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Oxygen availability and motor unit activity in humans

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Summary. Six men were studied to determine the interrelationships among blood supply, motor unit (MU) activity and lactate concentrations during intermittent isometric contractions of the hand grip muscles. The subjects performed repeated contractions at 20% of maximal voluntary contraction (MVC) for 2 s followed by 2-s rest for 4 min with either unhindered blood circulation or arterial occlusion given between the 1st and 2nd min. The simultaneously recorded intramuscular MU spikes and surface electromyogram (EMG) data indicated that mean MU spike amplitude, firing frequency and the parameters of surface EMG power spectra (mean power frequency and root mean square amplitude) remained constant during the experiment with unhindered circulation, providing no electrophysiological signs of muscle fatigue. Significant increases in mean MU spike amplitude and frequency were, however, evident during the contractions with arterial occlusion. Similar patterns of significant changes in the surface EMG spectra parameters and venous lactate concentration were also observed, while the integrated force-time curves remained constant. These data would suggest that the metabolic state of the active muscles may have played an important role in the regulation of MU recruitment and rate coding patterns during exercise.

Key words: Motor unit - Recruitment - Rate coding - Metabolism - Electromyogram frequency power spectra

Introduction

Since the size principle of Henneman et al. (1965) was first proposed for results from cat motoneurons, strong evidence has been presented that in muscle contraction there is a specific sequence of recruitment in order of increasing motoneuron and motor unit (MU) size (Milner-Brown et al. 1973; Freund et al. 1975; Kukulka and Clamann 1981; De Luca et al. 1982). It has been well documented that MU recruitment and firing frequency (rate coding) depend primarily upon the level of force and the speed of contraction. When low-threshold MU are recruited, this has resulted in a muscle contraction characterized by low force-generating capabilities and high fatigue resistance. With requirements for greater force and/or faster contraction, high-threshold-fatiguable MU have been recruited (Freund et al. 1975; Henneman and Mendell 1981). However, Kukulka and Clamann (1981) and Moritani et al. (1986a) have demonstrated in human adductor pollicis muscle that for a muscle group with mainly type I fibres, rate coding played a more prominent role in force modulation. For a muscle group composed of both type I and II fibres, MU recruitment seems to have been the major mechanism for generating extra force above 40% to 50% of maximal voluntary contraction (MVC; De Luca et al. 1982; Kukulka and Clamann 1981; Moritani et al. 1986a; Moritani and Muro 1987).

During cycling exercise, an increase in plasma lactate concentration has been shown to be already apparent at approximately 50%-70% of the rate of maximal oxygen uptake ($\dot{V}O_{2max}$) and well before the aerobic capacity is fully utilized (Jorfeldt et al. 1978; Wasserman et al. 1985; Sahlin et al. 1987). During an incremental exercise test on a cycle ergometer, however, Sjogaard (1978) has shown that the peak force for a pedal thrust was less than 20% MVC at exercise intensities eliciting $\dot{V}O_{2max}$ at 60 rpm. Despite these relatively low force outputs and moderate speeds of contraction during cycling, glycogen content of types IIa and IIb fibres has been found to be progressively decreased (first in type IIa and finally type IIb fibres) during prolonged submaximal exercise (Vollestad et al. 1984). Furthermore, Gollnick et al. (1974) have also shown that in isometric contractions, slow twitch units are the only ones to be depleted of glycogen at force developments up to 15%-20% MVC. Above this level, fast twitch units are also depleted of glycogen. This would suggest that with this type of muscle contraction the availability of oxygen and the developed force has influenced recruitment of fast-twitch units

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since blood flow has been shown to be usually restricted during sustained contractions of about 20% MVC (Humphreys and Lind 1963). Thus, these available data, would suggest that not only the force and speed of contraction, but also the availability of oxygen may affect the recruitment of high threshold MU.

The purpose of the present study was, therefore, to determine the inter-relationships among oxygen availability, motor unit activity and blood lactate concentrations during intermittent isometric contractions of the handgrip muscle.

