*	5.3 ± 1.3
The data after the 1-s and 6-s MVC are not shown for clarity. All values are expressed as mean ± SD (n = 15)									
*Significant difference with control ($P < 0.05$)									

With regard to long (≥ 30 s) fatiguing MVCs, conduction velocity was decreased immediately after (1 s) the 60-s MVC ($P < 0.05$, Table 1), but, unexpectedly, it remained unchanged at 1 s after the 30-s MVC ($P > 0.05$). After the long (≥ 30 s) MVCs, conduction velocity was not minimal at 1 s after exercise, but rather it

decreased during the first 15 s after exercise, when it reached its lowest value, after which it started recovering gradually, reaching pre-fatigue values within 5–10 min.

Conduction velocity was also estimated using the cross-correlation technique and the results were in all aspects similar to those reported above using the delay between the positive peaks of the bipolar M waves ($P > 0.05$). Specifically, data of conduction velocity estimated by both methods are provided in Table 1.

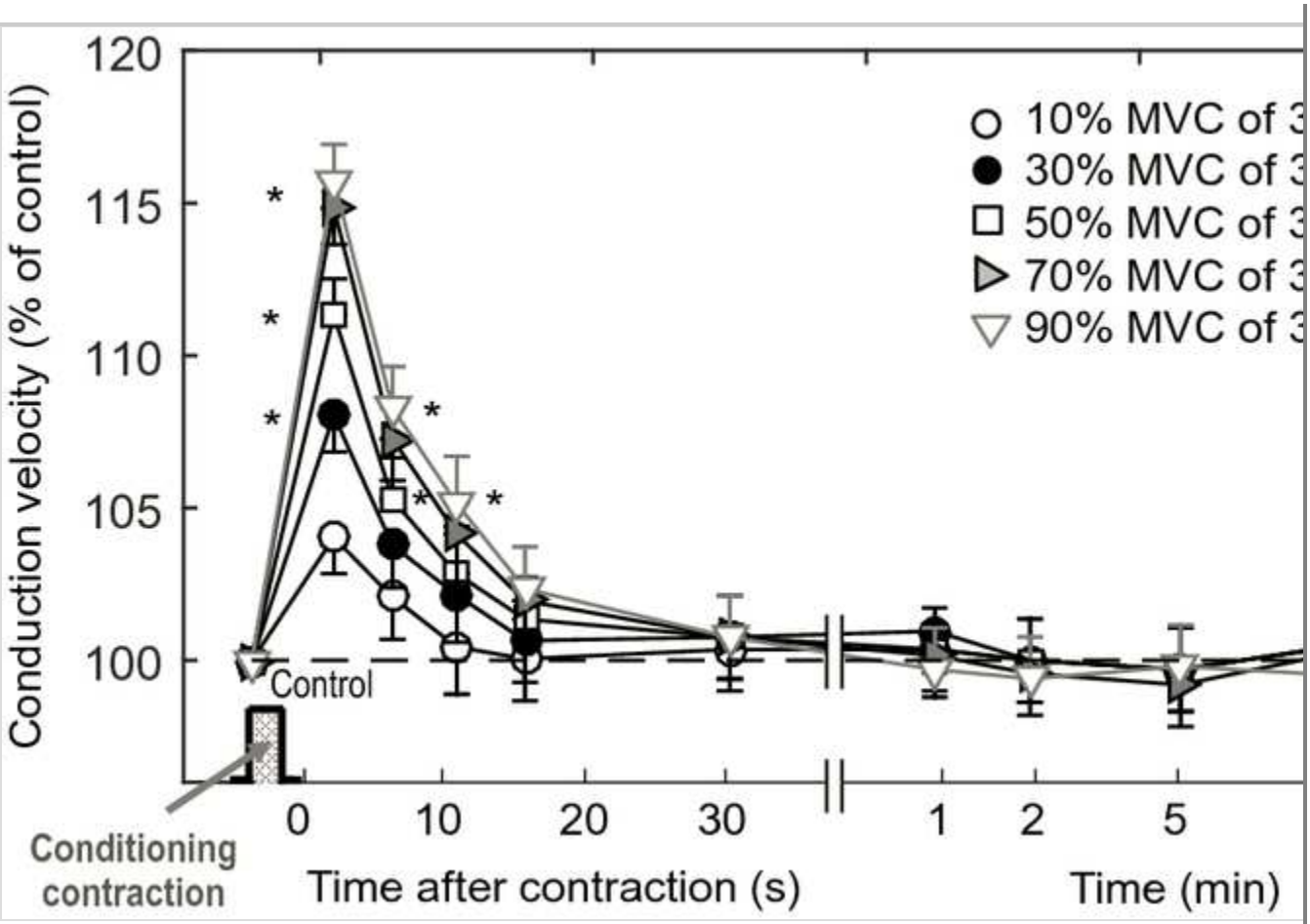
The average force at the end of the 3-s, 6-s, 10-s, 30-s, and 60-s MVCs declined by, respectively, 5 ± 1%, 9 ± 2%, 14 ± 3%, 30 ± 7% and 60 ± 13% of the force recorded at the onset of the MVCs ($P < 0.05$).

