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Research article

INFLUENCE OF POSTURE ON PULMONARY O_2 UPTAKE KINETICS, MUSCLE DEOXYGENATION AND MYOLECTRICAL ACTIVITY DURING HEAVY-INTENSITY EXERCISE

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ABSTRACT

The aim of the present study was to test the hypothesis that compared to upright posture, slower oxygen uptake (VO_2) kinetics resulting from exercise at the same relative metabolic load in the supine posture will be associated with increased muscle de-oxygenation and greater myoelectrical activity. Nine subjects completed one 12-min heavy-intensity constant-load exercises in each of the supine and upright postures on an electronically braked cycle ergometer at a same gain in metabolism per unit increase in work intensity (10.8 ± 1.3 vs. 11.8 ± 1.1 $mlO_2 \cdot min^{-1} \cdot W^{-1}$ in upright and supine, respectively) on separate days. Breath-by-breath VO_2 kinetics were analyzed with a double exponential model to characterize the primary and slow component phases. Myoelectrical activity (RMS) of the vastus lateralis (VL), rectus femoris, and biceps femoris muscles was recorded at different epochs of the exercise. Oxygenation of the VL muscle was recorded continuously by near-infrared spectroscopy. In supine compared with upright cycling, the primary time constant of VO_2 kinetics was significantly increased (32.7 ± 10.7 s vs. 23.5 ± 6.7 s, respectively) while the absolute magnitude of VO_2 slow component was decreased ($p < 0.05$) but not the relative amplitude. VL de-oxygenation was higher ($p < 0.05$) in supine cycling throughout the exercising period whereas RMS values for all muscles did not change appreciably over time. Our findings suggest that lowered oxygen supply induced by supine heavy exercise, alters oxidative metabolism dynamics and increases muscle de-oxygenation. However, cycling supine did not increase markedly the rate of muscle fatigue.

KEY WORDS: muscle perfusion, heavy cycling exercises, NIRS, VO_2 slow component.

INTRODUCTION

Changes in muscle oxygen delivery and muscle perfusion are known to affect force or power output of the muscle (Hepple, 2002; Wright et al., 1999). When blood flow is diminished both muscle endurance and oxygenation decrease, leading to muscle fatigue (Tachi et al., 2004). Motor unit firing and recruitment patterns have been showed to be altered during ischemia (Moritani et al., 1992), suggesting that the lack of oxygen availability increases motor unit discharge rate of high-threshold units (i.e. which are more likely to lead to a fatigue state). Limb elevation or muscle heart configuration-related changes are known to engender circulatory changes leading to a decrease in perfusion pressure (Wright et al., 1999) and muscle fatigue (Lind et al., 1978; Tachi et al., 2004). The postural effects on the rate of muscle fatigue are thus induced by changes in the perfusion pressure (Eiken, 1988; Fitzpatrick et al., 1996). A recent study of Egana and Green (2005) demonstrated that the endurance of the calf

muscle was improved when the body was tilted from the horizontal to an incline posture. This indicated that the fatigue resistance was mediated by an increase in blood flow when the body was tilted up at least for static exercises.

In a different context, diminished perfusion pressure induced by position-related changes has been shown to decrease the oxygen supply and alter the oxidative metabolism dynamics as reflected by a slower pulmonary oxygen uptake (VO_2) kinetics (Convertino et al., 1984; Hughson et al., 1991) for moderate-intensity constant-load test in supine compared to upright postures at the same absolute workload. Gravity adds about 40-50 mmHg perfusion pressure to the arterial supply of the quadriceps muscles in the upright posture, and therefore could have important implications in blood flow regulation during exercise. Based on that, the hypothesis advanced in the literature is that, muscle blood flow is inadequate at the onset of supine compared to upright exercise at the same power output (Folkow et al., 1971; MacDonald et al., 1998). However, to our knowledge, no study has examined the relationships between pulmonary VO_2 and local muscle de-oxygenation kinetics during supine and upright heavy exercise and their influences on the rate of quadriceps muscle fatigue for a same relative metabolic load. The question about how fast the ATP demand can be covered under different body posture changes for the same relative metabolic demand and its effects on muscle function remains unclear. We hypothesized that supine cycling exercise will slow the VO_2 kinetics and will increase muscle de-oxygenation and myoelectrical activity, leading to a possible earlier fatigue occurrence.

During heavy-intensity exercise, an additional slow component of VO_2 kinetics is superimposed on the primary VO_2 response (Barstow et al., 1996; Cleuziou et al., 2005; Koga et al., 1999). It is well admitted in the literature that the exercising limbs are considered as the major determinants of the VO_2 slow component (Poole et al., 1991). The observed increase in muscle deoxygenation signal over the slow component period suggests that the local balance between muscle delivery and utilization was altered during the slow component, and is consistent with the origin of the VO_2 slow component being predominately in the exercising muscle (Delorey et al. 2005, Bringard et Perrey, 2004). However, to our knowledge, the balance between local muscle O_2 delivery and utilization during the slow component of VO_2 with different muscle perfusion (e.g. supine vs. upright postures) have not been studied, except in part by Koga et al. (1999). However as ventilatory threshold (VT) and maximal VO_2 are significantly higher in upright compared to supine cycling exercise (Koga et al., 1999) and because VO_2 slow component occurs for exercise intensities above VT (Gaesser and Poole, 1996), a given absolute workload performed in supine cycling will be relatively lower when performed in upright cycling. As a result, a higher slow component will occur in supine heavy exercise. Relative metabolic load is more important in determining the VO_2 kinetics features (e.g. magnitude of the slow component, Gaesser and Poole, 1996) rather than the workload *per se* (Perrey et al., 2001b). If supine exercise is really associated with a relative perfusion inadequacy to the working muscles (Hughson et al., 1993; Leyk et al., 1994), this should be exacerbated during supine heavy exercise even at a high metabolic load. Therefore, the second aim of this study was to test the hypothesis that the supine exercise would be associated with a larger slow component (and a higher rate of muscle fatigue shown by EMG signal changes) compared with the upright exercise at a same relative metabolic load.

