

The slow component of $\dot{V}O_2$ in professional cyclists

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Abstract

Objectives—To analyse the slow component of oxygen uptake ($\dot{V}O_2$) in professional cyclists and to determine whether this phenomenon is due to altered neuromuscular activity, as assessed by surface electromyography (EMG).

Methods—The following variables were measured during 20 minute cycle ergometer tests performed at about 80% of $\dot{V}O_{2MAX}$ in nine professional road cyclists (mean (SD) age 26 (2) years; $\dot{V}O_{2max}$ 72.6 (2.2) ml/kg/min): heart rate (HR), gas exchange variables ($\dot{V}O_2$, ventilation ($\dot{V}E$), tidal volume (V_T), breathing frequency (f_b), ventilatory equivalents for oxygen and carbon dioxide ($\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ respectively), respiratory exchange ratio (RER), and end tidal PO_2 and PCO_2 (P_{ETO_2} and P_{ETCO_2} respectively)), blood variables (lactate, pH, and $[HCO_3^-]$) and EMG data (root mean from square voltage (rms-EMG) and mean power frequency (MPF)) from the vastus lateralis muscle.

Results—The mean magnitude of the slow component (from the end of the third minute to the end of exercise) was 130 (0.04) ml in 17 minutes or 7.6 ml/min. Significant increases from three minute to end of exercise values were found for the following variables: $\dot{V}O_2$ ($p < 0.01$), HR ($p < 0.01$), $\dot{V}E$ ($p < 0.05$), f_b ($p < 0.01$), $\dot{V}E/\dot{V}O_2$ ($p < 0.05$), $\dot{V}E/\dot{V}CO_2$ ($p < 0.01$), P_{ETO_2} ($p < 0.05$), and blood lactate ($p < 0.05$). In contrast, rms-EMG and MPF showed no change ($p > 0.05$) throughout the exercise tests.

Conclusions—A significant but small $\dot{V}O_2$ slow component was shown in professional cyclists during constant load heavy exercise. The results suggest that the primary origin of the slow component is not neuromuscular factors in these subjects, at least for exercise intensities up to 80% of $\dot{V}O_{2MAX}$.

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It is well documented that oxygen uptake ($\dot{V}O_2$) tends to slowly rise during any constant load exercise test involving sustained lactic acidosis—that is, above the lactate or ventilatory thresholds. This phenomenon is called “the slow component of $\dot{V}O_2$ ” and is typically defined as the continued rise in $\dot{V}O_2$ beyond the third minute of exercise.^{1–6} The $\dot{V}O_2$ slow component has been mostly identified in cycle ergometry tests.⁷

Several factors have been proposed as determinants of the $\dot{V}O_2$ slow component during heavy exercise and include lactate,^{2 4 8–10} adrenaline (epinephrine),^{5 12} cardiorespiratory work,^{11–13} temperature,^{3 13} potassium,¹⁴ lower chemical-mechanical coupling efficiency,^{3 6} and what appears to be the most important factor, a progressive recruitment of less efficient motor units with exercise duration.^{15–17} As most of the slow component has been shown to arise from the exercising limbs,^{3 18} the mechanisms responsible may indeed have their main effect on the working skeletal muscles. One previous study by Shinohara and Moritani¹⁶ found a significant increase in the integrated electromyographic (iEMG) activity of the quadriceps muscle in untrained subjects during constant load exercise, suggesting, at least in part, a certain involvement of neuromuscular factors—for example, recruitment of additional motor units—in the aetiology of the $\dot{V}O_2$ slow component. On the other hand, several authors^{1 8 12} have shown that endurance training significantly attenuates the magnitude of the slow component, and that such a training effect may be largely attributed to neuromuscular adaptations—for example, recruitment pattern of motor units.⁷ To date, however, most research in the field has been conducted on healthy subjects who are not well trained. There have been surprisingly few attempts¹⁹ to analyse the kinetics of $\dot{V}O_2$ during heavy constant load cycle ergometry tests—that is, at 80–90% of $\dot{V}O_{2MAX}$ —in elite endurance athletes. For example, it would be of great interest to determine the role neuromuscular factors play in the slow component phenomenon in top level endurance athletes—for example, those exposed to the demands of elite competition, such as Olympic class marathon runners and professional cyclists. It was recently shown in our laboratory that professional road cyclists show remarkable physiological characteristics^{20–22} compared with their elite amateur counterparts. These include (a) a characteristic efficient breathing pattern²¹, (b) the capacity to perform at high workloads (about 90% of $\dot{V}O_{2MAX}$) over long periods of time (60 minutes or more)²², (c) a considerable reliance on fat metabolism even at high power outputs (determined by the blood lactate response and respiratory exchange ratio)²⁰, and, most importantly, (d) several neuromuscular adaptations shown by EMG—for example, a greater resistance to fatigue of slow motor units.²⁰