Recovery of conduction velocity after brief contractions of different intensities

To get insight into the mechanisms responsible for the short transient increase in conduction velocity after the brief (≤ 10 s) MVCs, we examined the changes in conduction velocity following contractions of different intensities, but with the same (3 s) duration, as shown in Fig. 4. It can be seen that, for all contraction intensities, conduction velocity increased immediately after the conditioning contraction (with the maximum values being reached 1 s after the contraction) and then returned to baseline values very rapidly, within 15 s ($P < 0.05$). The extent of the increase in conduction velocity after the brief contraction increased with contraction intensity from 10 to 70% MVC ($P < 0.05$). Noteworthy, both the extent and time course of recovery of conduction velocity was identical after the contractions at 70, 90, and 100% MVC ($P > 0.05$).

Fig. 4

Time course of recovery of muscle fibre conduction velocity after conditioning voluntary contractions with the same duration (3 s), but different intensities. The data of conduction velocity after the 100% MVC were not shown for clarity, but was similar to that after the 70 and 90% MVC. All data are expressed in percentage of control values and reported as mean ± SE ($n = 15$). *Significant difference with control ($P < 0.05$)



Important note: For the sake of clarity, some results of the study were included in the "supplementary material" section placed at the end of the manuscript. This supplementary data comprise the changes in the time and frequency characteristics of the M-wave (peak-to-peak duration and median frequency, respectively).

Discussion

The main findings of the present study were: (1) muscle conduction velocity increased immediately after contractions of brief (≤ 10 s) duration, but this increase only lasted for a few seconds (~ 15 s); (2) the extent of the increase in conduction velocity was similar after the 3-s, 6-s, and 10-s MVCs, and the time course of recovery was similar after all these contractions; (3) the magnitude of the increase in conduction velocity after a brief contraction augmented with the intensity of the contraction up to 70% MVC; (4) conduction velocity was not decreased immediately after a 30-s MVC, and (5) conduction velocity did not reach its minimum immediately after a long (≥ 30 s) MVC contraction, but rather

it decreased during the first 15 s after exercise.

Peculiarities of the short-term increase in conduction velocity after brief contractions

We found that conduction velocity increased transiently after an MVC of 1-s duration (no decline in MVC force), and this increase was observed even after brief (3 s) contractions of low intensity (30% MVC). Thus, it seems that muscle fatigue (i.e. a reduction in the maximal force generating capacity) is not involved in this phenomenon. Other unique peculiarities of this phenomenon are the following. First, a conditioning MVC as short as 1 s was sufficient to increase significantly conduction velocity, suggesting that the mechanism responsible for this short-term increase in velocity is strikingly fast. Also interesting is the fact that the extent of the increase in conduction velocity was similar after the 3-s, 6-s, and 10-s MVCs: this indicates that the mechanism underlying the increase in conduction velocity almost reached its maximal capacity within the third second of an MVC. Another remarkable finding is that, after the initial increase, conduction velocity decreased very quickly (15 s) to baseline levels: this recovery time is relatively short compared to the normalisation time of extracellular K^+ concentration (Vyskocil et al. 1983). However, what is more surprising is the finding that the recovery time of conduction velocity was practically identical after the 3-s, 6-s, and 10-s MVCs, despite the fact that the 10-s MVC induced greater fatigue than the 3-s MVC [the decline in MVC force was greater after the 10-s MVC (14 ± 3%) than after the 3-s MVC (5 ± 1%), $P < 0.05$]. Taken together, the above observations suggest that the mechanism(s) responsible for the short-term increase in conduction velocity are not related to the ionic membrane processes, as it can be the case for contractions leading to muscle fatigue.