Methods

Subjects and experimental protocols. Six male subjects volunteered for this study. They were fully informed about the nature of the experiments including possible risks and gave their written informed consent. The subject was in a prone position on a table. The maximal voluntary grip strength was then measured three times with a modified grip strength dynamometer previously described (Moritani et al. 1984) and the highest value was recorded as maximal voluntary grip strength (MVC). In the control situation (CON), the subject performed repeated intermittent isometric contractions at 20% of MVC (2-s contraction followed by 2-s rest period) for 4 min. In the experimental situation (EXP), the subject performed the identical repeated intermittent isometric contractions for 4 min but with the circulation occluded between the 1st and 2nd min and given unhindered circulation during the last 2 min (Fig. 1). The arterial occlusion was induced by a pressure cuff around the upper arm which was inflated to 200 mmHg (26.7 kPa) within 4 s by a cuff inflator. Care was taken to eliminate movement of the upper body by tightly strapping down the subject's chest and lower torso to the table. Venous blood samples were obtained from a catheter inserted proximally into the brachial vein. Samples were taken every 30 s during CON and at every 15 s during EXP. Blood was analysed for lactate enzymatically in duplicate and averaged. The two experimental conditions were chosen at random interspersed by 30 min.

Force and electromyogram recordings. During the whole experiment, the force signal was amplified through a DC amplifier (Grass 7P122, Massachusetts, USA) and displayed on a digital oscilloscope (Kikusui 5020A, Tokyo, Japan) placed within view of the subject for visual feedback. The level and duration of the target force were marked on the oscilloscope. The force and EMG signals were visually and continuously monitored by the experimenter for any artefacts by means of a Tektronix 4-channel, dual

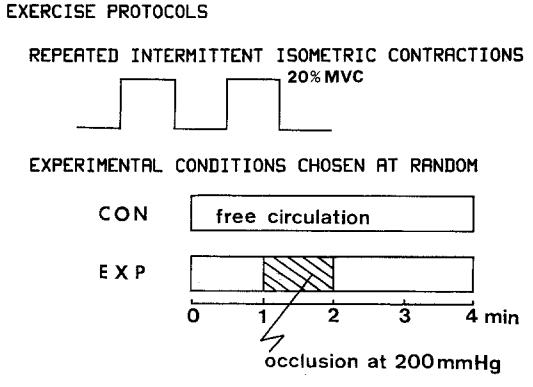

Fig. 1. Diagram of experimental conditions and exercise protocol. *MVC*, Maximal voluntary contraction; *CON*, control; *EXP*, experimental conditions

time storage oscilloscope (Tektronix, Texas, USA). The force signal was digitized at a sampling rate of 100 Hz via an A-D converter. The surface EMG and intramuscular spike signals were digitized at sampling rates of 1 and 20 kHz, respectively, with 16-bit fast A-D converters paced by precision pulse train cards and stored on a floppy disk by the use of an HP 9836 desk-top computer (Hewlett Packard, Colorado, USA). The digitized force data were then processed off line for every 15 s for calculation of the force-time integral by the computer.

Surface EMG and intramuscular spike analyses. Our EMG instrumentation and analysis procedures have been described elsewhere in more detail (Moritani et al. 1985, 1986b). Briefly myo-electric signals were recorded from bipolar surface electrodes (6-mm pick-up diameter with 2-cm interelectrode distance) placed over the belly of the flexor carpi radialis-palmaris longus muscle. Intramuscular MU spikes were also recorded from high impedance bipolar fine wire electrodes inserted approximately 1.5 cm under the skin. After correcting for DC offset, the simultaneously digitized surface EMG signals and intramuscular spikes were displayed on a computer screen for pattern recognition by the experimenter and then subjected to further data processing.

To quantify the surface EMG signal amplitude and frequency components, analyses of the frequency power spectra were performed. For this purpose, a series of 2048 ms EMG data, time-locked to the onset of force production associated with contraction, were processed with the Hanning window function and 512 point fast Fourier transform (FFT) to obtain power spectra. Thus, each EMG power spectrum corresponding to each contraction was used to determine the mean power frequency (MPF) and the root mean square (rms) value of the signal amplitude. Similarly, the time-locked intramuscular MU spikes were isolated by a window discriminant subroutine and the total number of spikes and mean intramuscular MU spike amplitude were thus determined. These data were averged for every 15 s for each subject during the 4-min intermittent isometric contractions under CON and EXP, respectively. The data were pooled for the purpose of allowing statistical inferences.

Statistical analysis. The results were analysed using the Student's *t*-test for paired data. A significance level of $P \leq 0.05$ was chosen.

