Conduction Velocity of Quiescent Muscle Fibers Decreases During Sustained Contraction

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Gazzoni, Marco, Federico Camelia, and Dario Farina. Conduction velocity of quiescent muscle fibers decreases during sustained contraction. J Neurophysiol 94: 387–394, 2005; doi:10.1152/jn.01182.2004. We tested the hypothesis that conduction velocity of quiescent muscle fibers decreases during sustained contraction due to the activity of the active motor units in the muscle. Ten subjects trained for the identification of a target motor unit in the abductor pollicis brevis with feedback on surface EMG signals detected with a two-dimensional array of 61 electrodes. The subjects activated the target motor unit in two 10-s long contractions, before (contraction C1) and after (C3) a 3-min contraction (C2), all in ischemic condition. The target motor unit was not activated during C2. Eight of the 10 subjects (control group) performed a second experimental session identical to the first but with a resting period of 3 min instead of the contraction C2. Exerted force and target motor unit discharge rate were not different between the two subject groups and between C1 and C3 (mean ± SD, over C1 and C3; C2 group: 15.8 ± 10.4% maximal voluntary contractions and 13.1 ± 1.9 pps; control group: 15.6 ± 22.1% maximal voluntary contractions and 14.5 ± 1.9 pps, respectively). Muscle fiber conduction velocity of the target motor unit decreased in C3 with respect to C1 in the C2 group (3.59 ± 0.57 and 3.34 ± 0.47 m/s for C1 and C3, respectively; P < 0.05) but not in the control group (3.47 ± 0.68 and 3.46 ± 0.73 m/s). In the C2 group, the percent decrease in conduction velocity of the target motor unit between C1 and C3 (6.4 ± 7.1%) was not significantly different from the percent decrease in the average conduction velocity of the motor units active during C2 (9.6 ± 5.4%). In conclusion, the contraction-induced modifications in electrophysiological membrane properties of muscle fibers are partly independent on fiber activation.

INTRODUCTION

Muscle fiber conduction velocity provides direct information on fiber electrophysiological properties (Farina and Merletti 2004). Conduction velocity of a recruited motor unit decreases over time during sustained contraction (Juel 1988) due to the repetitive generation of the action potential. An important determinant of the decrease in conduction velocity is the modification of the extracellular environment. Muscle fiber activity may also affect the electrolyte balance across the muscle fiber membrane (McKenna 1992) with an increase in the extracellular potassium concentration (Sjøgaard and McComas 1995). Because the fibers of different motor units are intermingled between each other (Brandstater and Lambert 1973; Kugelberg and Edstрем 1968), the alteration in the concentration of ion species across the fiber membrane during muscle contraction may also affect the membrane properties of fibers that are not active during the contraction. Thus it may be expected that membrane properties of muscle fibers change during sustained contraction, partly independent on their activity. However, this hypothesis has never been tested in vivo in single motor units. Eventual changes in muscle fiber membrane properties due to the contraction-induced alteration of the extracellular environment would underline an important limitation of the use of in vitro studies for interpreting the changes occurring at the fiber membrane level with sustained contraction.

Multi-channel surface EMG recordings can be applied to analyze the membrane properties of muscle fibers both at global and single motor unit level. However, surface EMG has poor spatial selectivity that hinders the identification of single motor units, especially at medium/high contraction levels. To overcome this problem, we recently proposed the use of a visual feedback on multi-channel surface EMG signals to identify and follow the activity of single motor units (Farina et al. 2004b). With this method, it was possible to analyze each action potential generated by a target motor unit in normal and ischemic conditions (Farina et al. 2004a,b). The possibility of identifying and recalling the same motor unit with surface EMG feedback allows the analysis of changes in motor unit conduction velocity modulated by sustained contraction, ischemia, or other conditions.