Why the phenomenon has not been documented in previous research

For various reasons, the present phenomenon of a short-term increase in conduction velocity after a brief contraction has not been documented in previous studies. First, in the past, researchers were more interested in understanding why muscle conduction velocity decreases in the presence of muscle fatigue. Accordingly, investigators tested exercise protocols that induced acute muscle fatigue, which normally consisted on prolonged high-intensity

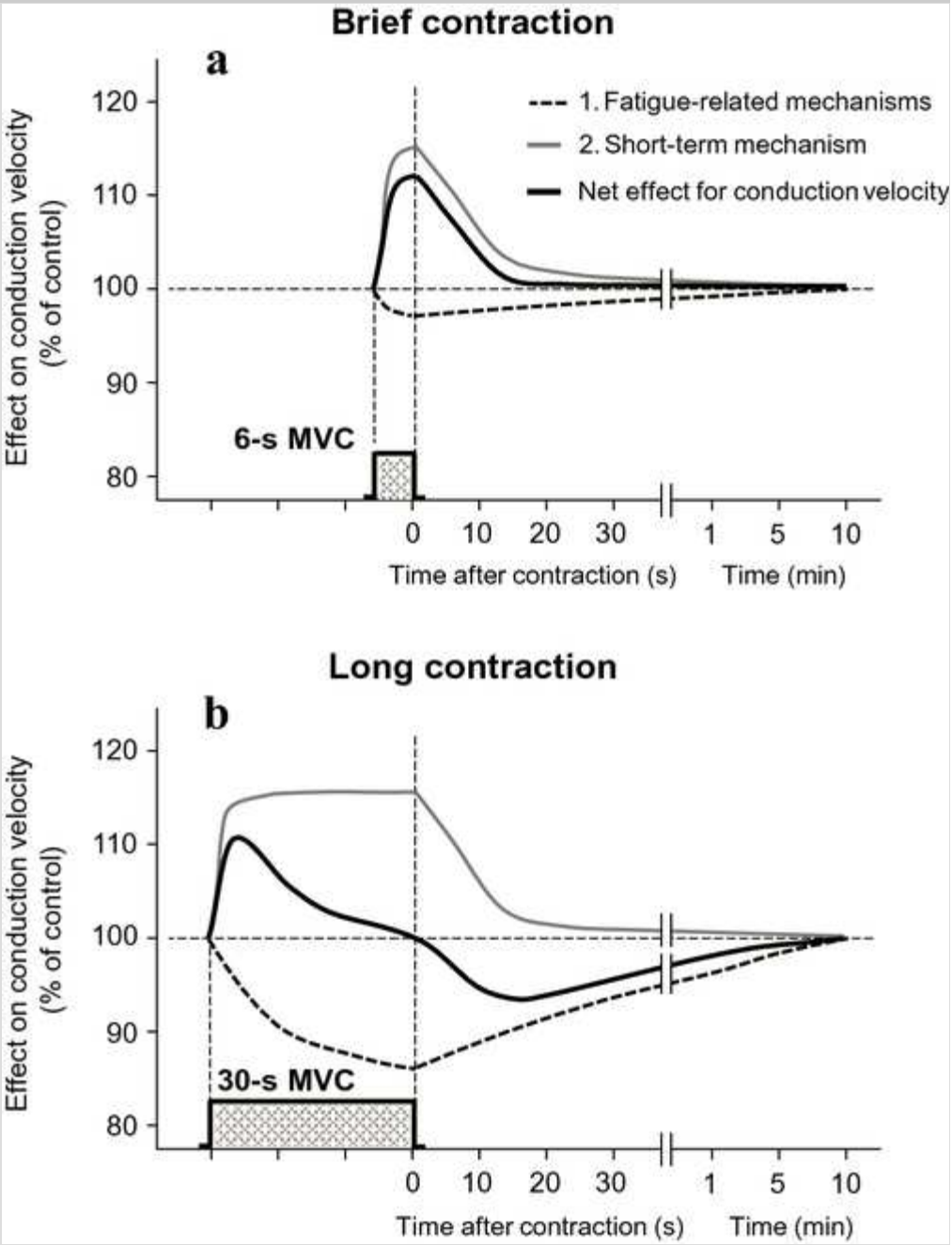
exercise. Second, previous researchers did not systematically examine the changes in conduction velocity produced by contractions of different durations and intensities, i.e. they normally tested the effects of contractions of a given duration and intensity (Van der Hoeven et al. 1993). Another factor that has likely prevented the observation of this phenomenon is that, in past experiments, the first seconds following a contraction were not characterised in sufficient detail: generally, none or just one measure/sample of conduction velocity was taken within the first 15 s of the recovery. Indeed, in previous studies on conduction velocity, the first measure was taken at 30 s (Cupido et al. 1996), and at 1 min (Van der Hoeven et al. 1993) into the recovery.