Results

Lactate concentrations and force changes

Group data on lactate concentrations and force-time integral changes are presented in Fig. 2. During CON, venous plasma lactate concentrations showed a slight initial increase which was followed by an almost constant value of approximately 1.9 $mmol \cdot l^{-1}$. During EXP, however, the lactate concentrations showed large and statistically significant increases of approximately 3 $mmol \cdot l^{-1}$, shortly after the release of the arterial occlusion and remained significantly elevated during the rest of EXP. In contrast, no significant changes in the force-time integrals were observed throughout the 4-min intermittent isometric contractions either during CON or during EXP (Fig. 2).

Changes in MU activities

A typical set of changes in the intramuscular MU spike recordings is shown in Fig. 3. Only 1-s samples of MU

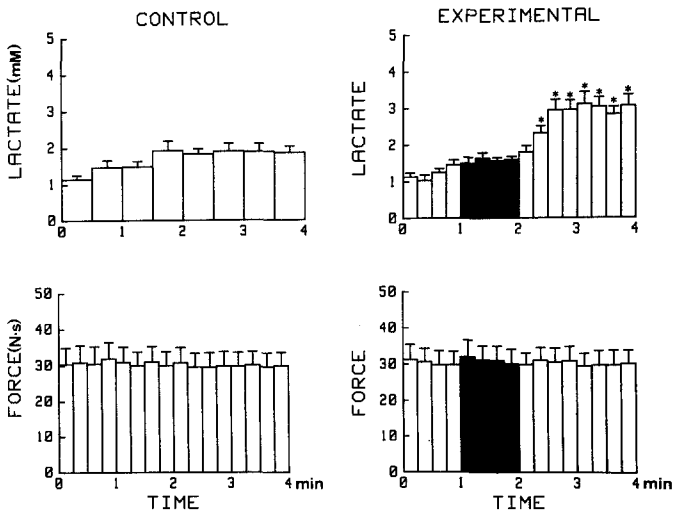

Fig. 2. Changes in force-time integrals and blood lactate concentrations (mean and SEM, $n=6$) during experiments with unhindered circulation (control) and arterial occlusion given between 1st and 2nd min (experimental)

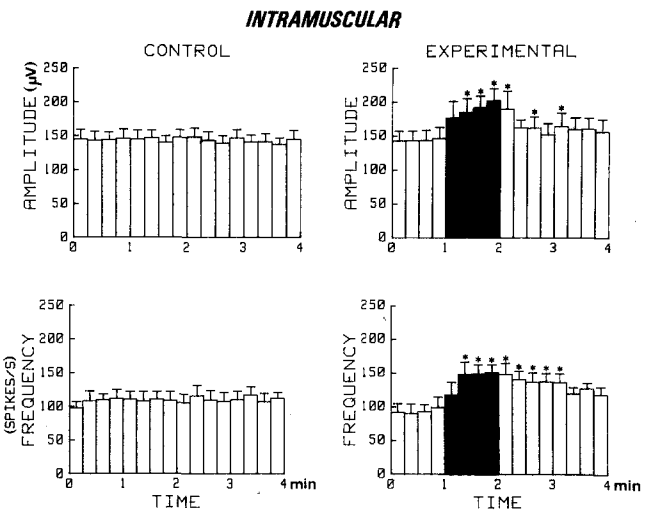

Fig. 4. Group data (mean and SEM, $n=6$) showing the changes in motor unit spike amplitudes and firing frequencies observed during unhindered circulation (control) and arterial occlusion (experimental) trials

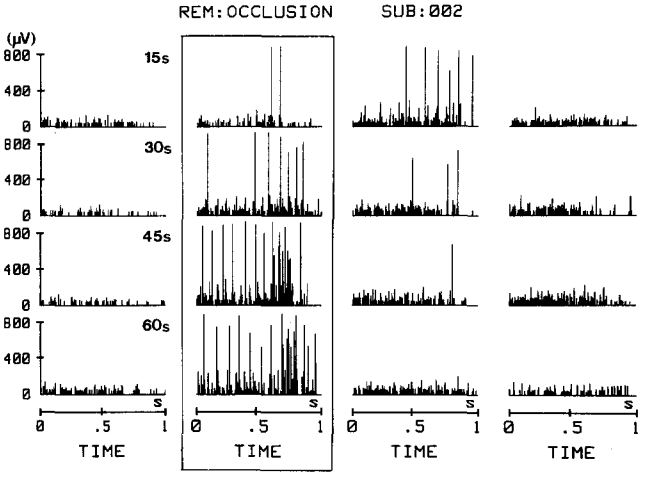

Fig. 3. A typical set of changes in the intramuscular spikes observed during experimental conditions (unhindered circulation plus occlusion between 1st and 2nd min). In this figure only 1-s samples of motor unit spikes obtained at every 15-s interval (raw data) are given for clarity. Thus, in this figure *each column* represents 1 min of repeated intermittent isometric contraction

