Conduction velocity in human muscle
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LONG-LASTING SUPERNORMAL CONDUCTION VELOCITY AFTER SUSTAINED MAXIMAL ISOMETRIC CONTRACTION IN HUMAN MUSCLE


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ABSTRACT

We studied muscle fatigue and recovery on healthy males by means of surface and invasive EMG, on biceps brachii and brachioradialis muscle. Fatigue was induced by 1 min of maximal voluntary contraction (MVC), recovery was studied during 60 min (surface EMG) and 15 min (invasive EMG). The main finding was a long-lasting 'supernormal' muscle fiber conduction velocity (MFCV) during recovery. After a normalization phase of 5 minutes, the MFCV continued to increase reaching a steady state at 'supernormal' values after 10-12 min. Mean MFCV increase at 20% MVC after 15 min recovery 0.58 m.s⁻¹ (12%). The changes in median power frequency paralleled the MFCV increase. Post fatigue integrated EMG (IEMG) values were increased at all contraction levels. We suggest that this IEMG increase is mainly a result of the MFCV increase. In the invasive experimental setup we confirmed the MFCV changes. The relative increase in fastest and slowest fibers indicates an equal effect on type I and II fiber types. A possible explanation is muscle fiber swelling, in combination with altered membrane properties.
INTRODUCTION

In the last 2 decades analysis of the surface EMG has become an important tool in the study of local muscle fatigues. During maximal voluntary contraction (MVC) several changes are observed. The mean integrated EMG (IEMG) shows a gradual decrease in amplitude, preceded by a short increase (Milner-Brown and Miller, 1989). The median power frequency (MPF) shows a rapid shift to the lower frequencies (Naeije and Zorn, 1982; Petrofsky and Lind, 1980; Sadymo and Miyano, 1981; Stulen and DeLuca, 1982). Both central and peripheral factors are suggested to explain these phenomena. Central factors proposed are changes in recruitment of motor units (Arendt-Nielsen et al., 1989; Bigland-Ritchie and Woods, 1984), synchronization of motor units (Datta and Stephens, 1990), decreasing firing frequency (Fuglsang-Frederiksen and Ronager, 1988; Woods et al., 1987) and motor unit firing time statistics (Broman et al., 1985; van Boxtel and Schomaker, 1984). The most important peripheral factors are caused by changes in the muscle fiber conduction velocity (MFCV) (Eberstein and Beattie, 1985; Lindstrom et al., 1970; Milner-Brown and Miller, 1986; Stulen and DeLuca, 1982; Zwarts and Arendt-Nielsen, 1988). During recovery under aerobic conditions a normalization of the MFCV and MPF appears within 5 to 7 minutes (Milner-Brown et al., 1986). However, some authors mention an overshoot of the MPF in the recovery phase some minutes after a fatiguing contraction (Hara, 1980; Zwarts et al., 1987). This phenomenon prompted us to a more detailed study of the MFCV during the recovery phase after maximal contraction by means of surface and invasive EMG techniques.

MATERIALS AND METHODS

All experiments were carried out on healthy males after we had obtained their informed consent. Three approaches were used to investigate the changes in EMG parameters during MVC and recovery. (1a) A surface EMG experiment on 12 subjects. Age 29-40 years, mean 33.6; (1b) an experiment by means of surface EMG on four subjects from the first group with a reduced number of measurements during the recovery period, ages 29-37 years, mean 33.0; and (2) an experiment on the same four subjects, by means of an invasive method for MFCV estimation.

1. Surface method

EMG recording
The biceps brachii was chosen for the experiments since this muscle is easily accessible for MFCV determination according to the technique described by Lynn (Lynn, 1979), and Sollie et al. (Sollie et al., 1985). All measurements were done on the left side. The arm was fixed in a horizontal semiflexed position with an angle of 120 degrees, supported at the elbow and the wrist. The isometric force of the elbow flexion was measured at the wrist with a strain gauge. The exerted force was displayed on a voltmeter in front of the subject and simultaneously recorded on paper. Three silver electrodes (diameter 2 mm) were placed in a rigid bipolar array with a common centre electrode, interelectrode distance 10 mm. Localization was parallel to the fiber direction, nearly half way between the innervation zone and the distal tendon. The skin was cleaned with isopropyl
alcohol and lightly abraded with emery paper. The electrode array was attached with adhesive tape.
The two EMG signals were amplified differentially (Disa EMG amplifier type 14C13) and bandpass
filtered (20-500 Hz). The EMG signals were synchronously digitized by a 12-bit A/D converter
with two different sample rates: 6024 Hz (velocity estimation) and 1024 Hz (power spectra) over
two connected signal periods of 0.34 and 2.05 sec respectively. In order to ensure optimal
localization of the electrodes, a real-time estimation of the resulting MFCV and the cross
correlation coefficient over a quarter of the signal were determined in combination with a graphic
display of the digitized signals.