The purpose of this investigation was to analyse the kinetics of the $\dot{V}O_2$ response to heavy (about 80% of $\dot{V}O_{2MAX}$) constant load exercise in a group of professional road cyclists. Gas exchange/blood variables and EMG data corresponding to exercising muscles were

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measured to determine which factors—for example, recruitment pattern of motor units—may be responsible for the slow component of $\dot{V}O_2$ in these athletes.

Methods

SUBJECTS

Nine professional road cyclists were selected for the investigation. Their mean (SD) age, height, and weight were: 26 (2) years, 180.7 (4.4) cm, and 70.2 (6.1) kg respectively. A previous physical examination (including electrocardiographic (ECG) and echocardiographic evaluation within the previous month) ensured that each participant was in good health. Several of the subjects are among the best cyclists in the world—that is top 20 in 1998 ranking of the Union Cycliste Internationale (UCI). The racing awards obtained by the subjects include: gold medal in the UCI World Championships for professional cyclists (both road race and time trial), silver medal in the Olympic Games (time trial), 1st position in the Vuelta a España, 4th and 8th positions in the Tour de France, 4th position in the Giro de Italia.

Since the 1998 Tour de France there has been increasing evidence of recombinant human erythropoietin (rHuEPO) administration to some professional cyclists. In the hypothetical situation of the use of this drug in our subjects, this could artificially increase oxygen transport capacity and thereby alter several of the physiological variables estimated. Although it is currently impossible to detect rHuEPO, the UCI requires that professional cyclists undergo frequent blood tests during the season to confirm that their packed cell volume is consistently below 0.50. In this manner, blood doping with rHuEPO can be partly prevented. Before participation in the study, we obtained venous blood samples from each subject to check that their packed cell volume was below this limit.

STUDY PROTOCOL

Informed consent was obtained from each participant in accordance with the guidelines of the Complutense University. Each subject reported to our laboratory on two consecutive days during the competition period of the season (in May). On the first day, the subjects performed a maximal exercise test (ramp protocol) and on the second they performed a submaximal constant load test to determine the $\dot{V}O_2$ slow component. Both tests were performed on the same bicycle ergometer (Ergometrics 900; Ergo-line, Barcelona, Spain) with the subject adopting the conventional (upright sitting) cycling posture. This posture was characterised by a trunk inclination of about 75° and by the subject placing his hands on the handlebars with elbows slightly bent (about 10° of flexion). All the tests were performed under similar environmental conditions (21–24°C, 45 to 55% relative humidity). The subjects were cooled with a fan throughout the bouts of exercise.