The aim of this study was to test the hypothesis that a sustained contraction determines changes in the conduction velocity of motor units, independently of their activation. Thus single motor units were analyzed with multi-channel surface EMG feedback before and after a low-force sustained contraction during which they were not activated. To induce appreciable changes in metabolite concentration during relatively short and low-force contractions, the muscle was studied in ischemic conditions. This resembled the situation at higher force contraction levels when the intramuscular pressure overcomes the systolic pressure and the muscle becomes ischemic.

METHODS

Subjects

Ten healthy male subjects (age, 26.3 ± 2.1 (SD) yr; height, 1.82 ± 0.06 m; weight, 72.4 ± 6.9 kg) participated in the study. The local...
ethics committee approved the study, and all subjects signed an informed consent form before participation.

**Surface EMG recordings**

Surface EMG signals were detected with a two-dimensional array of 61 silver electrodes (1 mm diam, 3-mm interelectrode distance, 5 columns and 13 rows without the 4 corner electrodes; Fig. 1A) from the abductor pollicis brevis of the dominant hand. The small electrode size and interelectrode distance allowed high spatial selectivity (Reucher et al. 1987). The EMG signals were amplified with four 16-channel EMG amplifiers (LISiN-Prima Biomedical & Sport, Treviso, Italy), band-pass filtered (3-dB bandwidth, 10–500 Hz), sampled at 1,650 Hz, converted to digital form by 12-bit A/D converters, and displayed in real time as bipolar derivations to the subject.

The electrode grid was located with the columns in the direction of the muscle fibers and covered the distal semi-fiber length (from the innervation zone to the distal tendon) and part of the proximal semi-fiber. Before electrode placement, the skin was abraded with abrasive paste (Meditec-Every, Parma, Italy). The grid was fixed on the skin by adhesive tape (Fig. 1B), and a reference electrode was placed at the wrist.

**General procedures**

A custom-designed brace was used to measure abduction force (Fig. 1B). The subject’s wrist was fixed in a padded wood support with the head of the thumb phalanx in touch with a load cell (model 8523-50N, Burster, Gernsbach, Germany). The force signal was amplified (Force Amplifier, MISO-II, LISiN) and recorded in parallel with the EMG signals.

The subjects performed three maximal voluntary contractions (MVCs) separated by 2-min rest, after which the electrode grid was located over the abductor pollicis brevis. The subject was asked to identify the activity of a single motor unit (target motor unit) in the surface EMG recordings. To do so, he varied the force level until the activity of a single motor unit could be visually identified from the surface recordings. The feedback consisted of the display of the bipolar surface EMG signals in segments of 500 ms, as previously described (Farina et al. 2004b). The subject selected the column of the matrix that provided the best feedback. The amplification factor was adjusted to optimize the feedback.

The training phase lasted ~20 min, after which ischemia was induced in the hand with a cuff inflated around the forearm at 180 mmHg pressure. The blocking of blood flow was confirmed by the absence of a palpable peripheral pulse. The subject rested for 5 min in ischemic conditions and then activated the target motor unit for 10 s at ~12 pulses per second (pps; contraction C1; Fig. 1C) using the surface EMG feedback. Immediately after contraction C1, he performed a 3-min contraction at a force level just below the recruitment threshold of the target motor unit (contraction C2). To avoid recruitment of the target motor unit during the entire duration of contraction C2 (subthreshold contraction), the subject used the surface EMG

![Fig. 1. A: 2-dimensional array of 61 electrodes (3-mm interelectrode distance in both directions) used for surface EMG recording. B: brace for measuring abduction force. The subject’s hand was fixed in a padded wood support. Electrode grid was placed on the abductor pollicis brevis. C: schematic representation of the contraction sequence. In the C2 group, contractions C1, C2, and C3 were performed with surface EMG feedback to assure the activity (during C1 and C3) and absence of activity (during C2) of the target motor unit. In the control group, C2 was substituted by a 3-min rest period. Thus in both subject groups, the target motor unit was not active in the 3-min time interval between contractions C1 and C3.](http://jn.physiology.org/content/jn/94/7/388/F1)

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feedback and continuously checked for the absence of the target motor unit action potentials. No feedback on the exerted force was provided. After contraction C2, the subject repeated a contraction identical to C1, with the target motor unit activated at ~12 pps for 10 s (contraction C3). Comparison of the target motor unit conduction velocity between contractions C1 and C3 allowed us to determine if the sustained contraction C2 affected the properties of nonactive muscle fibers.