**TABLE 1.** Mean values and standard error of the mean (SEM) of force, muscle fiber conduction velocity,
surface method (S-MFCV), median power frequency (MPF) and integrated EMG (IEMG) at start and at
end of 1 min maximal voluntary contraction.

<table>
<thead>
<tr>
<th></th>
<th>Start Mean</th>
<th>SEM</th>
<th>End Mean</th>
<th>SEM</th>
<th>R</th>
<th>p'</th>
</tr>
</thead>
<tbody>
<tr>
<td>FORCE (N)</td>
<td>181</td>
<td>(13.0)</td>
<td>93</td>
<td>(6.5)</td>
<td>-0.99</td>
<td>0.0001</td>
</tr>
<tr>
<td>S-MFCV (m.s⁻¹)</td>
<td>4.50</td>
<td>(0.13)</td>
<td>2.94</td>
<td>(0.12)</td>
<td>-0.96</td>
<td>0.0007</td>
</tr>
<tr>
<td>MPF (Hz)</td>
<td>97.05</td>
<td>(6.1 )</td>
<td>45.5</td>
<td>(4.1 )</td>
<td>-0.93</td>
<td>0.0028</td>
</tr>
<tr>
<td>IEMG (µV)</td>
<td>576</td>
<td>(73.3)</td>
<td>460</td>
<td>(32.3)</td>
<td>-0.85</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Linear regression analysis.*

**Data analysis**

Data were analyzed by a microcomputer (PDP 11/23) off line. The conduction velocity (S-MFCV)
was calculated after interpolation, which raises the sample frequency to 12048 Hz. Only correlation
coefficients higher than 0.85 were accepted. The power spectrum was computed over the
frequency range of 5-250 Hz by applying the fast-fourier transform over the digitized signal. The
frequency resolution was 0.5 Hz. After applying the Papoulis window for spectral smoothing this
was reduced to 5.0 Hz. The MPF was calculated from the spectra obtained. In addition, the mean
integrated value of the EMG signal (IEMG) was calculated.

**2. Invasive Method**

Experiments were performed on both the biceps brachii (short head) and on the brachioradialis
muscle. All measurements were performed in resting muscles. The experiments were carried out on
a Nicolet EMG apparatus (Viking I). We used and slightly modified the method of Troni (Troni *et al.* 1983). A stimulation needle electrode (Dantec 13L64, area of uninsulated tip: 2 mm²), was
placed in the distal part of the short head of the biceps brachii muscle or the distal part of the
brachioradialis muscle, 5-15 mm beyond the fascia. A silver surface electrode was used as anode 10
to 15 mm distal. The muscle was stimulated with gradually increasing strength until a clear twitch was palpable (1 - 2 mA, 0.2 msec, 1 Hz). Guided by the twitch, a concentric needle electrode (Dantec 13L50) was placed 5 cm (biceps brachii) or 8 - 10 cm (brachioradialis) proximal and manipulated until reproducible polyphasic action potentials were seen, amplitudes 20 - 500 µV. The signals were amplified and bandpass filtered, 500 Hz - 10 kHz; the time base varied between 5 - 10 msec per division. Care was taken to place the electrodes perpendicular to the skin. The latencies were measured at the positive turning points. A 4-trace storage was used to ensure the reproducibility of the action potentials. Only spikes larger than 20 µV were used for calculations. All latencies were measured, and resulting MFCV (I-MFCV) was calculated. As parameters were used mean MFCV, fastest and slowest MFCV, and ratio of fastest to slowest MFCV (F/S ratio), indicating the range of conduction velocities.

