Metabolic and myoelectrical effects of acute hypoxaemia during isometric contraction of forearm muscles in humans: a combined $^{31}$P-magnetic resonance spectroscopy–surface electromyogram (MRS–SEMG) study

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1. Failure of muscle force during sustained fatiguing contraction is associated with myoelectrical and metabolic alterations. However, the inter-relationships between these two types of events remain unclear. The purpose of this study was to examine the effects of decreased oxygen availability during sustained contraction on myoelectrical and metabolic changes, thereby addressing the issue of fatigue.

2. $^{31}$P-Magnetic resonance spectra and surface electromyograms were simultaneously recorded in six subjects (three women and three men) performing isometric contraction of forearm flexor muscles sustained at 60% maximum value of force under aerobic or acute hypoxaemic conditions (inhalation of a gas mixture containing 12% O2).

3. The 5 min hypoxaemic rest preceding contraction did not affect the phosphocreatine level and pH value. Under both conditions of oxygen availability, the magnitude of metabolic changes remained similar and the duration of contraction was unaffected (similar workload). However, hypoxaemia significantly reduced the rate of changes in integrated surface electromyogram activity measured in the high-frequency band. Correlative analysis of magnetic resonance spectroscopy and surface electromyogram data shows that for a given surface electromyogram change, metabolic variations were always larger under hypoxaemic conditions.

4. These results suggest that hypoxaemia does not alter metabolic changes, i.e. decrease in pH and phosphocreatine during static contraction. The downward shift of the relationships between myoelectrical and metabolic changes under hypoxaemia points to the existence of a better excitation–contraction coupling in acute hypoxaemia compared with normoxia and this is indicative of an adaptive mechanism.

INTRODUCTION

Recordings of surface electromyograms (SEMGs) are commonly used to analyse the electrical activity of skeletal muscle groups during sustained fatiguing contractions [1–5]. It has been well documented that failure of muscle force during sustained contraction at high sustained force level or dynamic efforts against loads is associated, at least under normoxic conditions, with a shift of the SEMG power spectrum towards lower frequencies. This shift has been defined either from a leftward shift of the median power frequency in SEMG spectrum [2] or from a fall in the ratio of SEMG energies in high- (H) and low- frequency (L) bands [6]. Peripheral and central factors have been proposed to mediate this phenomenon. Since the first study reported by Miller et al. [7], several studies have analysed the possible relationships between metabolic and myoelectrical parameters throughout fatigue trials [8–11] but no conclusive results have been reported. More recently, from a correlative analysis of metabolic and SEMG changes recorded on the tibialis muscle, Vestergaard Poulsen et al. [12] reported an increased myoelectrical activity after static contraction and interpreted it as an impairment of the excitation–contraction coupling. However, these authors did not measure the transmission of myopotentials and thus no information was available on muscle membrane excitability. We have previously demonstrated that the energetic status of muscle and especially intracellular acidosis was unlikely to be the mediator of SEMG alterations and the disconti-
nuity of muscle contraction throughout isometric contraction [8]. Interestingly, it has been proposed that these typical SEMG changes, recorded throughout the fatigue process, could be mediated by the activation of group III–IV muscle afferents (called metaboreceptors) relaying the information from the peripheric muscle to the central nervous system [13]. Also, it is known that low arterial oxygen partial pressure (hypoxaemia) is a potent stimulus for the activation of metaboreceptors in animals [14]. It has been shown previously in humans that acute hypoxaemia affected SEMG changes in respiratory and skeletal muscles [15] probably as a result of enhanced reflex pathway elicited by the activation of muscle metaboreceptors. On the contrary, the metabolic consequences of hypoxaemia still remain controversial [16–19] and determinants of myoelectrical alterations remain unknown.

In the present study we performed a comparative analysis of metabolic and SEMG changes during static contractions performed under normoxic or hypoxaemic conditions. In the latter condition, fatigue trials began after a stable hypoxaemia was obtained. We aimed to obtain additional information on the possible association between SEMG and metabolic changes recorded at the onset of fatigue and to simultaneously analyse the effects of reduced oxygen availability on electrical and metabolic activity of muscle. Data on fatigue-induced SEMG and metabolic changes under normoxic conditions have been reported previously [8]. All metabolic changes were assessed by 31P magnetic resonance spectroscopy (MRS) in vivo.