METHODS

Subjects

Nine trained male subjects volunteered to take part in this study. Their mean (SD) values for age, height, and body mass were 25.8 (3.3) years, 1.76 (0.06) m, and 67.2 (8.1) kg, respectively. The study protocol complied with the Helsinki declaration for human experimentation. All the experimental procedures used in this study were in accordance with the standards of the Local Ethical Committee on Human Experimentation. Possible risks and benefits were explained and written informed consent was obtained from all subjects after a full explanation of the protocol. None of them suffered from muscle soreness, knee injury, or peripheral vascular disorder. Subjects were asked to avoid caffeine intake within the 8 hours preceding the tests. The subjects were all fully familiar with laboratory exercise testing procedures.

Experimental design

In order to standardize the exercising posture, supine and upright cycling exercises were performed with the same distance separating the hip of each subject to the crank shaft axis of the cycle ergometer. Feet position on the pedals was also maintained identical in both exercises. For supine and upright tests, subjects were asked to not grip their hands and not use their arms to avoid any upper body contribution during the exercise.

The tests were performed on an electromagnetically braked cycle ergometer

(Ergoline, Ergoselect 100 P, Germany). In supine cycling, the crank shaft was positioned 33 cm above the level of the heart. A home made apparatus was used to lock subjects' shoulders and prevent from possible rear movements inherent to the forces applied on the pedals (see Figure 1).

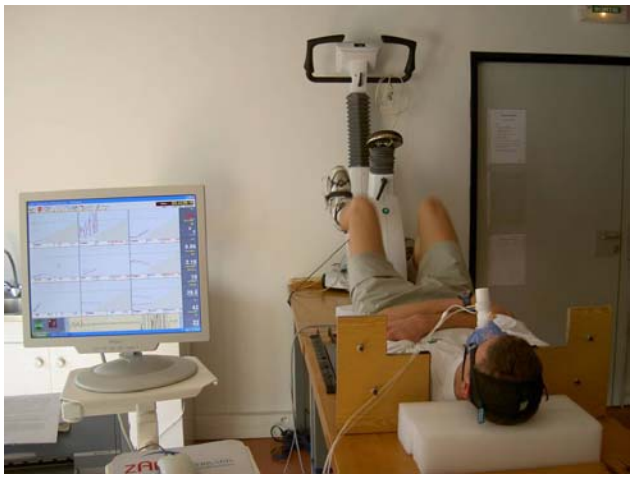

Figure 1. Schematic view of a supine exercise test, showing the experimental setting.

Subjects came to the laboratory on four occasions. Two preliminary tests were realized in order to determine the first ventilatory threshold (VT_1) specific to each cycling posture. Then, subjects were asked to perform randomly two constant workload tests, one in each of the upright and supine postures. During these tests, VO_2 was collected breath-by-breath. Electromyographic activity (EMG) of vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles was recorded at different epochs of the exercise. We also monitored VL muscle de-oxygenation at rest, during warm-up and exercising periods.

The subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, and to avoid strenuous exercise in the 48 hours preceding a test session. For each subject, tests took place at the same time of day (± 2 hours) to minimize the effects of diurnal biological variation (Carter et al., 2002).

Incremental exercise tests

As it is typically done in VO_2 kinetics studies with heavy exercise, subjects exercise at a relative percentage of workload between that at VT and maximal VO_2 for each position investigated. Because of the institution's restrictions on maximal testing of subjects without a physician present, incremental testing to voluntary exhaustion was not performed. Rather, subjects exercised at an absolute power (25 W) above their VT, which, from an extensive review of the literature on VO_2 kinetics, was a typical workload that was ~30% between that at VT and maximal VO_2 for the subjects tested in the present study (heavy domain). Some pilot studies in our laboratory allowed us to check that the same relative metabolic load was achieved between supine and upright cycling exercise by using this procedure. Further it has been recently showed that the steady-state VO_2 in supine and upright postures represented the same percentage of maximal workload (80%) achieved during the graded test in the same posture (Egana et al., 2006). On separate days (at least two days apart), a ramp test protocol ($25 W \cdot min^{-1}$) was thus conducted for each posture until at least one workload above the VT_1 intensity level was clearly discernable. VT_1 was determined by two experienced investigators from the point of increased minute ventilation (VE)-to-VO_2 ratio without a concomitant increase in VE-to-VCO_2 output ratio (Tordi et al. 2003); there was no case in which the two determinations of VT_1 differed.