*Figure 1. Mean muscle fiber conduction velocity, surface method (S-MFCV) and Median Power Frequency (MPF) changes during 1 minute maximal voluntary contraction. Bars indicate 1 SEM. Linear regression analysis, S-MFCV slope -0.027 m.s\(^{-2}\) (p=0.0007) and MPF slope -0.85 Hz.s\(^{-1}\) (p=0.0028). Note the more pronounced changes in MPF.*
**PROCEDURE**

**Experiment I (Surface EMG)**

The MVC was determined twice, the highest value was taken as 100% MVC. EMG measurements were performed at the beginning of the experiment and after the recovery period during short contractions (2.5 sec) in duplo at different force levels, i.e. 10-20-30-50-75 and 100% MVC. The mean value at each force level was used for calculations. Fatigue was induced by 1 minute MVC during which 7 measurements were performed. Recovery was studied at brief contractions at 20 and 50% MVC with 15 sec in between: 1, 2, 4, 6, 8, 10, 12, 14, 16, 18, 20, 25, 30, 35, 40, 45, 50, 55, 60 minutes after the MVC. Simultaneously skin surface temperature near the location of the surface electrodes and circumference of the upper arm were measured. During the experiments room temperature was kept above 22° celsius. On 4 subjects the same experiment was performed with measurements during recovery only 15, 30 and 60 minutes after the MVC, in order to study the effect of repeated contractions on the changes in EMG parameters.

### TABLE 2. Mean values (SEM) of muscle fiber conduction velocity, surface method (S-MFCV), median power frequency (MPF) and integrated EMG (IEMG) before fatigue (prefat) and after 14, 30 and 60 min recovery at 20% and 50% maximal voluntary contraction (MVC).

<table>
<thead>
<tr>
<th>% MVC</th>
<th>prefat</th>
<th>recovery (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>30</td>
</tr>
<tr>
<td>20</td>
<td>4.08 (0.14)</td>
<td>4.66 (0.22)</td>
</tr>
<tr>
<td>50</td>
<td>4.16 (0.11)</td>
<td>4.67 (0.19)</td>
</tr>
<tr>
<td>20</td>
<td>99.0 (7.0)</td>
<td>113.5 (8.3)</td>
</tr>
<tr>
<td>50</td>
<td>100.4 (5.6)</td>
<td>108.2 (6.2)</td>
</tr>
<tr>
<td>20</td>
<td>89.2 (12.7)</td>
<td>94.2 (15.4)</td>
</tr>
<tr>
<td>50</td>
<td>271.1 (37.3)</td>
<td>362.1 (49.3)</td>
</tr>
</tbody>
</table>

*Significant difference with prefatigue values, Wilcoxon's signed rank test, paired, 2-tailed.
Experiment II (Invasive EMG)
The first trials were performed before fatigue. Afterwards a cuff on the upper arm was inflated above the systolic blood pressure (>200 mm Hg). The subjects were asked to maintain a maximal isometric contraction of the muscle involved during 1 minute. Just after the contraction a new measurement was performed during ischemia. A slight reposition of the needle was sometimes necessary. Afterwards the cuff was released, which marked the beginning of the recovery phase. Measurements were performed after 1, 2, 4, 6, 8, 10, 12 and 15 minutes of recovery. On the first two occasions biceps brachii was tested on 2 subjects. The rest of the experiments were performed on the brachioradialis muscle of all 4 subjects, because of the cuff on the upper arm these measurements were easier to perform.

TABLE 3. Mean values (SEM) of force, integrated EMG (IEMG) and neuromuscular efficiency (NME): force / IEMG before fatigue (prefat) and after 60 min recovery at maximal voluntary contraction.

<table>
<thead>
<tr>
<th></th>
<th>prefat</th>
<th></th>
<th>recovery</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>FORCE (N)</td>
<td>199</td>
<td>(13.8)</td>
<td>187</td>
<td>(12.6)</td>
</tr>
<tr>
<td>IEMG (µV)</td>
<td>603</td>
<td>(77.1)</td>
<td>704*</td>
<td>(80.8)</td>
</tr>
<tr>
<td>NME</td>
<td>0.39</td>
<td>(0.06)</td>
<td>0.30*</td>
<td>(0.03)</td>
</tr>
</tbody>
</table>

*Significant difference from prefatigue values, Wilcoxon's signed rank test, paired, 2-tailed.
Statistical analysis
The change in force, MFCV, MPF and IEMG during 1 min MVC was studied with linear regression analysis. Wilcoxon’s signed rank test (paired samples, 2-tailed) was used to test the difference of means. Statistical significance was accepted at a level of 5%. We studied recovery curves using nonlinear regression analysis.