MATERIALS AND METHODS
Subjects
The study was conducted on the dominant forearm of six healthy volunteers (three females and three males). Subjects were not involved in any arm training and had no physical limitation to exercise. Their age ranged from 34 to 50 years old (mean 40.6 years). The informed consent of the participants was obtained for the study which was approved by the local Ethics Committee.

Exercise protocol
During training sessions, performed several days before actual MRS studies, the subjects were asked to quickly adjust the maximal value of force developed by their finger flexor muscles of the forearm. Maximal force measurements were repeated until three reproducible values were sustained for 1 s. Fatigue trials consisted of isometric contractions sustained at 60% of the respective maximal value of force for as long as possible while subjects were verbally encouraged. Otherwise, force was kept constant during the entire duration of contraction which coincided with MR data acquisition, i.e. 22 s blocks. Visual feedback was provided to the subjects by feeding the force transducer signal output (Schenck LY 11, Germany) into an analog voltmeter. Fatigue trials were performed on separate days under aerobic and hypoxaemtic conditions and were repeated twice by each subject. One to three days elapsed between experiments and the first trial was always performed under normoxic conditions. Hypoxaemia was produced by inhalation, via a face-mask, of a hypoxaemic gas mixture (12% oxygen in nitrogen) and isometric contractions began after a 5 min period of inhalation of the hypoxaemic gas mixture.

MRS
MR spectra were recorded at 4.7 T on a Bruker 47/30 Biospec system equipped with a horizontal superconducting magnet (bore diameter: 30 cm) operating at 81.15 and 200.14 MHz for 31P and 1H detection respectively. The subjects sat on a chair by the magnet and introduced their arm horizontally into the magnet bore. A 50 mm double-tuned surface coil was positioned over the belly of the finger flexor muscles of the forearm which was placed approximately at the same height as the shoulder to ensure a good venous return. Optimization of field homogeneity was done by monitoring the 200.14 MHz signal from the muscle water and fat protons. 31P-MRS data were acquired after 55 μs radiofrequency pulses applied at 93 ms intervals. Spectra were time-averaged over 22 s (24 scans, sweep width = 6000 Hz, 4 × 10⁸ data points collected) and sequentially recorded during 88 s of rest and throughout the fatigue trials. A 15 Hz line-broadening function was applied before Fourier transformation. A micropipette filled with a solution of phenyl phosphonic acid was positioned at the surface coil centre to accurately monitor global changes in spectral intensity.

MRS data processing
After Fourier transformation and deconvolution of free induction decays using a line broadening of 15 Hz, spectra were transferred to an IBM RISC 6000 workstation and processed using the NMR1 spectroscopy processing software (New Methods Research, Inc., Syracuse, U.S.A.) as described previously [8]. Relative concentrations of each phosphorylated metabolite were expressed, after correction with appropriate saturation factors, as percentage of area of the phosphocreatine (PCr) resonance measured at rest and arbitrarily set to 100%. Intracellular pH was calculated from the chemical shift of Pi relative to PCr at −2.45 ppm with respect to 85% H3PO4 [20]. Due to the fast sampling of data, the first MR spectrum was discarded to eliminate intensity distortions due to T1.
relaxation effects. The values measured at rest were averaged over the three sequential spectra recorded during the last 66 s of the resting period preceding the contraction phase. During contraction, slopes of curves describing PCr changes were calculated with all the experimental points recording during exercise. Slopes of pH changes were calculated with all points except the first data point of contraction which was generally higher than the resting value.

SEMG measurements

SEMG activity was recorded from two small silver/silver chloride electrodes (DISA 13L20, U.S.A.) placed over the belly of the flexor digitorum longus muscle. Inter-electrode distance was 10 mm. Amplification of SEMG signals was obtained with a bandwidth of 10–50 Hz and 100–500 Hz for low (L) and high (H) frequencies respectively. The choice of bandwidth was guided by data from studies in humans [2, 15]. In both H and L bandwidths, SEMG signal was half-wave rectified. A moving time average (time constant: 100 ms) allowed us to integrate the signals, simultaneously giving the values of SEMG energy in H and L bands as well as the calculation of H/L ratio. H and L SEMG energies and the force (force transducer, Schenck LY 11) were continuously recorded on a Gould ES 1000 polygraph (Gould, France) throughout the sustained contractions. The mean values of H, L and H/L ratio measured for the first 3 s of sustained contraction were considered as initial conditions in each trial. SEMG variables were measured every 10 s and their values were expressed as a percentage of the corresponding initial data. Changes in H, L and H/L values versus the time were fitted to exponential curves. Regression was obtained through transformed data using least-squares methods. According to previous studies, correlation coefficients (R) of the regression analysis were always higher when exponential regression was used as compared with linear regression [5, 6]. In the present study, R values were always higher than 0.8. The absolute values for the time constant of the decay rate of SEMG variables (TcΔH, TcΔL and TcΔH/L) were used as measurements of the rate of frequency shift in SEMG power spectrum, analogous to the fatigue index already described [6].