spikes obtained at every 15-s interval are presented for clarity. During EXP, a consistent pattern of MU activity with similar low-amplitude spikes could be observed prior to the arterial occlusion (Fig. 3, left panel and first column top to bottom). There were, however, large and progressive increases in both MU spike amplitude and firing frequency during the period of arterial occlusion (Fig. 3, second column top to bottom), suggesting new MU recruitment and increased MU discharge rate of relatively high-threshold units. It is noteworthy that the large MU spike amplitudes observed during the occlusion were still seen even 30 s after the complete release of occlusion (e.g. Fig. 3, third column top three rows). By the end of the 3rd min (1 min after occlusion release), the large MU spikes had disappeared and only the low-amplitude MU spikes similar to those observed at the beginning of the experiment without occlusion seemed to be involved. During CON, on the other hand, no such spontaneous changes in MU amplitude and spike frequency were observed, but the analysis showed almost constant MU activities with low-amplitude spikes throughout the entire 4 min of intermittent isometric exercise (Fig. 4). These finding were consistent and invariant features in all the subjects tested.

Group data on mean MU spike amplitude and firing frequency (number of MU spikes per second) are shown in Fig. 4. Nearly constant MU amplitude and firing frequency were maintained throughout the entire period of exercise during CON. There were, as expected, no significant differences in the MU spike amplitude and frequency between CON and EXP prior to the arterial occlusion. However, significant increases in both MU spike amplitude and firing frequency were observed not only during the period of contractions with the occlusion, but also during the period of recovery up to 1 min from the release of the occlusion (Fig. 4). During the 3rd and 4th min into recovery, the mean MU spike amplitude and frequency were still greater after occlusion than during CON but these differences were not statistically significant.

Surface EMG changes

Figure 5 gives group data on the results of the analyses of the surface EMG power spectra during CON and EXP. The rms value of EMG amplitude and MPF showed no systematic changes and stayed almost constant at the end of the 2nd min until the end of the experiment during CON. During EXP the relatively constant rms values observed at the beginning of the experiment, however, showed progressive and significant in-

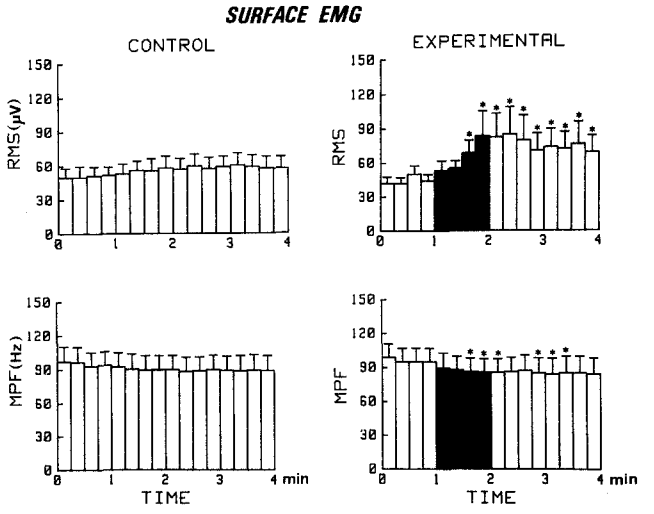

Fig. 5. Group data (mean and SEM, $n=6$) showing the changes in surface electromyogram (*EMG*) amplitude (root mean square, *rms*) and mean power frequency (*MPF*) during unhindered circulation (control) and arterial occlusion (experimental) trials

creases during the last 30 s of occlusion and reached quite high values (approximately twofold increase) for the next 60 s. These rms changes were followed by a progressive decline but the differences in the rms values between CON and EXP were still significant even at the end of the 4-min period (Fig. 5, top right). The MPF data also indicated similar results in that the relatively stable MPF values observed during CON and prior to the occlusion during EXP decreased significantly during the last 30 s of occlusion and remained depressed throughout the rest of the experiment.