Constant workload tests

Then, on two separate days, subjects were asked to perform a 12-min constant workload cycling exercise test at a constant and similar pedal frequency comprised between 60-80 rpm. The workload was set to correspond to posture specific VT_1 plus 25 W. As said before, this level of intensity is known to elicit the slow component phenomenon during heavy cycling and appears to be sufficient enough to induce muscle fatigue-related changes (Xu and Rhodes, 1999). Each constant exercise was preceded by 3-min of rest, followed by a 3-min warm-up period at 20 W.

Measurements

Before each constant workload test in supine and upright postures, mean arterial pressure (MAP) was taken with a standard sphygmomanometer and a stethoscope during quiet resting from the right brachial artery. MAP was the sum of diastolic blood pressure added to one third the pulse pressure; pulse pressure was the difference between systolic and diastolic pressures. We elected the supine and upright postures with the arm extended at heart level. Heart rate (HR) was continuously recorded telemetrically at rest and during the exercising period using a heart rate monitor (Polar Electro Oy, Kempele, Finland).

Oxygen uptake

Breath-by-breath gas exchange data were carried out throughout the tests (i.e. during the rest, warm-up and exercising periods) with the help of a metabolic cart (ZAN 680, Oberthulba, Germany) using standard algorithms, allowing for the time delay between gas concentration and volume signals.

Subjects breathed through a low dead space (40 ml), low resistance (<0.05 $kPa \cdot L^{-1} \cdot s^{-1}$ constant up to 14 $L \cdot s^{-1}$) mouthpiece and turbine assembly. Gases were continuously drawn from the mouthpiece through a 2 m capillary line of small bore, and analyzed for O_2 and CO_2 concentrations by optical spectrometer and infrared absorption analyzers, respectively. Expiratory volume was determined by a flow sensor calibrated before each test using a known volume syringe (1 l, ZAN 680, Oberthulba, Germany,). Gas analyzers were calibrated before each test from both the room air and gas of known concentration. Respiratory gas exchange variables (VO_2, production of carbon dioxide, VCO_2 and VE) were calculated and displayed for every breath.

Muscle de-oxygenation

Local muscle oxygenation profiles were assessed using the NIRS technique. The NIRS signal provides continuous, non-invasive monitoring of the relative concentration changes in oxygenation ([HbO_2]), deoxy-hemoglobin ([HHb]) and total hemoglobin ([Hbtot]) concentrations (Sako et al., 2001). Because of the overlap of the spectrum it is not possible to distinguish between changes in hemoglobin (Hb) and myoglobin (Mb). However, in human skeletal muscle, the ratio of Hb to Mb concentration is >5 (Mancini, 1997) so the signal is usually considered as deriving mainly from Hb. In the present study, only changes in VL muscle de-oxygenation were continuously monitored at 6 Hz using a near-infrared spatially resolved spectroscopy oximeter (NIRO-300, Hamamatsu Photonics, Japan). VL muscle was chosen due to its great involvement in cycling exercise (Ericson et al., 1985). NIRO-300 optodes were housed in an optically dense plastic holder, ensuring that their position relative to each other was fixed and invariant. The probe (i.e. the optodes support) was secured on the cleaned skin surface with tape and then covered with a black home-made cotton tissue, thus minimizing the intrusion of extraneous light and loss of infrared light from the field of interrogation. The probe was placed over the VL muscle belly, ~12 cm above the right external part of the patella. Skinfold thickness was measured on the area we investigated using a skinflod caliper (Holtain Ltd., Crymmych, UK), and was divided by 2 to determine the adipose tissue thickness (i.e. fat + skin layer) covering the muscle. The obtained values were 3.8 ± 1.3 mm, and were well below the minimum required, allowing the NIRS proton to penetrate through the muscle (Ferrari et al., 1997; van Beekvelt et al., 2001). The absorption of light at different wavelengths (775, 810, 850 and 910 nm) was analyzed according to the modified Beer–Lambert's law. A 3.8 differential pathlength factor (DPF) was used for the VL muscle (Delorey et al., 2005), thus changes in HHb concentrations are reported as a change from baseline in micromolar units (μM). It has been proposed that HHb was less sensitive to blood volume changes than its counterpart HbO_2 (Delpy and Cope, 1997). Thus we used HHb as an estimator of changes in intramuscular oxygenation and O_2 extraction. After each test, probe emplacement was carefully marked.

Muscle activity

Surface EMG from the muscle belly of VL, RF and BF were recorded during the 12-min constant workload exercise. We used bipolar Ag/AgCl electrodes (Contrôle Graphique Medical, Brie-Comte-Robert, France) with a diameter of 9 mm and an inter-electrode distance of 25 mm maintained constant. The ground electrode was placed on the wrist. Low impedance at the skin-electrode surface (< 5 kΩ) was each time obtained by abrading the skin with emery cloth and cleaning with alcohol. EMG signals were band-pass filtered at 30-500 Hz and sampled at 2000 Hz, with the Biopac A/D device (MP30, Biopac, Inc., Santa Barbara, USA). After each test, care was taken to mark the electrodes emplacement.