Statistics

Several analyses of variance have been performed using General Linear Models. The General Linear Models procedure of the SAS software (SAS Institute Inc., U.S.A.), options REPEATED, LSMEANS and CONTRAST were used. For each parameter, two-way analyses of variance with repeated measures (the repeated variables being time and oxygen condition) have been performed. Post-hoc Wilk's λ-tests were used to analyse the effects of oxygen availability for each parameter. For each condition of oxygen availability, the correlation between SEMG and metabolic parameters was examined using simple regression. The significance level for testing hypotheses was fixed to 0.05.

RESULTS

Normoxic fatigue trial

The mean duration of isometric contraction was 85±3 s (Table 1). Values of maximal force developed by flexor digitorum contraction under normoxic conditions ranged between 23 and 43 kg and they were significantly (P<0.05) higher in males (37±4 kg) than in females (25±2 kg). The pattern of quantitative SEMG changes during sustained contraction was as previously described in the same muscle group [6], namely energy in the low-frequency (L) band linearly increased while SEMG activity integrated in the high-frequency (H) band first increased and then tended to decrease during the last 22 s of the contraction (Fig. 1A). This resulted in a weak decrease in H/L ratio at the end of contraction. Time constants (Tc) of changes in H, L and H/L ratio are summarized in Table 2 and time-dependent SEMG changes during sustained contraction, under normoxia and hypoxaemia, are presented in Figs 1A and 1B.

Table 1. Morphometric characteristics of subjects and duration of sustained forearm contractions

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Body weight (kg)</th>
<th>Duration of exercise (s), Normoxic (Hypoxaemia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>44</td>
<td>50</td>
<td>88 (88)</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>33</td>
<td>53</td>
<td>88 (88)</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>42</td>
<td>52</td>
<td>66 (88)</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>51</td>
<td>67</td>
<td>88 (110)</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>45</td>
<td>75</td>
<td>88 (88)</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>45</td>
<td>85</td>
<td>88 (88)</td>
</tr>
<tr>
<td>Mean ± SE</td>
<td></td>
<td></td>
<td></td>
<td>85 ± 3 (90 ± 4)</td>
</tr>
</tbody>
</table>

Intracellular pH (pHi) transients increased from 6.96±0.03 at rest to 7.01±0.06 at the onset of contraction (Fig. 2). Then, pHi as well as PCr decreased linearly throughout the trials (Figs 1C and 1D). When contraction ended, the PCr content was 49±5% of its initial level and the pH value had reached 6.78±0.05.

Figure 2 shows the graphical plots of SEMG changes and metabolic variations throughout sustained contraction. Correlations between the time constants of SEMG changes and the slopes of pH changes (P = 0.08 for H band and 0.14 for L band) or the slopes of PCr changes (P = 0.09 for H band and 0.22 for L band) were not statistically significant. These results suggest that electrical and metabolic changes are dissociated during the fatigue trial.
Hypoxaemic fatigue trial

The mean duration of sustained contraction was 90 ± 4 s and was not affected by hypoxaemia. Otherwise, the workload was not significantly different between hypoxaemic and normoxic fatigue trials. Compared with normoxic fatigue, acute hypoxaemia mainly affected SEMG changes whereas contraction-induced metabolic variations were roughly similar. We previously determined that PaO₂ was rapidly and significantly lowered after inhalation of a hypoxaemic gas mixture whereas Paco₂ did not change [15]. During the 5 min hypoxaemic period before isometric contraction, PCr level and pH value did not vary significantly. At the end of this rest period,

![Graph A](image1)

![Graph B](image2)

![Graph C](image3)

![Graph D](image4)

**Table 2. MRS and SEMG indices characterizing individual responses to isometric contraction of forearm flexor muscles sustained at 60% of maximal force under normoxic and hypoxaemic conditions.** *MRS parameters measured at end of exercise. **Time constants (Tc) of changes in high (H) SEMG and low (L) SEMG frequency bands and H/L ratio, expressed in s⁻¹. Results are presented as means ± SEM. Statistical significance: *P < 0.05.*