Eight of the 10 subjects participated in a second experimental session, at least 2 days after the first. The second session was identical to the first, with the only difference that, between contractions C1 and C3, instead of contraction C2, the subject rested for 3 min in ischemic conditions (Fig. 1C). This session served as a control to assure that ischemia by itself did not induce modifications in muscle fiber conduction velocity.

Signal analysis

The target motor unit action potentials in C1 and C3 were detected off-line from the interference EMG signals with a segmentation-classification algorithm previously described (Gazzoni et al. 2004). The column of the electrode grid that detected action potentials of the target motor unit with the highest amplitude was used for conduction velocity estimation. From the double differential signals computed from the selected column, those distal to the innervation zone showing clear propagation and small shape changes of the potential waveform were selected for conduction velocity estimation. Conduction velocity was computed from each identified action potential of the target motor unit with a multi-channel algorithm previously described (Farina et al. 2001). Peak-to-peak amplitude, duration (area divided by the peak-to-peak amplitude; Nandedkar et al. 1986), and mean power spectral frequency of the target motor unit action potentials were estimated from the central channel of those used for conduction velocity estimation. Discharge rate was computed as the inverse of the time interval between subsequent detected discharges of the target motor unit.

For the subject group that performed contraction C2 (C2 group), global conduction velocity, average rectified value, and mean power spectral frequency were estimated during C2 from consecutive and nonoverlapping EMG signal portions of 1 s, using the same channels as for the analysis of the target motor unit action potentials. Global conduction velocity was computed with the same algorithm used for single motor unit conduction velocity estimation (Farina et al. 2001). Global variables reflected the properties of all active motor units in the C2 contraction.

Data were fit with a regression line, which defined the initial value (intercept of the regression line at time t = 0) and the rate of change over time (slope of the regression line) of the computed variables. The percent decrease was defined as the difference between the value in the end and in the beginning of the contraction, divided by the initial value and expressed in percentage.

Statistical analysis

Data are presented as means ± SD. The target motor unit properties were analyzed using two-way mixed model ANOVA. The repeated measure was the contraction (C1 and C3), and the between-group factor was the subject group (C2 group and control group). Exerted force in the three contractions of the C2 group was analyzed with one-way repeated measures ANOVA, with the contraction as the factor (C1, C2, and C3). Significances revealed by ANOVA were followed by posthoc Student-Newman-Keuls (SNK) pair-wise comparisons. Global surface EMG variables were compared in the beginning and end of C2 with the Wilcoxon matched pairs test. Significance was accepted for P < 0.05.

RESULTS

Figure 2 reports examples of surface EMG signals and related force traces recorded during the three contractions of a subject in the C2 group. The maximum of the cross-correlation function between the surface action potentials of the target motor unit detected in the two contractions was in all cases, for the two subject groups, >0.85. This indicated that most likely the same motor unit was activated with feedback in C1 and C3. No activity of the target motor unit was detected during C2 in all subjects.

Subthreshold contraction of the C2 group

The force exerted in C2 (6.5 ± 4.1% MVC) was lower than that in C1 (ANOVA: F = 5.14, P < 0.05; SNK: P < 0.05). Global conduction velocity (initial value, 3.45 ± 0.66 m/s), average rectified value (147 ± 79 µV), and mean power spectral frequency (120 ± 14 Hz) significantly decreased in the 3-min C2 contraction (9.6 ± 5.4, 30.2 ± 30.3, and 6.3 ± 5.6%, respectively; P < 0.01).

Contractions C1 and C3

None of the computed variables significantly changed over time during C1 and C3 in both subject groups. Thus the initial variable values (intercept of the regression lines) were assumed as representative of the contraction and used for further statistical analysis.