The mechanism causing the short-term increase in conduction velocity is also present after a long contraction

It follows from the present results that, as a muscle contraction is initiated, a mechanism is triggered rapidly in the muscle, which has an increasing effect on conduction velocity. The presence of such “fast” mechanism can only be unequivocally demonstrated after a (brief) contraction, i.e. a contraction that induces negligible or no fatigue (see the model of Fig. 5). This is because, as a contraction is prolonged, the fatigue effects may counteract and mask the increasing effect of this “fast” mechanism on conduction velocity.

Fig. 5

Proposed model for the two group of mechanisms acting on conduction velocity during and after a short (**a**) and a long (**b**) maximal voluntary contraction. The first group of mechanisms, fatigue-related, works to decrease conduction velocity during the contraction, while these mechanisms act to increase conduction velocity slowly during recovery. The presence of a second mechanism, “short-term”, is inferred from the present findings: this mechanism acts to increase conduction velocity during the contraction, whereas this mechanism works to decrease velocity of muscle fibres during the recovery. A schematic time course of each process, consistent with actual measurements, is indicated



It could be argued that, when a contraction is sustained for a long period, this fast mechanism is greatly reduced or abolished. However, our results provide several pieces of evidence indicating that, even after a long fatiguing contraction, a mechanism having a short-term increasing effect on conduction velocity is operative. First, immediately after (1 s) the 30-s MVC, the high levels of extracellular K^+ concentration attained (Vyskocil et al. 1983) would be expected to cause a clear drop in conduction velocity (Juel 1988). However, we found that conduction velocity remained unchanged 1 s after the 30-s MVC, indicating that there must be another mechanism that compensated the depressing effect of K^+

accumulation on conduction velocity (see the model of Fig. 5). Second, it would be expected that, after the 30-s and 60-s MVCs, conduction velocity would be minimal immediately after (1 s) the cessation of exercise (when extracellular K^+ concentration was at its maximum), after which it would increase progressively towards resting levels. However, after the long MVCs, we observed that conduction velocity was not minimal at 1 s after exercise, but rather it decreased significantly between 1 and 15 s after exercise ($P < 0.05$), when it reached its lowest value, before start recovering. This complex recovery pattern of conduction velocity reinforces the idea that, after a muscle contraction, various processes having both increasing and decreasing effects on conduction velocity do coexist.