<table>
<thead>
<tr>
<th>Subject</th>
<th>ΔPCr (%)</th>
<th>Rate of PCr decrease</th>
<th>pH*</th>
<th>Rate of PCr decrease</th>
<th>TcH** (s⁻¹)</th>
<th>TcL** (s⁻¹)</th>
<th>TcH/L** (s⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normoxia</td>
<td>Normoxia</td>
<td>Normoxia</td>
<td></td>
<td>Normoxia</td>
<td>Normoxia</td>
<td>Normoxia</td>
</tr>
<tr>
<td>1</td>
<td>32 (52)</td>
<td>–9.4 (–4.9)</td>
<td>6.95 (6.94)</td>
<td>–0.06 (–0.04)</td>
<td>30 (61.5)</td>
<td>34 (67)</td>
<td>30 (43.5)</td>
</tr>
<tr>
<td>2</td>
<td>60 (55)</td>
<td>–16.8 (–10.7)</td>
<td>6.69 (6.60)</td>
<td>–0.1 (–0.14)</td>
<td>47 (30)</td>
<td>65 (35)</td>
<td>200 (58)</td>
</tr>
<tr>
<td>3</td>
<td>77 (70)</td>
<td>–8.9 (–20.8)</td>
<td>6.52 (6.70)</td>
<td>–0.25 (–0.11)</td>
<td>13 (47)</td>
<td>20 (43)</td>
<td>22 (241)</td>
</tr>
<tr>
<td>4</td>
<td>42 (45)</td>
<td>–11.6 (–10.8)</td>
<td>6.83 (6.82)</td>
<td>–0.06 (–0.08)</td>
<td>49 (62)</td>
<td>102 (85)</td>
<td>49 (95)</td>
</tr>
<tr>
<td>5</td>
<td>60 (40)</td>
<td>–12.9 (–11.6)</td>
<td>6.82 (6.85)</td>
<td>–0.13 (–0.07)</td>
<td>39 (74.5)</td>
<td>41 (65)</td>
<td>126 (149)</td>
</tr>
<tr>
<td>6</td>
<td>53 (65)</td>
<td>–13.4 (–21.6)</td>
<td>6.64 (6.78)</td>
<td>–0.10 (0.04)</td>
<td>41 (36)</td>
<td>45 (41)</td>
<td>55 (85)</td>
</tr>
<tr>
<td>Mean ± SE</td>
<td>47 ± 5 (55 ± 3)</td>
<td>–12 ± 1 (–13 ± 3)</td>
<td>6.76 ± 0.05 (6.78 ± 0.05)</td>
<td>–0.12 ± 0.03 (–0.08 ± 0.02)</td>
<td>35 ± 4 (53 ± 6)</td>
<td>49 ± 9 (56 ± 7)</td>
<td>70 ± 18 (114 ± 22)</td>
</tr>
</tbody>
</table>
Metabolic and myoelectrical study of muscle contraction

PCr level (100 ± 1%; mean ± SE) and pH (6.99 ± 0.01) were not significantly different from the control values measured in normoxia. When contractions were performed under hypoxaemic conditions, the time course of PCr consumption and pH decrease was not significantly different to the changes recorded under normoxic conditions (Figs 1C and 1D), although a trend towards reduced metabolic changes was noted for hypoxaemic contraction. In contrast, hypoxaemia induced an overall decrease in SEMG energy during sustained contraction and modified the pattern of SEMG changes throughout fatigue trials (Fig. 1): the rate of EH increase was significantly reduced as evidenced by a decreased time constant of ΔEH (Table 2) while the time constant of ΔEL did not vary. In addition, no decline in EH at the end of the fatigue trial was noticed in contrast to the observation made in normoxia. There was a downward shift of the relationships between SEMG changes and metabolic indices (Fig. 2). Thus, for a given magnitude of SEMG changes, the extent of metabolic variations, i.e. magnitude of PCr changes and intracellular acidosis, was always larger under hypoxaemic than under normoxic conditions.

DISCUSSION

The present data show that acute reduction of oxygen supply to forearm muscles during sustained static contraction did not exert any apparent effect upon exercise-induced metabolic variations. However, SEMG changes were markedly attenuated and a downward shift of the relationships between SEMG and metabolic changes was noted, indicating an increased energy consumption for the same myoelectrical activity.