MAXIMAL EXERCISE TEST

For the maximal test, a ramp protocol was followed until exhaustion. This type of protocol has been used for the physiological evaluation of professional cyclists in several previous studies conducted in our laboratory.^{20–23} Starting at 0 W, the workload was increased by 25 W/min, and pedalling cadence was kept constant at 70 to 90 rev/min. A pedal frequency meter was used by the subject to maintain this cadence. Each exercise test was terminated (a) voluntarily by the subject, (b) when pedalling cadence could not be maintained at 70 rev/min (at least), or (c) when established criteria of test termination were met.²⁴

During the tests, gas exchange data were collected continuously using an automated breath by breath system (CPX; Medical Graphics, St Paul, Minnesota, USA) to estimate the following variables: $\dot{V}O_2$, pulmonary ventilation (\dot{V}_E), tidal volume (V_T), breathing frequency (f_b), ventilatory equivalents for oxygen ($\dot{V}_E/\dot{V}O_2$) and carbon dioxide ($\dot{V}_E/\dot{V}CO_2$), respiratory exchange ratio (RER), and end tidal PO_2 (P_{ETO_2}) and PCO_2 (P_{ETCO_2}). The ventilatory thresholds 1 and 2 (VT_1 and VT_2 respectively) were also identified. VT_1 was determined using the criteria of an increase in $\dot{V}_E/\dot{V}O_2$ with no increase in $\dot{V}_E/\dot{V}CO_2$ and the departure from linearity of \dot{V}_E ,²⁵ whereas VT_2 was taken as that corresponding to an increase in both the $\dot{V}_E/\dot{V}O_2$ and $\dot{V}_E/\dot{V}CO_2$.²⁵ VT_1 and VT_2 were detected by two independent observers. If there was disagreement, the opinion of a third investigator was sought. The power output (W) eliciting both VT_1 and VT_2 (WVT_1 and WVT_2 respectively) was recorded to establish the workload for the submaximal constant load test. Heart rate (HR, in beats/min) was continuously monitored during the tests from modified 12 lead ECG tracings (EK56; Hellige, Freiburg, Germany).

Capillary blood samples were taken from fingertips (25 μ l) every two minutes during the tests and immediately after exercise for the determination of blood lactate concentration using an electroenzymatic analyser (YSI 1500; Yellow Springs Instruments, Yellow Springs, Ohio, USA).

Based on methodology described elsewhere,²⁶ the exercise intensity corresponding to the blood lactate threshold was determined by two independent observers by examining the blood lactate-workload relation during each test.

CONSTANT LOAD TEST

The submaximal constant load tests were performed over a 20 minute period at a fixed power output equidistant to those eliciting VT_1 and VT_2 respectively (power output = $(WVT_1 + WVT_2)/2$). This power output corresponded to about 80% of the subjects' $\dot{V}O_{2MAX}$. Each 20 minute test was preceded by a 10 minute warm up period performed at 70 W involving three minutes at WVT_1 and two minutes of gradual workload increases until the target power output was attained. During both the warm up and exercise periods, pedalling cadence was kept constant at 70–90 rev/min. Gas exchange

Table 1 Physiological variables recorded during the maximal tests

	Mean (SEM)
$\dot{V}O_{2MAX}$ (litres/min)	5.1 (0.1)
$\dot{V}O_{2MAX}$ (ml/kg/min)	72.6 (2.2)
W _{MAX}	511.7 (13.4)
LT (W)	365.0 (11.9)
LT (% $\dot{V}O_{2MAX}$)	73.9 (2.3)
VT ₁ (W)	356.0 (9.3)
VT ₁ (% $\dot{V}O_{2MAX}$)	73.8 (2.7)
VT ₂ (W)	439.3 (12.3)
VT ₂ (% $\dot{V}O_{2MAX}$)	88.2 (1.3)

$\dot{V}O_{2MAX}$, maximum O₂ consumption; W, power output; LT, lactate threshold; VT, ventilatory threshold.

data and HR were monitored as in the maximal tests. In addition, blood and surface EMG variables were determined as detailed below.