FORCE AND DISCHARGE RATE. Exerted force (C2 group: 19.3 ± 17.4 and 12.3 ± 5.7% MVC, for C1 and C3; control group: 16.3 ± 21.2 and 14.9 ± 18.7% MVC, respectively) and discharge rate (C2 group: 14.0 ± 2.8 and 12.3 ± 2.2 pps for C1 and C3, respectively; control group: 15.3 ± 2.0 and 13.7 ± 2.3 pps) were not significantly different between the two subject groups and between C1 and C3.

TARGET MOTOR UNIT ACTION POTENTIAL. Figure 3 shows examples of single motor unit action potentials detected with the two-dimensional array of electrodes. The detection of motor unit action potentials with the array allowed the analysis of the EMG potential distribution over the skin due to single motor unit activity. The column of the grid with the maximum amplitude potentials corresponded to the location of the motor unit over the skin plane (Fig. 3). Increasing distance from the source in the transverse direction decreased the amplitude of the potentials. Potentials detected along fiber direction (i.e., by electrodes in a column) were similar in shape and delayed between each other. The estimation of the delay between action potential waveforms is inversely related to conduction velocity. The innervation zone was identified as the point of inversion of propagation of the action potentials along the columns.

Action potential conduction velocity depended on the contraction (ANOVA: F = 5.6, P < 0.05). Posthoc SNK test revealed that the target motor unit conduction velocity was significantly lower in C3 than in C1 for the C2 group (n = 10 motor units, 3.59 ± 0.57 and 3.34 ± 0.47 m/s for C1 and C3, respectively; P < 0.05), but it was not different between C1 and C3 in the control group (n = 8 motor units, 3.47 ± 0.68 and 3.46 ± 0.73 m/s). The percent decrease in conduction velocity between C1 and C3 in the C2 group was significantly different from zero and had large variability among subjects.
It was not significantly different from the percent decrease in global conduction velocity between the beginning and end of C2 (Fig. 3).

Peak-to-peak amplitude (C2 group: 895 ± 344 and 955 ± 322 μV for C1 and C3, respectively; control group: 639 ± 732 and 640 ± 690 μV) and duration (C2 group: 4.05 ± 0.22 and 4.20 ± 0.25 ms for C1 and C3, respectively; control group: 4.11 ± 0.21 and 4.25 ± 0.22 ms) of the target motor unit action potential were not different between the two groups and between C1 and C3. Mean power spectral frequency of the target motor unit action potentials was lower in C3 than in C1 for the C2 group (ANOVA: $F = 13.5$, $P < 0.01$; SNK: $P < 0.01$), whereas it was not different between the two contractions for the control group. The percent decrease in C3 with respect to C1 in the C2 group (6.5 ± 5.1%) was not different from the percent decrease in global mean frequency observed in C2.

**DISCUSSION**

Surface EMG feedback was applied to study changes in electrophysiological properties of inactive muscle fibers after a sustained contraction in ischemic conditions. The main result was that conduction velocity of quiescent motor units significantly decreased with muscle contraction. The decrease in inactive motor unit conduction velocity was not different from the decrease in average conduction velocity of the active motor units. It could be excluded that the observed changes in conduction velocity were only due to ischemia because conduction velocity of the target motor unit did not change in ischemic conditions without muscle contraction (control group).

**Identification of single motor units with surface EMG feedback**

We have previously shown that multi-channel surface EMG is an effective feedback for controlling the activity of single motor units at low contraction levels (2–5% MVC) in normal and ischemic conditions (Farina et al. 2004a,b). This study indicates that the same technique can be applied for the identification of motor units at contraction levels of ~20% MVC on average, in ischemic conditions. To enhance the feedback in a condition of relatively high background activity of other motor units (Fig. 2), we used a two-dimensional surface EMG recording system instead of the one-dimensional array applied in previous studies. This increased the likelihood...
that the subject could identify specific EMG channels in which the discrimination of the target motor unit was possible (Fig. 2). All subjects were able to recruit and derecruit the target motor unit over the entire experimental session.