Figure 3. Recovery of muscle fiber conduction velocity, surface method (S-MFCV) and median power frequency (MPF) during 60 minutes after 1 minute maximal voluntary contraction (MVC). Contraction level 20% MVC. Time zero: last value during 1 minute MVC. Mean values, SEM. Non linear regression analysis, exponential association. S-MFCV recovery $T_{1/2}$=2.1 minute, $R^2$=1.00, MPF recovery $T_{1/2}$=1.4 minute, $R^2$=0.99. Dotted lines: Prefatigue value at the same force level.
RESULTS

Experiment I: Surface EMG

Fatigue Phase
During 1 minute MVC the force declined rapidly, almost linearly. The mean remaining force after 60 sec was 54% of the value at the start of the experiment, mean slope $-1.4 \text{ N.s}^{-1}$ (p=0.0001). The S-MFCV and MPF also showed a rapid decline (fig 1), this effect was most pronounced in the case of the MPF (table 1). S-MFCV slope $-0.027 \text{ m.s}^{-2}$ (p=0.0007) and MPF slope $-0.85 \text{ Hz.s}^{-1}$ (p=0.0028). The IEMG showed a slight increase, and after 20 sec a steady decrease until the end of the contraction (fig 2), slope $-2.6 \mu \text{V.s}^{-1}$ (p=0.02).

Recovery Phase
During recovery S-MFCV and MPF reached precontraction values within 4-6 minutes both followed by an overshoot in all subjects, at both force levels tested. A steady state was reached after 10 - 14 minutes. The changes in MFCV and MPF persisted during the remaining registration time. The mean values after 14, 30 and 60 minutes are given in table 2. The two parameters (S-MFCV and MPF) showed nearly the same relative increase, with a slight tendency to decrease after 45 minutes (fig 3). At 20% and 50% MVC the changes became significant (p < 0.05) after 6 minutes compared to the prefatigue values at the same force level. The recovery curves at 20% (50%) MVC followed an exponential association (fig 3). S-MFCV recovery $T_{1/2}=2.1$ (1.5) minute, $R^2=1.00$ (1.00), MPF recovery $T_{1/2}=1.4$ (1.3) minute, $R^2=0.99$ (0.99). IEMG values stabilized after 15-20 minutes and remained higher than the initial values both at 20% and 50% contraction level. Skin temperature showed a maximum 4-6 minutes after the fatigue phase with a normalization to prefatigue values after 16-18 minutes. The circumference of the upper arm showed an increase in all subjects, maximum 8.0 SD 1.8 mm after 2 min recovery, followed by a gradual decrease (fig 4). The changes remained significant until the end of the registration time. The force and IEMG changes are summarized in table 3. After one hour recovery time the force had not completely recovered. The IEMG showed higher post fatigue values at all contraction levels (fig 5), with an even higher value at MVC in all subjects, mean increase 17%. This results in a reduced neuro muscular efficiency (NME), calculated by dividing force by IEMG. Reducing the number of measurements during the recovery phase, resulted in the same increase of the S-MFCV and MPF after 15 and 30 minutes, but a tendency to decrease to normal values after 60 minutes at both force levels tested (fig 6).

Experiment II: Invasive EMG

After 1 minute MVC the mean MFCV had decreased significantly, following the same pattern in biceps brachii and in brachioradialis. For calculation the data of both muscles were summarized. Mean values before vs. after fatigue were $3.43 \text{ m.s}^{-1}$ (SD=0.16) and $2.84 \text{ m.s}^{-1}$ (SD=0.31) (table 4). During the recovery phase a rapid return to prefatigue values was seen after 4-5 minutes, followed
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by an overshoot which stabilized after 10-12 minutes (figs. 7 and 8). The recovery curve followed an exponential association, $R^2 = 0.995$, $T_{1/2} = 2.9$ minute. The mean MFCV value after 15 minutes was 3.93 m.s$^{-1}$ (SD 0.14). The MFCV values were significantly higher than the prefatigue values. The changes in MFCV were most pronounced in the case of the fastest fibers measured. Maximal MFCV changes of fastest fibers 1.32 m.s$^{-1}$ (SD=0.27) and of slowest fibers 0.93 m.s$^{-1}$ (SD=0.14). The F/S ratio showed no significant changes.