Before the start of the experimental protocol, a 21 gauge butterfly needle was inserted into the antecubital vein of each subject. The catheter was kept patent by periodic flushing with a heparinised saline solution. Blood samples were collected every five minutes during the tests. During each sampling period (about 15 seconds), a 1 ml aliquot was initially withdrawn to clear the catheter, and a 1.5 ml blood sample was subsequently collected using a heparinised syringe. A portion of each sample was taken for: (a) the immediate estimation of PCO₂ and pH using an automated blood gas analyser (ABL5; Radiometer, Copenhagen, Denmark); (b) the immediate determination of lactate concentration using an automated lactate analyser (YSI 1500); (c) determination of haemoglobin and packed cell volume. Blood bicarbonate concentration [HCO₃⁻] was calculated using the pH and PCO₂ values. Venous

blood levels of lactate and HCO₃⁻ were corrected for relative changes in plasma volume using haemoglobin and packed cell volume values, by a previously described method.²⁷

EMG recordings were taken from the vastus lateralis (at about one third of the perpendicular distance from the superior border of the patella to the greater trochanter). Pairs of surface electrodes (Bluesensor Medicotest Ag/AgCl electrodes; Rugmarken, Denmark) were attached to the skin 4 cm apart. The electrodes were placed longitudinally with respect to the underlying muscle fibre arrangement. A reference electrode was placed over the anterior superior spine of the iliac crest. Before electrode placement, the skin was shaved, abraded using sandpaper, and cleaned with alcohol to minimise source impedance. A saline EMG electrode gel was placed between the electrode and the underlying skin to enhance conductivity. The wires used to measure myoelectrical activity (connected to the electrodes) were well attached with tape to

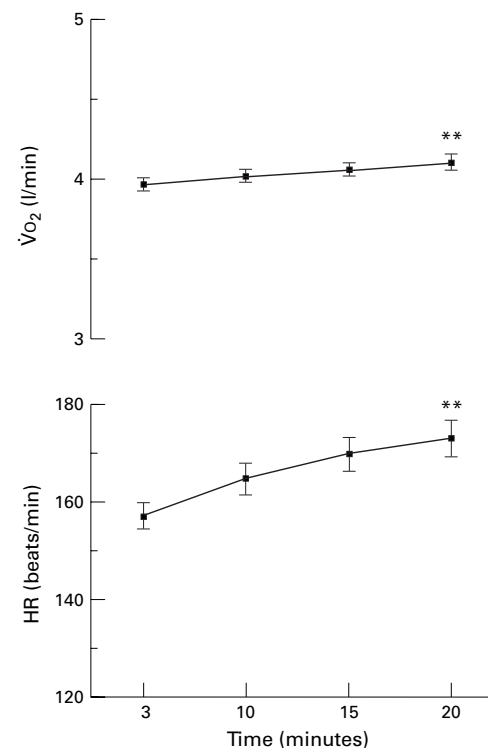


Figure 1 Oxygen uptake ($\dot{V}O_2$) and heart rate (HR) responses during the constant load tests. All values are expressed as mean (SEM). ** $p < 0.01$ for third minute v 20th minute.