Data analysis

Occasional errant data points (e.g. consequent to a cough, sneeze or sigh) were at first visually deleted from the respiratory data set to enhance the underlying characteristics. Then, non-linear regression techniques were used to fit the VO_2 kinetics data during the warm-up (20W) to exercise transitions in both positions with a bi-exponential model commonly used in the literature for heavy cycling (Barstow et al., 1996; Cleuziou et al., 2005; Koga et al., 1999). The mathematical model consisted of two exponential terms, each representing one phase of the response (primary and slow components). Based on previous literature (Barstow et al., 1996), the model was constrained to aid in identification of the key parameters. As suggested by Whipp et al. (1982), VO_2 values recorded at the early beginning of the transition (< 20 s) were visually excluded from the analysis since they do not contribute to the muscle metabolism. The computation of best-fit parameters was chosen by the program to minimize the sum of the squared differences between the fitted function and the observed response. Data fitting were analyzed with successive iteration, using the squared differences method. The equation was:

$$y(t) = VO_2(b) + A_P \times (1 - e^{-(t - TDp)/\tau p}) + A_S \times (1 - e^{-(t - TDs/\tau s})$$

where VO_2(b) is the average value of VO_2 during the 3-min warm-up, A_P and A_S are the asymptotic amplitudes of the exponentials; TDp and TDs are the time delays, and τp and τs are the time constants of the primary and slow component phases, respectively.

The increase in VO_2 above baseline level as a function of the net increase in power output was also calculated. Then, following the procedure outlined previously (Lamarra et al., 1987) the 95% confidence intervals for estimation of the primary component (as determined by τp) were calculated. We found a single transient to be sufficient in our subjects as the large amplitude of response was associated with a good signal-to-noise ratio. Moreover, we cannot exclude the fact that the breath-to-breath variability may possess biological significance, although Lamarra et al. (1987) suggested stochastic properties of the breath-to-breath noise. In the present study, the lack of relationship between the residuals and the time ($p > 0.1$) and the sum of the residuals lower than 10^{-6} support the view of these authors. Even if a single transition may be spoiled by inherent noise, the use of a higher-order model (two exponential terms) during heavy exercise is justifiable because the parameters estimated from the data produced a statistically significant reduction in the error between the modeled and measured responses compared to a first-order model (monoexponential fit). A higher order model was not accepted as a superior representation of the response if the decrease in summed-square error was sufficient to offset the loss in degrees of freedom associated with the increased number of model parameters as determined by an F-test (F=model variance · residual variance^{-1}) (Motulsky and Ransnas, 1987). Plots of residuals were also examined to help determine the appropriate fits.

At rest, mean [HHb] values were averaged on a 30 s interval at the 2nd minute for the two postures. During the exercise, [HHb] values were averaged on a 30 s interval basis at the 3rd minute of the warm-up, and at the 2nd and 11th minutes of the exercise. These times were chosen since they allowed us to investigate a large range of the exercising period and corresponded to the different phases of the VO_2 kinetics-related adaptations. In this study, we also analyzed [HHb] kinetics with a mono-exponential model (Delorey et al., 2003). Kinetics of [HHb] during the rest-to-exercise transition were fitted from the time of initial increase in HHb to 90 s with a mono-exponential model of the form in equation for O_2 uptake kinetics without the slow component (Delorey et al., 2003).

Analysis of the EMG amplitude for the three muscles investigated have been performed with the Biopac software (Acqknoweldge, BSL Pro 3.6.7, Inc., USA). Root Mean Square (RMS in mV) EMG values were calculated for the three muscles on a burst by burst basis around the 2nd, 6th and 11th minutes of the constant workload cycling exercise for 6 consecutive contractions before being averaged together. Then for each minute investigated (i.e. 2, 6 and 11), average RMS values were divided by the corresponding values of VO_2, in order to estimate the adjustment of RMS to the muscle metabolism (RMS/VO_2 ratio, Jammes et al., 1998).

Statistical analysis

Data are presented as mean values (± SD). At rest, the results regarding the differences between upright and supine postures for HR, MAP, [HHb] and VO_2 values were analyzed using the Student's *t*-test for paired data. During the constant workload tests, similar analyses were carried out to compare the mean power output and the parameters of VO_2 and [HHb] kinetics. Two-way repeated measures ANOVA with *post hoc* Student-Newman-Keuls tests were used to test for difference in average [HHb] and EMG values among specific time of exercise and between postures. Significance was set at $p < 0.05$.

RESULTS

The nine subjects cycled at 197.2 (42.3 W) in upright position and at 149.9 (30.0 W) in supine position ($p < 0.05$). All the subjects were able to cycle for 12 minutes in both postures.

Heart rate, mean arterial pressure and VO_2 kinetics

At rest, HR and MAP values were significantly higher in upright compared to supine postures (+ 12.4 ± 7.7 beats·min^{-1}, + 9.2 ± 0.7 mmHg, $p < 0.05$, respectively). During the exercise steady-state HR values averaged between the 10th and 11th minutes were significantly higher in upright cycling (+18.0 ± 8.4 beats·min^{-1}).