**DISCUSSION**

**Fatigue Phase**

In our surface experiment, during 1 minute MVC we found a simultaneous decrease in force, MPF and MFCV. The changes were most pronounced in the MPF. A comparable decrease in force and surface EMG parameters is well known in literature (Eberstein and Beattie, 1985; Hara, 1980; Stulen and DeLuca, 1982; Zwarts et al. 1987).

*Figure 4. Changes of surface temperature ($\Delta$ Temp) and changes of the circumference of the upper arm ($\Delta$ circumference) during 60 minutes recovery after 1 minute maximal voluntary contraction. Mean values, SEM. Dotted lines: Prefatigue values.*
The results of the invasive measurements are interesting. Through the stimulation of a muscle fiber bundle with a fixed frequency central factors which can influence the MFCV are excluded. A spectrum of conduction velocities can be found with a rather fixed range, which can be expressed as a ratio: fastest divided by the slowest (Zwarts, 1989). It can be assumed that these potentials represent the MFCV of type I and II fibers, the latter being the fastest. Earlier we have found that this method gives comparable results both in biceps brachii and in brachioradialis muscle (van der Hoeven and Zwarts, 1990). In our invasive experiment we found a clear decrease of MFCV during 1 minute MVC. It is obvious that the whole complex is delayed (fig 8). Since both fiber types are supposed to be excited, this indicates that both type I and type II fibers are prone to the same MFCV disturbing effect. Although the MFCVs of the fastest fibers decline most, the relative changes, on the basis of the F/S ratio, are similar for fast and slow fiber types.

The decrease of MFCV during MVC is assumed to be caused mainly by the rapid accumulation of metabolic by-products, especially lactate. At these high force levels the muscle is working under anaerobic conditions, due to the high intramuscular pressure, which obstructs the local blood flow (Hagberg, 1981; Rohmert, 1960). The increasing lactate concentration results in a pH decrease and subsequently a lowering of the membrane potential. Type II muscle fibers were most sensitive to this effect (Tesch et al. 1983). The declining membrane potential results in a slowing down of the MFCV (Sahlin and Ren, 1989). However, on the basis of our I-MFCV results it is likely that relatively both type I and II fibers are equally prone to this MFCV disturbing effect. This suggests that the so-called 'fatigue resistant' type I fibers (Burke et al. 1973) are equally affected by fatigue, at least with respect to the MFCV under ischemic conditions. This is in

![Figure 5. The relationship between integrated EMG (IEMG) and force level (as percent of initial maximal force). Circles: prefatigue values. Squares: values after 60 minutes recovery. Bars: SEM. Note the higher IEMG postfatigue at all force levels, including maximal force.](image)

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agreement with the findings of Sahlin et al. (1987). They concluded that the soleus of the rat, mostly composed of type I fibers, cannot be termed fatigue resistant under anaerobic conditions since the decrease of isometric twitch tension did not differ from fast twitch motor units.

Recovery Phase

During the recovery phase in both experimental settings we found a quick return to pre-fatigue values within about 4-6 minutes, with an overshoot to higher 'supernormal' values, which reached a steady state after 10-14 minutes. The surface EMG experiment showed that this effect can be seen both in S-MFCV and in MPF and that it lasts as long as 60 minutes, at both force levels tested. The same effect can be seen in our invasive experiment. The changes observed are most pronounced in the fastest fibers measured, but the relative changes, expressed as F/S ratio, did not change significantly. This indicates that the increase in MFCV concerns both fiber types to the same relative extent. The equal increase in S-MFCV and MPF suggests that the overshoot observed in MPF is mainly due to the increase in MFCV.

Contrary to the mechanisms underlying surface EMG changes during fatigue, the recovery phase after fatiguing contraction receives relatively little attention. Most investigations were stopped at the moment that 'baseline' values had been obtained. In a series of surface EMG experiments on elbow flexors Hara (1980) found a recovery of the 'slow wave proportion' within 5-6 minutes after MVC until exhaustion. Remarkable was an overshoot in the recovery phase; a decrease in low frequency content (20-40 Hz) and an increase of high frequency content (100-150 Hz) of the EMG in nearly all subjects. The time course of this 'overshoot' was not mentioned. Other changes observed were a decreased maximum force until 20 minutes recovery time, in combination with an increased value of the IEMG. These facts are in agreement with our finding of a long-lasting 'overshoot' in MPF.