Hypoxaemia and SEMG changes

SEMG provides a reliable measure of muscle activity during voluntary contractions. Figure 2 shows that increased activation of motor units is necessary to maintain a constant level of force as described previously [21]. However, despite the same workload being sustained under normoxic and hypoxaemic conditions, the rate of increase in SEMG energy in the high-frequency band was reduced in hypoxic muscle. These observations are similar to that previously reported for the adductor pollicis and the diaphragm executing inspiratory

Fig. 2. Correlated evolution of quantitative SEMG changes in the high- or low-frequency bands and pH (A, B) or PCr content (C, D) during fatigue trials performed under normoxic (○) and hypoxic (●) conditions. Results are presented as means ± SEM.
manoeuvres in hypoxaemic individuals [15]. Failure of muscle force during rhythmic or sustained contraction at a high force level is classically associated with a shift of SEMG power spectrum towards lower frequencies. The rate of decrease in median power frequency as well as the fall in the ratio of SEMG activities integrated in high- and low-frequency bands (H/L ratio) are indices commonly used to measure quantitative SEMG changes during high intensity tasks. The changes in muscle fibre conduction velocity or in compound muscle mass action potential (M-wave), two indices used to analyse the sarcolemmal depolarization, are peripheral factors which could partly explain the changes in SEMG power spectrum during fatiguing contractions. Some authors reported no change in sarcolemmal transmission of myopotentials under normoxic [2] as well as hypoxic conditions [22], whereas others found a significant decrease in normoxia [7, 23] or ischaemia [24, 25]. Reduction of SEMG by hypoxaemia during isometric contractions sustained at constant force level corroborates previous data by our group [15] under the same experimental conditions and also those reported by Kayser et al. [26] who showed that 1 month acclimatization to 5050 m altitude induced a severe reduction in total integrated SEMG variations at a given workload. Such a reduction in SEMG changes during sustained contraction was not found in hypoxaemic patients suffering from chronic respiratory insufficiency [27]. Peripheral and central mechanisms may be responsible for the hypoxia-induced decrease of SEMG variations. One of the peripheral mechanisms may be an increased efficiency of excitation–contraction coupling by acute hypoxaemia. Also, alteration of motor unit recruitment in a hypoxic muscle, i.e. reduced activation of fast fibres, may be associated with less ATP used per unit force resulting in an increased efficiency. Alterations of SEMG changes measured in hypoxic muscle may also result from a control mechanism originating in the central nervous system. Indeed, the central nervous system can modulate the firing rate of exercising muscle based on oxygen availability [28]. This may result from a direct influence of hypoxaemia on the motoneuron firing rate. However, acute moderate hypoxaemia is more often responsible for increased neuronal excitability, the depressor effects occurring only when hypoxaemia is severe. As already documented [14], hypoxaemia may also modulate the activity of spinal motoneurons through changes in spontaneous activity of group III–IV muscle afferents and muscle spindles. In parallel, muscle hypoxia as well as muscle fatigue attenuate the response of muscle mechanoreceptors to muscle contraction or high-frequency vibrations [14, 29]. It is well known that the afferent path of muscle proprioceptors exerts excitatory influences on α motoneurons. Thus, it may be hypothesized that the combination of increased inhibitory influences carried by group III–IV muscle fibres and reduced facilitatory proprioceptive pathways should considerably depress the discharge of α motoneurons when muscles get tired under hypoxic conditions, accentuating the muscle wisdom phenomenon (defined as the decline in motor unit discharge rates during contraction in relation to slowing of relaxation). Recent data obtained with in vitro preparations of rat soleus, extensor digitorum longus and diaphragm have demonstrated that quantitative SEMG analysis allows an estimate of motor unit recruitment and thus of muscle fibre type composition [30]. We showed in normal individuals executing isometric tasks that hypoxaemia affected, in different ways, the changes in EH and EL in different muscle groups [15]: in adductor pollicis, a muscle comprising a majority of fatigue-resistant and oxygen-dependent ST fibres, hypoxaemia reduced predominantly the rate of EL changes but not that of EH, whereas in the diaphragm, a mixed muscle group, hypoxaemia depressed both EH and EL variations during isometric contractions. Since flexor digitorum muscles contain approximately 40% ST fibres and 60% FT fibres [31], the present data of a prominent depressor effect of hypoxaemia on the rate of EH changes seem logical. Thus, SEMG changes under hypoxaemia seem to be related to the relative prevalence of ST and FT fibres. The entire significance of fatigue-induced control of myoelectrical activity remains unclear. Based on our present observations and in agreement with previous studies [12], it seems unlikely, at least under aerobic conditions, that the changes in energetic state of muscle are responsible for the reflex control of muscle contraction through the activation of muscle metaboreceptors.