Figure 2 represents the VO_2 kinetics for a representative subject for the two postures at a similar relative metabolic load. The increase in VO_2 from baseline level per unit increase of work intensity before the appearance of the slow component of the VO_2 kinetics was not significantly different between upright and supine cycling (10.8 ± 1.3 vs 11.8 ± 1.1 $mlO_2 \cdot min^{-1} \cdot W^{-1}$, respectively). The main differences among VO_2 kinetics parameters between the two postures are reported in Table 1. The VO_2 primary component amplitude (Ap) was

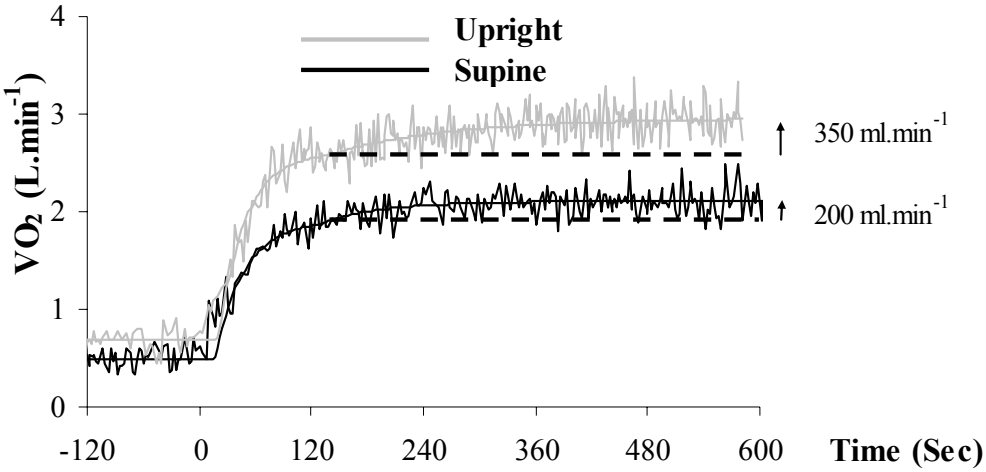

Figure 2 – Representative O_2 uptake kinetics for one subject during a 12 min constant workload cycling exercise in supine and upright postures at a constant and similar pedal frequency comprised between 60-80 rpm. The heavy workload was adjusted to correspond to a same relative metabolic load between the two postures. Horizontal dashed lines represent the primary component's asymptotic amplitudes of the VO_2 kinetics.

greater in upright cycling compared to supine cycling; similar findings were found regarding the slow component. However the relative magnitude of the slow component represented a similar percentage of the total increase in VO_2 during upright and supine cycling exercise but with large standard deviations. The time constant of the primary component was significantly higher in supine compared with upright cycling. The mean 95% confidence intervals for the estimation of τp were ± 3.9 s and ± 5.9 s during upright and supine cycling.

Muscle de-oxygenation changes

Values of [HHb] were significantly higher in supine cycling than in upright during the warm-up period and at the 2nd and 11th minutes of exercise (Figure 3). For both postures, *post-hoc* tests revealed that [HHb] was significantly higher at the 2nd and 11th minutes compared with the warm-up period. For each time analyzed, [HbO_2] and [Hbtot] values were significantly lower in supine compared with upright postures ($p < 0.05$).

Regarding [HHb] kinetics, no differences were found between the two exercising postures. However, whatever the posture [HHb] time constant and time delay of the primary component (i.e. τp [HHb] and TDp [HHb], respectively) were significantly lower than those of the corresponding values of VO_2 (Table 2).

Myoelectrical activity changes

There was a significant increase in the VL RMS amplitude during upright cycling from the 2nd minute of exercise to the 6th and 11th minutes (+20% and +25%, respectively, Figure 4). In supine cycling, no differences in RMS were found over time.

RMS/VO_2 ratio was significantly higher in supine compared to upright postures only at the 2nd minute (0.0065 *vs.* 0.0052 mV $mlO_2 \cdot kg^{-1}$, Figure 4).

Then this ratio significantly decreased over time in supine posture. No significant differences

Table 1 – VO_2 response parameters in upright and supine cycling at the same relative intensity. Mean values are (± SD) ($n = 9$).

	Upright	Supine
VO_2 (b) ($L \cdot min^{-1}$)	.80 (.13)	.68 (.08) *
Ap ($L \cdot min^{-1}$)	1.91 (.49)	1.53 (.33) *
τp (s)	23.5 (6.7)	32.7 (10.7) *
As ($L \cdot min^{-1}$)	.44 (.20)	.22 (.13) *
τs (s)	126.3 (60.7)	142.5 (101.8)
As (%)	19.1 (7.4)	13.2 (8.1)

VO_2 (b) is the average value of VO_2 during the 3-min warm-up, A_P and A_S are the asymptotic amplitudes and τp and τs are the time constants of the primary and slow component phases, respectively. * Significantly different from upright cycling ($p < 0.05$).