TABLE 4. Mean values of muscle fiber conduction velocity, invasive method (I-MFCV), slowest and fastest conducting fibers before (prefat), after 1 min maximal voluntary contraction (fatig) and after 15 min of recovery

<table>
<thead>
<tr>
<th></th>
<th>prefat</th>
<th>fatig</th>
<th>recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>m.s^-1</td>
<td>m.s^-1</td>
<td>m.s^-1</td>
</tr>
<tr>
<td>I-MFCV</td>
<td>3.43</td>
<td>2.84'</td>
<td>3.93'</td>
</tr>
<tr>
<td>slow</td>
<td>2.98</td>
<td>2.51'</td>
<td>3.43'</td>
</tr>
<tr>
<td>fast</td>
<td>4.00</td>
<td>3.22'</td>
<td>4.54'</td>
</tr>
</tbody>
</table>

'Significant difference with prefatigue values, Wilcoxon's signed rank test, paired, 2-tailed.'
The long-lasting 'supernormal' MFCV could be attributed to various factors. Changes in MU discharge frequency can account for MFCV variations (Nishizono et al. 1989). However, the constant stimulation rate as used in the invasive EMG setup eliminates this hypothetical effect as a causative factor. The same holds true for explanations on the basis of central factors such as recruitment changes. Temperature changes influence MPF, IEMG and, probably, MFCV (Petrofsky and Lind, 1980). The skin temperature values, which, according to Saltin and Hermansen (1966), reflect central and muscle temperature changes, in our experiment normalized within 16 - 18 minutes. This makes a major temperature effect on the changes in MFCV observed unlikely. Alternative explanations are:

1. Increase in muscle fiber diameter on the basis of fiber swelling resulting in a decreased internal resistance and, hence, a higher conduction velocity.
2. Long-lasting changes in membrane properties influencing the capacitance and / or resistance.

Bouissou et al. (1989) suggested the first explanation on theoretical grounds: 'the increasing water content of muscle during intense dynamic exercise in combination with the well-known dependence of MFCV on muscle fiber radius'. However, hitherto experimental evidence for this hypothesis has been lacking. Sahlin et al. (1978) found an increased water content in human muscle after exercise until exhaustion. The increase was divided equally over the intracellular and extracellular space. Sjogaard et al. (1985) also demonstrated an increased water content in human muscle; after maximal exercise, the largest increase occurred in intracellular water. In a fatigue study in rats Peeze Binkhorst et al. (1989) found an 28% increase in cross-sectional area 6 hours

\[ \text{Figure 6. Relative changes of muscle fiber conduction velocity, surface method (S-MFCV) at 20} \% \text{ MVC during 60 minutes recovery. Circles: frequent measurements. Filled squares: measurements only after 15, 30 and 60 minutes recovery. Dotted line: prefatigue value. The two experiments were performed on the same 4 subjects.} \]
after 1 hour submaximal exercise, although they could not correlate this finding with an increased water content. This fiber swelling returned to normal after 24 hrs. MFCV was not measured in their experiments. Our finding of an increased circumference of the upper arm also supports this hypothesis.

Nandedkar et al. (1985) use a formula for simulation purposes; the propagation velocity is calculated for each muscle fiber as a linear function of the fiber diameter by the formula

\[ \text{propagation velocity (m.s}^{-1}) = 2.2 + 0.05 \times (\text{diameter} - 25) \, (\mu\text{M}) \]

Applying this formula on the slowest and fastest I-MFCV results gives a hypothetical fiber diameter range for the slowest and fastest conducting fibers: 41 to 61 µM. These numbers are roughly matching the experimental data of Polgar et al. (1973) in a postmortem study. Using this formula on the basis of our experimental I-MFCV data would result in a hypothetical fiber diameter increase: slowest conducting fibers 41 to 50 µM, fastest fibers 61 to 72 µM, relative diameter increase 25% and 18%. Resulting cross sectional area increase 49% and 39%. These figures are higher than the experimental findings of Peeze Binkhorst et al. (1989) who found an increase in cross sectional area of 28% in rat muscle. Therefore, an additional effect as a result of changes in membrane properties seems likely.