Possible mechanisms underlying the short transient increase in conduction velocity

We propose that the short-term increase in conduction velocity after short contractions is most likely related to the changes in muscle fibre diameter that occur during a contraction. It is known that, as a muscle fibre is shortened during a contraction, its conduction velocity increases due the increase in fibre diameter (more specifically, the reduction in fibre transverse internal resistances) (Martin 1954; Fortune and Lowery 2012). It might be hypothesised that the increase in muscle fibre diameter persists for a few seconds (~ 15 s) after cessation of the contraction, before returning to normal values. Thus, the short transient increase in conduction velocity observed after a brief contraction would occur because the thickness of muscle fibres remains increased for a short period after the contraction. This hypothesis would explain most of the present observations. First, the increase in fibre diameter occurs at the very moment a muscle contraction is initiated: this would explain why the increase in conduction velocity was observed even after a contraction as short as 1 s. Second, the increase in fibre diameter probably reaches a plateau during the first seconds of a sustained maximal contraction: this would explain our observation that conduction velocity increased at a similar extent after the 3-s, 6-s, and 10-s MVCs. Third, muscle fibres shorten progressively (and their diameters increase more and more), as the intensity of a contraction increases (Hodges et al. 2003): this would account for our finding that the magnitude of the increase in conduction velocity augmented when contraction intensity was increased. Finally, it is worth noting that the increase in fibre thickness is a “mechanical-geometrical” mechanism, and thus it must be operative even after a prolonged

fatiguing contraction. This might be the reason why conduction velocity was not decreased after the 30-s MVC: the depressing effect of K^+ accumulation was compensated by the transient increase in fibre diameter after this contraction.

The possible involvement of muscle temperature on the present results deserves comment. It is known that, as a contraction is sustained, the gradual increase of muscle temperature acts to increase conduction velocity (Stålberg 1966). However, the short-term increase in conduction velocity reported here was observed even after brief contractions, i.e. before any significant increase in temperature could occur in the muscle. Thus, muscle temperature can hardly be the explanation for this phenomenon. Nevertheless, because temperature increases slowly during a contraction, it may have an effect on conduction velocity after prolonged contractions. Indeed, the observation that conduction velocity was not decreased after the 30-s MVC could partly be due to the fact that the depressing effects of fatigue (K^+ accumulation) on conduction velocity were counteracted by the increased muscle temperature. On the other hand, restrictions in blood supply have been shown to decrease conduction velocity (Farina et al. 2005b). However, the transient increase in conduction velocity reported here was observed even after maximal contractions, when the high intramuscular pressure impeded blood flow.

Finally, a comment is in order on the effect of muscle length on conduction velocity (Arendt-Nielsen et al. 1992). In the present experiments, the length of muscle fibres was likely different at the beginning and at the end of a prolonged contraction. Likewise, muscle fibre length was probably different after contractions of different intensities. For future investigations, it would be interesting to assess muscle fascicle length under the experimental conditions tested in the present study.

A possible model for the mechanisms that affect conduction velocity after a contraction

From the above it can be concluded that, both during and after a muscle contraction, there are at least two groups of mechanisms acting in opposite directions on conduction velocity. A model of the possible effects of these competing mechanisms is represented in Fig. 5. The first group of mechanisms (“fatigue-related”) contribute to increase the extracellular K^+ concentration during a contraction (decreased muscle perfusion due to increased intramuscular

pressure). After the contraction, the fatigue effects are slowly reduced, a reduction mediated by various mechanisms, such as diffusion of K^+ into the capillaries and the action of the Na^+–K^+ pumping. Thus, the recovery of fatigue-related mechanisms was assigned a long time constant. On the other hand, we propose that a second mechanism having an increasing effect on conduction velocity comes into play from the onset of the contraction. At cessation of the contraction, muscle fibres tend to recover their normal length, and thus reduce its diameter during recovery. The recovery time for this second mechanism is extremely fast (15 s), and thus was assigned a short time constant.

The relative importance of these two groups of mechanisms depends on the duration (and intensity) of the contraction. After a brief contraction (plot a), the increase in extracellular K^+ concentration is very low and thus would play a minor/negligible role in the recovery of conduction velocity: the dominant factor in this scenario would be the short-term mechanism. In contrast, after a long ($\geq$ 30 s) fatiguing contraction (plot b), the high accumulation of extracellular K^+ ions would act to decrease conduction velocity, and the slow clearance of such K^+ ions would dominate the recovery of conduction velocity. It is important to note that, because the recovery of the short-term mechanism is much steeper and abrupt than that of the fatigue-related mechanisms, conduction velocity would decrease between 1 and 15 s after both the short and long contractions. Finally, it is probable that these two groups of mechanisms do not work in isolation, but may interact with each other.