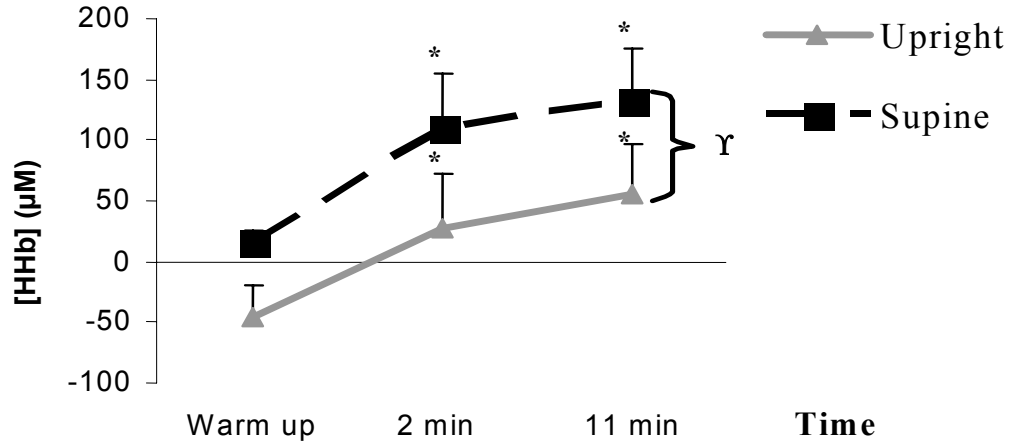

Figure 3. Muscle de-oxygenation ([HHb]) changes (mean values ± SD, n = 9) from the warm-up to the end of the exercise. Muscle de-oxygenation values are expressed as changes from baseline values (i.e. established in a steady state, before the 20 W warm-up). $p < 0.05$, * Significantly different from warm-up, ϒ Significantly different from supine cycling.

were found for upright exercise over time. Whatever the cycling position, RMS and RMS/VO_2 values for the RF and BF muscles did not show any significant changes throughout the exercise period.

DISCUSSION

In this work, we aimed to study the effects of circulatory difference induced by body posture on the oxidative metabolism dynamic adaptations related to muscle de-oxygenation and myoelectrical activity during cycling exercises for a same relative metabolic load. The main findings of this study are i) an increase of the primary component time constant during supine cycling; ii) a higher muscle de-oxygenation in supine cycling whereas no differences were found among [HHb] kinetics between the two postures; iii) and an increase in the VL RMS amplitude over time during upright cycling.

Heart rate, mean arterial pressure

In the present study, we did not measure muscle blood flow. However, it has been showed that despite a greater cardiac output (Hughson et al., 1991; 1993; Leyk et al., 1994) the effective blood flow to the working muscles decreases in supine posture (Eiken, 1988; Egana and Green, 2005; Folkow et al., 1971; MacDonald et al., 1998) as a consequence of a lower arterial pressure in the legs when the hydrostatic gradient effect is removed. Furthermore, body posture changes are known to determine a gravitational gradient that acts on both the cardiovascular and cardiopulmonary systems and consequently affect optimal blood flow and oxygen delivery (Jones and Dean, 2004).

VO_2 kinetics

With regards to the amplitude of the primary component (i.e. Ap in Table 1), differences between supine and upright heavy exercises can be logically attributed to differences in absolute power output (mean difference of 47.3 W). However as expected with the experimental approach used, our data indicate that the subjects performed similar relative work intensities in both upright and supine postures. The equal gain in metabolism per unit increase in work intensity in upright and supine postures does support the premise that subjects were performing equal relative work rates (10.8 ±1.3 *vs.* 11.8 ± 1.1 $mlO_2 \cdot min^{-1} \cdot W^{-1}$, respectively) before the appearance of the slow component in the "heavy" domain. There

Table 2. Comparison of VO_2 and [HHb] kinetics at the beginning of the exercise. Values are expressed as mean (± SD) (n = 9).

	Upright		Supine	
	VO_2	**[HHb]**	**VO_2**	**[HHb]**
TDp (s)	20.5 ± 2.4	14.7 ± 2.9 *	19.1 ± 3.8	14.1 ± 2.9 *
τp (s)	23.5 ± 6.7	10.5 ± 5.8 *	32.7 ± 10.7	10.0 ± 2.7 *

TDp, primary component time delays of [HHb] and VO_2 kinetics; τp, primary component time constants of [HHb] and VO_2 kinetics. * Significantly different to VO_2 values ($p < 0.05$).