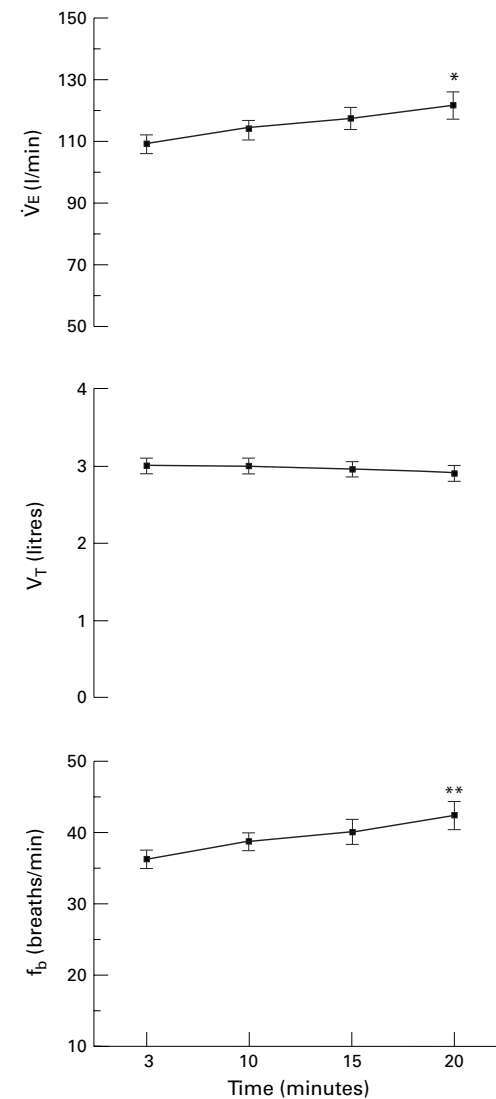


Figure 2 Ventilatory response during the constant load tests. All values are expressed as mean (SEM). Abbreviations: \dot{V}_E , pulmonary ventilation; V_T , tidal volume; f_b , breathing frequency. * $p < 0.05$ for third minute v 20th minute; ** $p < 0.01$ for third minute v 20th minute.

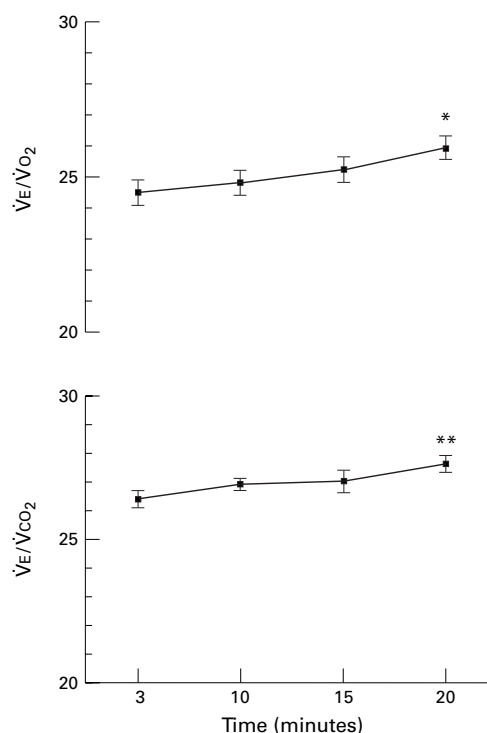


Figure 3 \dot{V}_E/\dot{V}_{O_2} and \dot{V}_E/\dot{V}_{CO_2} responses during the constant load tests. All values are expressed as mean (SEM). Abbreviations: \dot{V}_E , pulmonary ventilation; \dot{V}_{O_2} , oxygen uptake; \dot{V}_{CO_2} , carbon dioxide production. * $p < 0.05$ for third minute v 20th minute; ** $p < 0.01$ for third minute v 20th minute.

minimise artefacts from leg movements. Myoelectrical activity was recorded with a ME3000P analyser (ME3000P; Mega Electronics Ltd, Kuopio, Finland). The measure-