Discussion

In the present study, the constancy of both intramuscular MU spikes and surface EMG activity during isometric contraction indicated no electrophysiological signs of muscle fatigue when the circulation was unhindered. However, significant changes in these parameters were evident during contractions with arterial occlusion. Since the availability of oxygen and blood borne substrates, e.g. glucose and free fatty acids, were severely reduced during occlusion, a progressive recruitment of additional MU might have taken place to compensate for the deficit in force development (Bigland-Ritchie et al. 1986; Moritani et al. 1986b). This may have occurred if MU had become depleted of glycogen (Gollnick et al. 1974; Vollestad et al. 1984) or affected by some degree of intramuscular acidification (Wilkie 1986; Metzger and Moss 1987). This in turn would have interfered with the excitation-contraction coupling with a subsequent decrease in the force developed.

During a wide range of submaximal contractions not all of the available MU pool is recruited. We have demonstrated that there was a progressive decrease in MPF of the surface EMG signal during sustained contractions at 50% of MVC but this decline was accompanied by a significant increase in the rms value of the EMG amplitude and a progressive MU recruitment as evidenced by an increased number of MU with relatively large intramuscular spike amplitudes (Moritani et al. 1986b). These results and the present findings of significant increases in MU firing rate and MU spike amplitude associated with arterial occlusion thus suggest that not only the force and speed of contraction but also the availability of oxygen may have affected the recruitment of high threshold MU. In good agreement with this hypothesis, the ^{31}P nuclear magnetic resonance experiments (Bylund-Fellenius et al. 1981) have demonstrated that the rate of recovery in phosphocreatine:inorganic phosphate during the contraction was dependent on oxygen delivery. These results and recent evidence (Idström et al. 1985; Katz and Sahlin 1987) would suggest a causal relationship between oxygen supply and energy state in the contracting as well as in recovering skeletal muscles. In consideration of this evidence, it might be suggested that oxygen availability may have played an important role in regulating MU recruitment and firing frequency as there has been shown to exist a close link between the state of energy supply and the types of muscle fibres being recruited (Idström et al. 1985; Bigland-Ritchie et al. 1986; Moritani et al. 1985, 1986b).

At present, the specific messenger to which the motor control system respond with varying patterns of MU recruitment and firing frequency has not yet been clearly established. There is some evidence that the order of MU recruitment might be modified by changes in the proprioceptive afferent activity (Grimby and Hannerz 1976; Garnett and Stephens 1981; Kirsch and Rymer 1987). The observed large and progressive increases in both MU spike amplitude and firing frequency during the period of arterial occlusion would suggest new MU recruitment and an increased MU discharge rate of relatively high-threshold units. This might thus have been the result of such changes in the proprioceptive afferent inputs of the affected muscle fibres to the motoneurons as a consequence of severely reduced oxygen availability and/or a strong pressure applied during the arterial occlusion. However, the large MU spike amplitudes seen during the occlusion were still seen, even after the complete release of occlusion, for more than 30 s. Results from a recent study (Rotto and Kaufman 1988) have also shown that very large concentrations of sodium lactate (much larger than the lactate changes we observed) were required to stimulate group III and IV muscle afferent nerves. Therefore, any effects of increased pressure or metabolic by-products on the group III or IV afferent nerve fibres that might modify MU recruitment patterns could not have been major mechanisms for the increased MU recruitment and firing frequency of relatively high-threshold units.

Other and possibly more likely explanations might be that the stretch receptors in muscle spindles and Golgi tendon organs have signalled the need for adding more MU (Enoka and Stuart 1985; Nelson and Hutton 1985; Hutton and Nelson 1986; Kirsch and Rymer 1987) as a result of a decrease in the contractility of some MU affected by the reduced oxygen supply and/or depletion of

intramuscular glycogen stores (Gollnbick et al. 1974; Bylund-Fellenius et al. 1981; Vollestad et al. 1984; Idström et al. 1985). Or there may have been some influences on the motoneuron pool activity from the sensory afferent nerve fibres originating in the "metaboreceptors" (Mahler 1979; Sjogaard 1990).

The nonsignificant increase of venous blood lactate concentration during the period of arterial occlusion is very difficult to explain, particularly in the absence of muscle lactate data. It can only be speculated that the venous blood samples obtained during arterial occlusion might not have represented the time course of changes of muscle lactate concentration. The significant increases in blood lactate concentration after the release of occlusion could then be explained as a result of muscle lactate being washed out after the restoration of blood flow.

In summary, our data indicated alteration of MU firing and recruitment patterns during contractions with ischaemia. It is suggested that the metabolic state of the active muscles may have played an important role in the regulation of MU recruitment and rate coding patterns during exercise.

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