Limitations of the study

In the present experiments, we did not measure the changes in muscle architectural parameters (fascicle length) during/after the contractions and, thus, we can only speculate that the short transient increase in conduction velocity observed after a brief contraction was attributable to fact that the diameters of muscle fibres remain increased for a short period after the contraction. Likewise, we did not measure extracellular K^+ concentration, and therefore, the argument that the decrease in conduction velocity after long contractions was due to a high accumulation of extracellular K^+ ions is not based on the present data. Finally, the possibility that the successive twitches elicited after the short-duration contraction could have slightly influenced the observed changes in conduction velocity should not be discarded and should be examined in future studies.

Implications, cautions and future research

The present findings are of importance to the fields of muscle physiology and sports science for various reasons. First, the velocity of impulse conduction along muscle fibres is considered as an indicator of sarcolemmal membrane excitability (Fortune and Lowery 2009, 2012). Therefore, the fact that conduction velocity is transiently increased after a contraction may be interpreted as a sign that membrane excitability is enhanced for a short period after cessation of contractile activity. This transient elevation of membrane excitability may be advantageous from a functional perspective, as it may “facilitate” impulse conduction during a contraction occurring shortly after a previous contraction: in other words, it permits that the action potentials start propagating at a velocity higher than baseline values during a contraction performed shortly after a previous contraction. From this perspective, this transient “enhancement” of membrane excitability may be considered a contraction-history effect such as the phenomenon of post-activation potentiation (Alway et al. 1987).

Second, it is known that alterations in neuromuscular propagation might contribute to the loss of force during a fatiguing contraction and it is therefore important to test the M-wave immediately after the contraction. However, we have shown that a brief muscle contraction induced a short-term effect not only on conduction velocity, but also on the amplitude and duration characteristics of the M-wave (for an example, see Figs. 1S and 2S in the supplementary material). Specifically, the amplitude of the M-wave second phase increased and M-wave duration decreased for the first 10–15 s after contractions of brief duration (Rodriguez-Falces and Place 2017). Thus, it is likely that some M-wave parameters, such as peak-to-peak amplitude and duration, may be slightly “affected” by the present short-term mechanism if the M-wave is elicited within the first 15 s following a muscle contraction.

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Future research should be conducted to confirm the present findings. A direct way to confirm the results would consist on estimating conduction velocity using an intramuscular technique, such as that developed by Troni et al. (1983).

Conclusion

In conclusion, it has been demonstrated that muscle conduction velocity

increased immediately after contractions of brief (≤ 10 s) duration, but this increase only lasted for about 15 s. This short-term increase in conduction velocity was observed even after maximal contractions as short as 1 s. The extent of the increase in conduction velocity was similar after the 3-s, 6-s, and 10-s MVCs, and the recovery time (~ 15 s) was similar after all these contractions. Collectively, these findings indicate that: (1) a mechanism having an increasing effect on conduction velocity is active from the onset of a muscle contraction; (2) the mechanism reaches its maximal capacity within the third second of an MVC; (3) the mechanism persists for a short period after the contraction; (4) the mechanism is likely unrelated to the ionic concentrations inside and outside the muscle cell and/or to muscle fatigue. Moreover, our observation that conduction velocity was not decreased immediately after a 30-s MVC evidences that the mechanism having an increasing effect on conduction velocity is operative even after long fatiguing contractions, which suggests that this mechanism should have a “mechanical-geometrical” character. It is tentatively suggested that this “mechanical” mechanism could be related to the fact that the diameters of muscle fibres remain increased for a short period after exercise. It is concluded that brief (≤ 10 s) muscle contractions induce a short-term increase in conduction velocity, lasting about 15 s, while long (≥ 30 s) contractions produce a long-term decrease in conduction velocity, lasting more than 2 min.