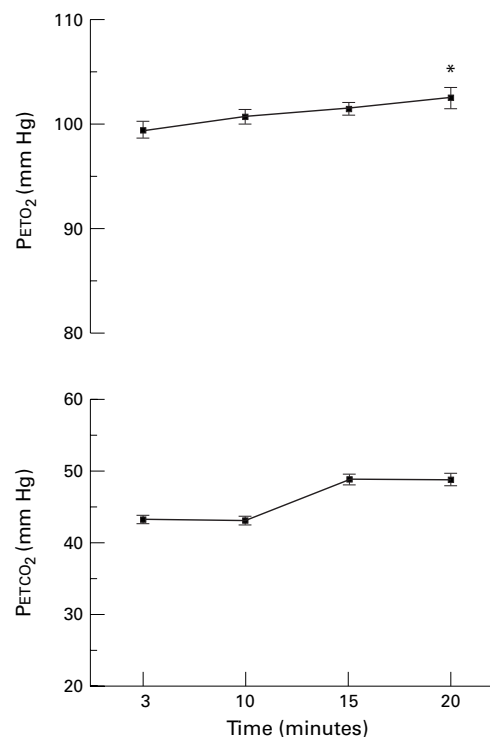


Figure 4 End tidal P_{O_2} and P_{CO_2} (PET_{O₂} and PET_{CO₂}) responses during the constant load tests. All values are expressed as mean (SEM). * $p < 0.05$ for third minute v 20th minute.

ment sensitivity of this instrument is $\pm 1 \mu V$, and its range for bipolar EMG signals is $\pm 5000 \mu V$. The raw EMG signals were band pass filtered from 20 to 480 Hz, amplified, and converted from analogue to digital at a sampling rate of 1 kHz. EMG power spectral density was then computed for two second sampling periods at fixed intervals throughout the tests. The root mean square voltage (rms-EMG, in μV)²⁸ and the mean power frequency (MPF, in Hz)²⁹ were calculated for each two second spectrum. The rms-EMG was used as an indicator of the "total myoelectric activity" of the exercising muscle because it has been previously shown that this computation is (a) an accurate measure of EMG amplitude and (b) highly correlated with the number of active motor units (fibre recruitment).²⁸ The MPF served to indicate the firing rate of motor units as it is linearly related to the action potential conduction velocity of the muscle fibre.³⁰

STATISTICAL ANALYSIS

The slow component of \dot{V}_{O_2} was defined as the difference ($\Delta\dot{V}_{O_2}$, in ml/min) between end exercise \dot{V}_{O_2} and the \dot{V}_{O_2} at the end of the third minute of constant load exercise. The latter was taken as the average \dot{V}_{O_2} from 2 min 40 s to 3 min 20 s, whereas end exercise values were taken as the average of the last two minutes of the tests (18 min 0 s to 20 min 0 s). Once the Kolmogorov-Smirnov test was applied to ensure a Gaussian distribution of the results, Student's *t* test for paired data was used to compare three minute and end exercise values of each physiological variable—that is, \dot{V}_{O_2} , \dot{V}_E , RER, rms-EMG etc. In addition, Pearson product-moment correlation coefficients were

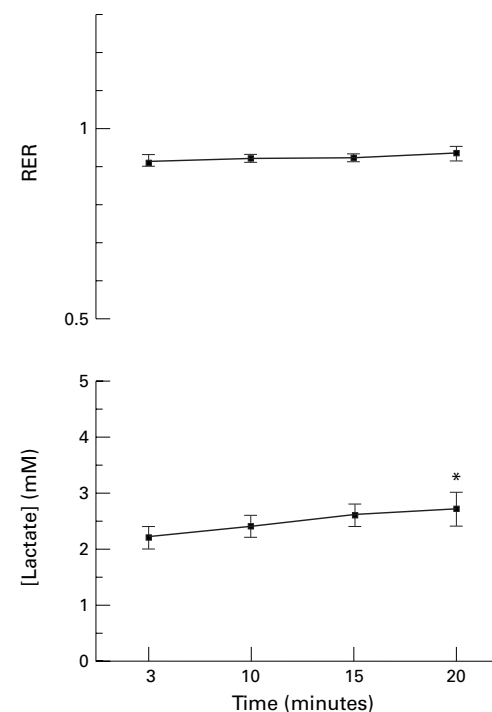


Figure 5 Respiratory exchange ratio (RER) and blood lactate during the constant load tests. All values are expressed as mean (SEM). * $p < 0.05$ for third minute v 20th minute.

calculated to determine whether there was a significant relation between the magnitude of the slow component ($\Delta\dot{V}O_2$) and that of the change in $\dot{V}E$ ($\Delta\dot{V}E$), HR (ΔHR), etc values recorded from the third minute to the end of exercise. The level of significance was set at