Interestingly, Hicks et al. (1989) found M wave potentiation shortly after maximal isometric contraction in human hand and foot muscles, followed by a gradual decrease. In an additional experiment in rat muscle in vivo, they related this phenomenon to increased sodium

![Figure 7. Fatigue and recovery phase of mean muscle fiber conduction velocity, invasive method (I-MFCV) before and after 1 minute maximal voluntary contraction and during 15 minutes recovery, all subjects. Triangles: biceps brachii, circles: brachioradialis. Solid line: mean values. Nonlinear regression analysis of mean values, exponential association. \( R^2=0.995 \), \( T_{1/2}=2.9 \) minute. Dotted line: Prefatigue level.](image-url)
pump activity, resulting in a membrane hyperpolarization. The hyperpolarization started to decline after 9 minutes, but was still present after 15 minutes (Hicks and McComas, 1989). Such a rise in membrane potential could also result in a higher MFCV.

Metzger and Fitts (1987) studied the time course of recovery. After a high frequency stimulation protocol in isolated rat diaphragm muscle, they found a pH recovery time to resting levels after about 10 minutes. Juel (1988) studied MFCV recovery in isolated mouse muscle, and found an action potential propagation velocity recovery curve similar to the intracellular pH recovery. Boska et al. (1990) using a $^{31}$P magnetic resonance spectroscopy (MRS) technique, also found in human muscle in vivo a pH normalization in 10 - 12 minutes after sustained MVC. Our finding of MFCV recovery reaching a steady state after 10-12 minutes is therefore likely to follow the pH recovery time course.

The time course of 'normalization' of the supernormal MFCV is not completely clear. However, the normalization time during recovery in the experiment with a reduced number of measurements takes about 60 minutes. Additionally this suggests that repeated contractions play a role in maintaining the effect.

In a combined EMG $^{31}$P nuclear magnetic spectroscopy experiment Miller et al. (1987) found 3 phases of recovery after 1-4 minutes MVC in adductor pollicis muscle. The M-wave

Figure 8. Examples of normal, reduced and supernormal muscle fiber conduction velocity, invasive method (I-MFCV) on one subject. Two traces superimposed. Calibration 200 µV / division, timebase 5 ms. A. Prefatigue situation. B. After 1 minute maximal voluntary contraction, ischaemia. C. 15 minutes recovery. Differences in signal aspect are due to slight needle repositions between the various measurements
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returned to normal within 4 minutes. High-energy phosphates, pH and force recovered within 20 minutes. They also found an increased IEMG signal, resulting in a decrease in Neuro Muscular Efficiency (force / IEMG) which did not recover within 60 minutes. They measured the IEMG at short contractions at 50% MVC, and do not give values at 100% MVC. The observed increase of IEMG was related to motor unit recruitment and synchronization, and the long-lasting decrease in NME to impaired excitation-contraction coupling. At lower force levels the increased IEMG could indeed be due to central factors such as increased motor unit recruitment, synchronization and a firing frequency increase because of tiredness. However, this does not hold true for maximum force, since in that case recruitment and motor unit firing frequency are assumed to be already maximal. Nevertheless, a synchronization effect cannot be excluded. Theoretical studies predict an increased power of the EMG signal with higher conduction velocities, at least at a relatively large distance, as in the case of surface EMG (Dimitrov et al. 1988). At great radial distances the total power is almost linearly related to propagation velocity (Lateva, 1988). Therefore, it is likely that the increased IEMG is also an effect of the increase of MFCV. In that case the IEMG is not an independent variable.

In conclusion, we found a clear reduction of MFCV during fatigue, using surface and invasive methods. The reduction in MFCV seems to affect both type I and II fibers equally. During recovery we found a long-lasting supernormal muscle fiber conduction velocity with both methods. The reported overshoot in MPF was confirmed, and seems mainly dependent on the increase in MFCV. To our knowledge, a long-lasting supernormal conduction velocity has not been reported in literature before. We found a simultaneous increase in IEMG as well. It is partly related to the changes in MFCV. We stress that due to these changes, the IEMG cannot be regarded as an independent factor. This implies that fatigue studies which are based on amplitude measurements of the surface EMG are prone to the disturbing effect of changes in signal properties, which makes the interpretation less straightforward than generally is accepted.

REFERENCES

Chapter 3

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