**Hypoxaemia and metabolic changes**

From a metabolic point of view, our data reveal that the extent of PCR breakdown and intracellular acidosis measured throughout sustained contraction performed under normoxic and hypoxaemic conditions were similar. The metabolic consequences of lowering PaO2 on exercising muscle are still contradictory. If reduction of oxygen supply to exercising muscle is suspected to turn metabolism towards anaerobic energy production (leading to lactate production), enhanced lactate production is not unanimously reported. In humans, hypobaric hypoxaemia (4000 m) slightly increased the blood lactate concentration at maximal work [32] whereas in normobaric hypoxaemia (12% O2 concentration) the peak blood lactate at Vmax does not vary [33]. In contrast, in dogs, acute hypoxaemia markedly increases the release of lactate from contracting gastocnemius muscle [19]. The magnitude of PCR changes reflects the balance between ATP breakdown during muscle contraction and ATP synthesis by aerobic and anaerobic pathways. Also, PCR decline is associated with proton consumption thereby accounting for the buffering effect of creatine kinase. The extent of intra-
cellular acidosis provides, taking into account the proton consumption associated with PCr hydrolysis, a quantitative index of anaerobic ATP production. The existence of similar metabolic changes induced by the same muscle workload imposed under normoxia or hypoxaemic conditions could then be accounted for by several mechanisms. First, it is noteworthy that sustained isometric contraction is associated with local muscle ischaemia due to reduced blood flow, a direct consequence of increased intramuscular pressure [34, 35]. This augmentation of intramuscular pressure suggests that at a high level of isometric workload the muscle already works with a reduced oxygen supply in normoxia. Our results further suggest, in agreement with Kemp et al. [36], that due to this local ischaemia, activation of oxidative ATP production is small, at least during the initial period of contraction and that for a given workload, metabolic changes are independent of oxygen availability. Also, the slow increase in oxygen consumption and heart rate during arm exercise compared with leg exercise, further confirms the magnitude of anaerobic energy production even under normoxic conditions [37]. Jensen-Urstad et al. [37, 38] also previously demonstrated modest effects of hypoxaemia upon oxygen consumption and lactate release during arm exercise. Considering the effects of \( \beta \)-adrenoceptor blockade upon lactate release in exercising muscles, they determined that a reduced oxygen supply to muscle (due to hypoxaemia) can be compensated by an increase in muscle blood flow [38]. Finally, Stainsby et al. [19] have previously reported that isotonic tetanic contractions of dog \textit{gastrocnemius-plantaris} muscle group performed under hypoxaemia were associated with reduced developed tension. This reduction was coupled to decreased oxygen consumption suggesting that hypoxaemia probably leads to a shutdown of muscle mechanical performance thereby accounting for a reduction of oxygen demand [19]. Our results did not show any reduction of mechanical performance associated with hypoxaemia. However, the SEMG profile evidenced a reduction of electrical activity. This strongly points to the existence of a better excitation–contraction coupling in acute hypoxaemia compared with normoxia, a phenomenon already shown in other skeletal muscles and diaphragm [15].

In conclusion, the main effect of hypoxaemia under the experimental conditions used in this study is a reduction of the magnitude of SEMG changes without obvious effect upon metabolic parameters such as PCr consumption and/or extent of acidosis. Other stimuli beside the fall in muscle pH seem to be responsible for the activation of muscle afferents during contraction. Animal data suggest that the rapid release of potassium from contracting muscle [39] and also the production of arachidonic acid [40] and prostaglandins [41] may activate group III–IV muscle afferents during static contraction. From the results of the present study we can hypothesize that hypoxaemia could have led to an adaptive mechanism resulting in reduced activation of motor units and overall diminution of muscle metabolism. Under those conditions, the extent of metabolic changes would have been larger if SEMG activation had been similar to what was recorded under normoxia. From this comparative analysis it can be inferred that metabolic and SEMG activities are not directly linked during static contraction, whereas the correlated evolution of these activities is modulated under hypoxic conditions, and reflects an adaptive mechanism.

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