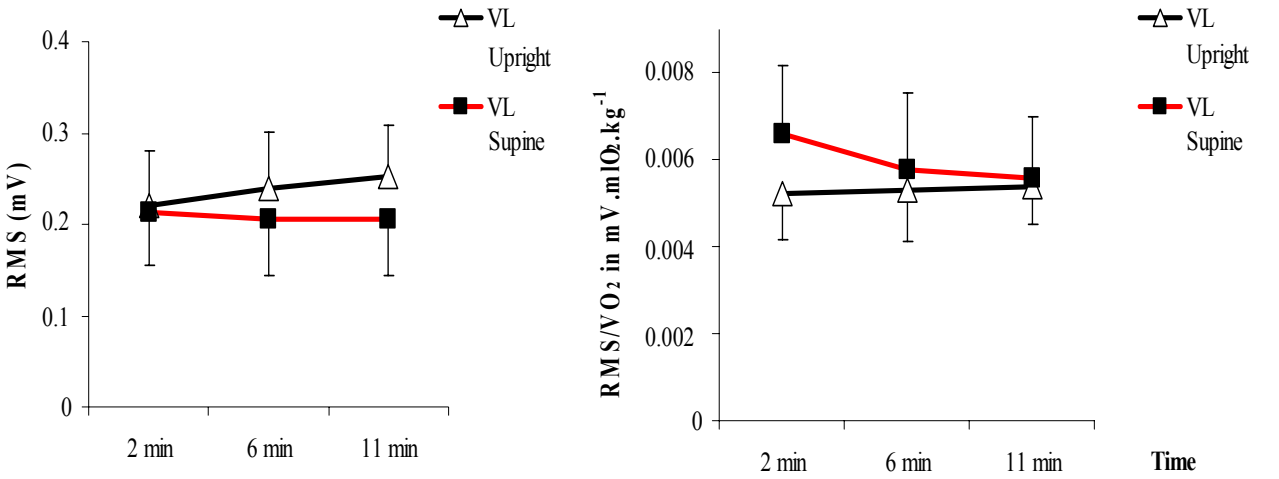

Figure 4 – Mean (± SD) RMS (on the left) and RMS/VO_2 ratio (on the right) values (n = 9) at the 2nd, 6th and 11th minutes of the exercise. $p < 0.05$, * Significantly different from min 2, ϒ significantly different from upright cycling.

appears to be a reasonable assumption that the VT_1 determined for each subject in each exercise posture represents the same %VO_2max. A recent study showed that VO_2 end-exercise values for constant-load cycling tests (upright and supine) expressed as a percentage of those measured during the graded test (~80% of maximal power output) in the same posture were not different between both postures (Egana et al., 2006). Consequently, our results suggest that the fiber pool recruitment was likely similar during the first 2-3 min of exercise (confirmed in part by identical VL RMS values at min 2 of the constant-load exercise in both postures, Figure 4).

Our results show that VO_2 kinetics primary component was significantly slower in supine cycling compared to upright cycling (τp decreased by ~39%). Even if a single transition was performed (first limitation), 95% confidence intervals for τp as 4 s and 6 s for upright and supine exercises were judged acceptable when comparing τp values that are substantially different between supine and upright exercise transients (Table 1). Our findings confirm past studies when moderate-intensity exercises were used but at the same absolute workload (Convertino et al., 1984; Hughson et al., 1991). Interestingly, it appears that the increase in power output between supine and upright postures in Koga et al. (1999) (almost a difference of 100 W) did not influence τp but lowered significantly the relative metabolic demand (in $mlO_2 \cdot min^{-1} \cdot W^{-1}$) in supine compared to upright heavy cycling exercises. Altogether even if in our study design only relative and not absolute workloads were compared (second limitation), we think that all observed differences among VO_2 kinetics (between the two experimental conditions) were likely due to posture-related changes in local blood flow and not to concomitant changes in power output. Speeding of VO_2 kinetics has already been reported in various experimental contexts: with endurance training (Philips et al., 1995), by inducing an important metabolic acidosis (Tordi et al., 2003) or by eliciting a muscle chemoreflex to increase MAP and blood flow to an exercising muscle mass (Perrey et al., 2001a). Based on the literature, we may suggest that O_2 transport appeared to slow the VO_2 kinetics in supine compared to upright cycling at the same relative metabolic demand, and was likely the limiting factor under the actual experimental conditions (Convertino et al., 1984; Hughson et al., 1991; 1993; MacDonald et al., 1998).

Moreover we showed for the first time that the VO_2 slow component amplitude (absolute terms) was significantly lower in supine than in upright cycling exercise performed at the same relative metabolic demand; a seemingly large physiological difference was observed in relative terms for slow component but was not significant due to large standard deviations. To the best of our knowledge, the only study investigating the effects of upright and supine cycling exercises on VO_2 slow component was the study of Koga et al. (1999) who found a higher VO_2 slow component (in both absolute and relative terms) in supine cycling but at the same absolute power output. It is well known that the magnitude of the slow component depends on the relative exercise intensity (Gaesser and Poole, 1996) and may in part explain the differences between the two studies. By analogy, our result is in full agreement with that of Cleuziou et al. (2005) who showed that under hypoxia, subjects exhibited a lower slow component compared to normoxic condition at identical exercise intensities (equivalent

relative metabolic demand) but an equivalent slow component amplitude in relative terms. They suggested that the decrease in inspired gas concentration was the main possible explanation for the lower slow component amplitude observed in their experimental design. Decrease in slow component amplitude could arise from differences in O_2 availability *per se* (probably not in the current study due to the same relative exercise intensity), or from possible change in muscle fiber recruitment as a consequence of the differences in O_2 availability (Pringle et al., 2003). In several experiments (Barstow et al., 1996; Borrani et al., 2001; Shinohara and Moritani, 1992), the slow component phenomenon has been linked to a progressive recruitment of fast twitch fibers. The RMS increase for VL observed in upright cycling confirms partly the above studies. However, all remaining EMG analyses for the two remaining muscles and mainly during supine condition suggest that it is still difficult to link EMG data to the slow component phenomenon (Scheuermann et al., 2001). We proposed that the reduction in the absolute amplitude of slow component in supine position might be explained by a greater contribution of the type I fibers to the exercise after 2-3 min. At an intensity above VT, a large number of type II fibers are recruited during the first 2-3 min of the heavy exercise before the entire type I fiber pool is active (Pringle et al., 2003).