**Membrane properties of quiescent muscle fibers**

The properties of the target motor unit were analyzed before and after a sustained low force contraction during which it was not active. To enhance the modifications in electrolyte concentrations during low force tasks, the contractions were performed with blood flow occlusion, which avoided metabolite washout. This condition resembles contractions at high force levels, when the intramuscular pressure exceeds the systolic pressure.

The decrease in motor unit conduction velocity between C1 and C3 could not be due to the presence of ischemia by itself because it was not observed in the control group. Moreover, in similar experimental conditions and in the same muscle, con-

**FIG. 3.** A and C: conduction velocity estimates and regression lines during the contractions of a subject of the C2 group and control group, respectively. For C1 and C3, each point in the plot corresponds to the conduction velocity value estimated from a single action potential identified as generated by the target motor unit at a specific time instant. Global conduction velocity estimates (epochs of 1 s) during C2 are shown for the C2 group subject. The subject of the control group rested for 3 min between C1 and C3. B and D: superposition of the action potentials from all identified discharges of the target motor unit in C1 and C3 for the subject of the C2 group and control group, respectively. Similarity of the shapes of the target motor unit potential templates in the 1st and 3rd contraction for all channels of the 2-dimensional array assured that the same motor unit was activated in C1 and C3.
The extracellular environment is modified by the activity of muscle fibers. K\(^+\) concentration affects fiber contractile properties and membrane excitability and is an important determinant of muscle fatigue (Jorgensen et al. 1988; Nielsen et al. 2004; Nordsborg et al. 2003; Sejersted and Sjogaard 2000), in both fast- and slow-twitch muscles (Cairns et al. 1997).

Increased extracellular K\(^+\) concentration decreases action potential conduction velocity (Kossler et al. 1991). In absence of blood flow, K\(^+\) accumulates at a faster rate than with normal circulation (Barcroft and Millen 1939). Blood flow has indeed a main role in the maintenance of the force level during muscle contraction, as shown by the comparison of sustained and intermittent maximal exercises (Pitcher and Miles 1997). Accordingly, larger K\(^+\) extracellular concentrations were observed after high-force isometric contractions with respect to intermittent contractions (Vyskocil et al. 1983). In the ischemic conditions studied, the accumulation of K\(^+\) thus progressed at a faster rate than with normal blood circulation.

Because the fibers of different motor units are intermingled (Brandstater and Lambert 1973; Kugelberg and Edstrom 1968), production of K\(^+\) due to the activity of recruited muscle fibers may influence the membrane properties of quiescent ones. The results of this study provide evidence for this hypothesis in single motor units analyzed in vivo and quantify the effect of muscle activity on nonactive muscle fibers. The percent decrease in conduction velocity of the target motor unit between C1 and C3 was not significantly different from the percent decrease in global conduction velocity during C2. Thus in the conditions analyzed, the main determinant of motor unit conduction velocity decrease was not the rate of activation but the modification of the extracellular environment induced by muscle contraction. It is expected that similar changes in conduction velocity could be detected during contractions with normal blood circulation by increasing the contraction level or the duration of the sustained contraction. Ischemia increased the magnitude of the change and allowed us to appreciate it with relative low contraction force and duration, necessary for a successful surface EMG feedback.

The percent decrease in conduction velocity had a large variability among subjects (Table 1). This was probably due to many factors that could not be controlled. The force level was not fixed but corresponded to the subject self-selected force for optimal feedback. The force exerted was indeed very different among different subjects. Accordingly, there was a large variability in the amplitude of the motor unit action potentials among subjects. During C2, the subjects varied the exerted force to avoid the recruitment of the target motor unit. This was probably the reason why EMG average rectified value decreased during C2. Moreover, it is expected that the effect of the activity of muscle fibers on quiescent ones depends on the relative location of the active motor units with respect to the target one. Due to the low contraction level, there was probably a large variability among subjects in the geometrical arrangement of the active motor units, which contributed to the spread of the values reported in Table 1. For higher force levels, this variability would probably be reduced. Although the percent decrease in conduction velocity in the C2 group had large intersubject variability, the muscle activation had a clear effect on the quiescent fibers, which was not observed in the control group.