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Author contributions

JR-F and NP designed the experimental study; JR-F performed the experiments; J R-F analysed the data; J R-F and NP interpreted the results of experiments; J R-F drafted the manuscript; J R-F and NP edited and revised the manuscript; JR-F and NP approved the final version of manuscript.

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Supplementary Information

Below is the link to the electronic supplementary material.

Supplementary file1 (DOCX 240 KB)

References

Aidley DJ (1998) The physiology of excitable cells, 4th edn. Cambridge University Press, Cambridge

Alway SE, Hughson RL, Green HJ, Patla AE, Frank JS (1987) Twitch potentiation after fatiguing exercise in man. Eur J Appl Physiol 56:461–466

Andreassen S, Arendt-Nielsen L (1987) Muscle fibre conduction velocity in motor units of the human anterior tibial muscle: a new size principle parameter. J Physiol 391:561–571

Bigland-Ritchie B, Johansson R, Lippold OC, Smith S, Woods JJ (1983) Changes in motoneurone firing rates during sustained maximal voluntary contractions. J Physiol 340:335–346

Brody LR, Pollock MT, Roy SH, De Luca CJ, Celli B (1991) pH-induced effects on median frequency and conduction velocity of the myoelectric signal. J Appl Physiol 71:1878–1885

Broman H, Bilotto G, De Luca CJ (1985) Myoelectric signal conduction velocity and spectral parameters: influence of force and time. J Appl Physiol 58(5):1428–1437

Christova P, Kossev A, Kristev I, Chichov V (1999) Surface EMG recorded by branched electrodes during sustained muscle activity. J Electromyogr Kinesiol 9(4):263–276

Cupido CM, Galea V, McComas AJ (1996) Potentiation and depression of the M wave in human biceps brachii. J Physiol 491:541–550

Farina D, Fosci M, Merletti R (2002) Motor unit recruitment strategies investigated by surface EMG variables. J Appl Physiol 92(1):235–247

Farina D, Arendt-Nielsen L, Graven-Nielsen T (2005a) Effect of temperature on spike-triggered average torque and electrophysiological properties of low-threshold motor units. J Appl Physiol 99:197–203

Farina D, Gazzoni M, Camelia F (2005b) Conduction velocity of low-threshold motor units during ischemic contractions performed with surface EMG feedback. J Appl Physiol 98:1487–1494

Fortune E, Lowery MM (2009) Effect of extracellular potassium accumulation on muscle fibre conduction velocity: a simulation study. Ann Biomed Eng 37:2105–2117

Fortune E, Lowery MM (2012) Effect of membrane properties on skeletal muscle fibre excitability: a sensitivity analysis. Med Biol Eng Comput 50(6):617–629

Hakansson CH (1956) Conduction velocity and amplitude of the action potential as related to circumference in the isolated fibre of frog muscle. Acta Physiol Scand 37(1):14–34

Hodges PW, Pengel LH, Herbert RD, Gandevia SC (2003) Measurement of muscle contraction with ultrasound imaging. Muscle Nerve 27(6):682–692

Homma S, Iwata K, Kusama T, Nakajima Y (1983) Effects of electrical constants on conduction velocity of action potentials measured with unidimensional latency-topography in frog skeletal muscle fibres. Jpn J Physiol 33(5):711–720

Juel C (1986) Potassium and sodium shifts during in vitro isometric muscle contraction, and the time course of the ion-gradient recovery. Pflugers Arch 406(5):458–463

Juel C (1988) Muscle action potential propagation velocity changes during activity. Muscle Nerve 11:714–719

Kössler F, Lange F, Caffier G, Küchler G (1991) External potassium and action potential propagation in rat fast and slow twitch muscles. Gen Physiol Biophys 10(5):485–498

Lateva ZC, McGill KC, Burgar CG (1996) Anatomical and electrophysiological determinants of the human thenar compound muscle action potential. Muscle Nerve 19(11):1457–1468

Mademli L, Arampatzis A (2005) Behaviour of the human gastrocnemius muscle architecture during submaximal isometric fatigue. Eur J Appl Physiol 94(5–6):611–617