Muscle de-oxygenation kinetics

The increased local O_2 extraction ([HHb]) observed in the present study in both exercising postures is consistent with the literature. Chance et al. (1992) and Neary et al. (2001) showed that the de-oxygenation magnitude was proportional to the exercise intensity level. Our study was the first to investigate muscle oxygenation changes induced by posture, among large muscle masses. Tachi et al. (2004) demonstrated that isometric exercise performed in a leg up condition induced a decrease in muscle oxygenation. Regarding our results, we observed the same tendency. In supine cycling [HHb] was significantly higher at all the time periods investigated (i.e. warm-up, 2^{nd} and 11^{th} minutes) compared to upright cycling. This confirms our hypothesis concerning an increase in muscle de-oxygenation related to a decrease in O_2 delivery (evidenced by a diminution of baseline HR and of local oxygenation), due to the modifications induced by the supine posture (i.e. hydrostatic pressure gradients in blood vessels oriented longitudinally in the body are larger in upright than in supine). Overall, significant increase in [HHb] appeared to reflect a mismatch between the oxygen supply and the increasing metabolic demand of the active muscle due to both the exercise intensity and the decrease in O_2 delivery. Regarding [HHb] kinetics, they were significantly faster than those of VO_2 in both supine and upright postures. These results are in agreement with those of DeLorey et al. (2003) and Bringard and Perrey (2004). For both postures, after a time delay of ~14 s, HHb increased rapidly toward a "steady-state" level (τp mean value of 10 s), suggesting that muscle perfusion or the local distribution of blood flow and O_2 delivery in the on-transient of heavy-intensity cycling exercise was not adequate to meet the metabolic demand of the muscle, thus requiring a rapid increase in O_2 extraction. However, by using the mean response time for the on-kinetics of HHb (TD plus τ) and the τp (i.e., the time constant of the metabolically relevant "primary phase") of the pulmonary VO_2 on-kinetics, the upright comparison is 24 and 26 s for VO_2 and HHb, and 33 s versus 24 s for the supine comparison. The difference in supine ($p < 0.05$), but not in upright may suggest that the kinetics of O_2 delivery was indeed limiting the VO_2 kinetics in supine. According to the Fick principle the VO_2 / [HHb] ratio can be used to estimate the muscle capillary blood flow (Ferreira et al. 2005). In the present study the muscle capillary blood flow kinetics was thus likely slower in supine position (Eiken, 1988; Folkow et al., 1971), suggesting that a delay occurred probably between O_2 supply-diffusion mechanisms and muscle demand.

RMS and RMS/VO_2

In the present study, our unexpected results about EMG were different of Tachi et al (2004). These last authors found an increase in the integrated EMG when subjects performed static dorsiflexion with their legs up compared to the same exercise realized with their legs down. Conversely, we observed an increase in the VL RMS amplitude only when subjects were cycling upright in which the slow component was found to be the highest. In supine cycling, RMS signal was constant throughout the exercise for all the muscles investigated. Steady RMS signal found in supine cycling can be explained by the position-related O_2 supply changes leading to a change in neuromuscular activity. Motor unit recruitment has been shown to be not only dependent on the level of force developed but also on the oxygen availability (Moritani et al., 1992). Regarding the EMG myoelectrical activity of RF and BF muscles, no differences were found between the two postures, and their respective RMS values remained steady throughout the exercise. This result shows that for the same relative exercise metabolic demand, VL muscle was likely the main muscle to

participate to the cycling task in upright posture (Van Ingen Schenau et al., 1995). The ratio between total EMG energy (RMS) to the corresponding VO_2 was higher in supine posture only for the VL muscle at the second minute of the exercise. Thereafter, this ratio decreased to the 6th minute of the exercise and remained stable as it did during upright cycling. A possible explanation of a higher RMS/VO_2 ratio at the minute 2 of supine exercise could be the existence of a longer time delay between the power production and the energy supply adjustment during the first minutes of exercise (Jammes et al., 1998).

CONCLUSION

This study aimed at evaluating the effects of heavy cycling exercise in supine and upright postures on the oxidative metabolism and muscle oxygenation kinetics in relation to muscle activity at the same relative metabolic load. In supine cycling, muscle de-oxygenation was greater, oxidative metabolism adaptation was slowed significantly, but RMS did not change for any of the investigated muscles. These results indicate that the decrease in O_2 supply induced by a decrease in hydrostatic pressure gradient in supine posture impaired oxidative metabolism adjustment without inducing premature muscle fatigue at the same relative metabolic demand.

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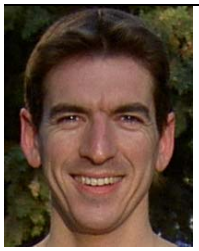

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KEY POINTS

- Hydrostatic pressure gradients in blood vessels oriented longitudinally in the body are lesser in supine than in upright posture.
- Lowered oxygen supply induced with supine exercise slows oxidative metabolism dynamics and increases muscle de-oxygenation during heavy exercise.
- Compared to upright, supine exercise did not increase markedly the rate of muscle fatigue at a same relative metabolic load.

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