### Table 1. Percent change in conduction velocity between contractions C1 and C3 for the 2 subject groups

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<thead>
<tr>
<th>Subject</th>
<th>C2 Group</th>
<th>Control Group</th>
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<tbody>
<tr>
<td>1</td>
<td>−9.8</td>
<td>−0.5</td>
</tr>
<tr>
<td>2</td>
<td>−7.2</td>
<td>−1.8</td>
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<tr>
<td>3</td>
<td>−13.6</td>
<td>−1.8</td>
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<tr>
<td>4</td>
<td>−9.5</td>
<td>−2.1</td>
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<td>5</td>
<td>1.7</td>
<td>5.8</td>
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<tr>
<td>6</td>
<td>−10.1</td>
<td>−3.6</td>
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<td>7</td>
<td>−7.2</td>
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<tr>
<td>8</td>
<td>−15.9</td>
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<tr>
<td>9</td>
<td>2.3</td>
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<td>10</td>
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Negative values correspond to a decrease of conduction velocity in C3 with respect to C1. Eight of the 10 subjects in the C2 group participated in the control session. CV, conduction velocity.
The change in conduction velocity induced similar relative changes in action potential mean frequency as those observed in previous work (Farina et al. 2004a). However, although conduction velocity decreased, the duration of the action potential did not significantly change, whereas it was expected to increase (Lindstrom and Magnusson 1977). This may be due to the variability in the estimation of duration (related to the estimation method) that may have masked the small changes expected (of the order of 6%).

Interpretation of surface EMG during sustained contractions

These findings also have relevance for the interpretation of global surface EMG variables during sustained contraction. Previous studies have shown that average muscle fiber conduction velocity (representative of the mean conduction velocity of all active motor units) initially decreases and then increases during endurance contractions (Gazzoni et al. 2001; Houtman et al. 2003). This was interpreted as due to a change in the recruited motor unit pool. Houtman et al. (2003) hypothesized that the initial conduction velocity decrease was due to the slowing of conduction velocity of the motor units active since the beginning of the contraction, while the subsequent increase revealed progressive recruitment of additional motor units due to fatigue. The results of this study suggest that the increase in average conduction velocity due to additional motor unit recruitment is probably smaller than that expected under the assumption of recruitment of fresh motor units. Thus the quantification of changes in the active motor unit population during sustained contraction, on the basis of the analysis of average conduction velocity time-course, is complicated by the membrane property modifications of the whole motor unit pool.

The change in conduction velocity of quiescent fibers in a submaximal contraction also complicates the interpretation of surface EMG amplitude with fatigue. It has been previously reported that, although EMG amplitude increases during submaximal fatiguing contractions, the amplitude of the surface EMG is significantly less than maximum at the endurance limit (Fuglevand et al. 1993). It was proposed that the reduced EMG amplitude was due to a deficit in the ability to activate the muscle maximally. However, these results underline at least two additional factors in the interpretation of EMG amplitude at the endurance point. Changed membrane properties of all fibers in the muscle may result in modifications of the twitch torque (increased contraction time) with concomitant decrease in discharge rates of additionally recruited motor units for maintaining tetanic fusion with respect to the condition of fresh fibers (Bigland-Ritchie 1981). Moreover, decreased conduction velocity increases surface EMG amplitude cancellation (Farina et al. 2004c; Keenan et al. 2005).

In summary, this study showed that conduction velocity of quiescent muscle fibers decreases during muscle contraction as a consequence of the alteration of the extracellular environment. Thus the electrophysiological properties of muscle fibers change during muscle contraction, partly independently of their activation.

GRANTS

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