Maganaris CN, Baltzopoulos V, Sargeant AJ (2002) Repeated contractions alter the geometry of human skeletal muscle. J Appl Physiol 93(6):2089–2094

Martin AR (1954) The effect of change in length on conduction velocity in muscle. J Physiol 125(1):215–220

Masuda T, Miyano H, Sadoyama T (1985) The position of innervation zones in the biceps brachii investigated by surface electromyography. IEEE Trans Biomed Eng 32:36–42

McGill KC, Lateva ZC (2011) History dependence of human muscle-fibre conduction velocity during voluntary isometric contractions. J Appl Physiol 111:630–641

Naeije M, Zorn H (1983) Estimation of the action potential conduction velocity in human skeletal muscle using the surface EMG cross-correlation technique. Electromyogr Clin Neurophysiol 23(1–2):73–80

Overgaard K, Nielsen OB, Clausen T (1997) Effects of reduced electrochemical Na+ gradient on contractility in skeletal muscle: role of the Na+–K+ pump. Pflugers Arch 434(4):457–465

Rodriguez-Falces J, Place N (2016) Differences in the recruitment curves obtained with monopolar and bipolar electrode configurations in the quadriceps femoris. Muscle Nerve 54(1):118–131

Rodriguez-Falces J, Place N (2017) New insights into the potentiation of the first and second phases of the M-wave after voluntary contractions in the quadriceps muscle. Muscle Nerve 55(1):35–45

Rodriguez-Falces J, Place N (2018) End-of-fibre signals strongly influence the first and second phases of the M wave in the *Vastus Lateralis*: implications for the study of muscle excitability. Front Physiol 9:162

Rodriguez-Falces J, Navallas J, Malanda A, Rodriguez-Martin O (2014) Comparison of the duration and power spectral changes of monopolar and bipolar M waves caused by alterations in muscle fibre conduction velocity. J Electromyogr Kinesiol 24:452–464

Rodriguez-Falces J, Duchateau J, Muraoka Y, Baudry S (2015) M-wave potentiation after voluntary contractions of different durations and intensities in the tibialis anterior. J Appl Physiol 118:953–964

Saltin B, Sjogaard G, Strange S, Juel C (1987) Redistribution of K+ in the human body during muscular exercise; its role to maintain whole body homeostasis. In: Shiraki K, Yousef MK (eds) Man in stressful environments. Thermal and work physiology. Thomas, Springfield, IL, pp 247–267

Schmitz JP, van Dijk JP, Hilbers PA, Nicolay K, Jeneson JA, Stegeman DF (2012) Unchanged muscle fibre conduction velocity relates to mild acidosis during exhaustive bicycling. Eur J Appl Physiol 112:1593–1602

Schneider J, Silny J, Rau G (1988) Noninvasive measurement of conduction velocity in motor units influenced by temperature and excitation pattern. In: Wallinga W, Boom HBK, DeVries J (eds) Electromyographical kinesiology. Elsevier, Amsterdam, pp 251–254

Sejersted OM, Sjøgaard G (2000) Dynamics and consequences of potassium shifts in skeletal muscle and heart during exercise. Physiol Rev

80(4):1411–1481

Stålberg E (1966) Propagation velocity in human muscle fibres in situ. Acta Physiol Scand Suppl 287:1–112

Stulen FB, DeLuca CJ (1981) Frequency parameters of the myoelectric signal as a measure of muscle conduction velocity. IEEE Trans Biomed Eng 28:515–523

Troni W, Cantello R, Rainero I (1983) Conduction velocity along human muscle fibres in situ. Neurology 33(11):1453–1459

Van der Hoeven JH, van Weerden TW, Zwarts MJ (1993) Long-lasting supernormal conduction velocity after sustained maximal isometric contraction in human muscle. Muscle Nerve 16:312–320

Vyskocil F, Hník P, Rehfeldt H, Vejsada R, Ujec E (1983) The measurement of K+ concentration changes in human muscles during volitional contractions. Pflugers Arch 399:235–237