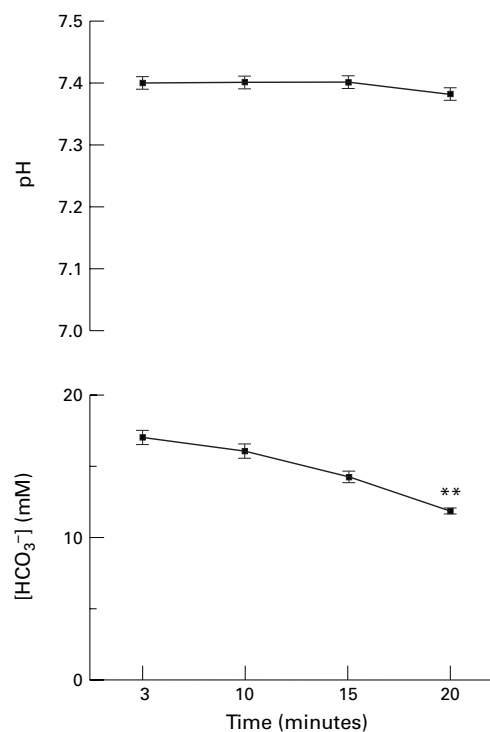


Figure 6 Blood pH and bicarbonate concentration ($[HCO_3^-]$) responses during the constant load tests. All values are expressed as mean (SEM). ** $p < 0.01$ for third minute v 20th minute.

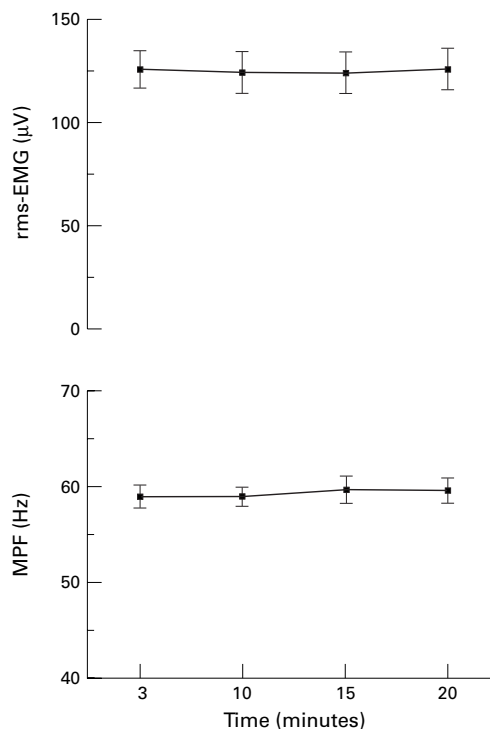


Figure 7 Electromyographic (EMG) (rms-EMG and mean power frequency (MPPF)) responses during the constant load tests. All values are expressed as mean (SEM). No significant differences between means.

0.05. Results are expressed as mean (SEM). Average values at 10 and 15 minutes of constant load exercise are also provided for descriptive purposes.

Results

MAXIMAL TESTS

Table 1 shows the physiological variables recorded during the first testing session.

CONSTANT LOAD TEST: $\dot{V}O_2$ SLOW COMPONENT

The power output at which the submaximal tests were performed averaged 400.4 (11.8) W (about 80% of $\dot{V}O_{2MAX}$) and was higher than that eliciting the lactate threshold in each of the subjects. The mean magnitude of the slow component was 130 (0.04) ml in 17 minutes (from the third minute to the end of exercise) or 7.6 ml/min.

Figures 1–7 show the response of the different physiological variables to the constant load exercise test. Significant increases were recorded in the following variables from the third minute to the end of exercise: $\dot{V}O_2$ ($p < 0.01$), HR ($p < 0.01$), $\dot{V}E$ ($p < 0.05$), f_b ($p < 0.01$), $\dot{V}E/\dot{V}O_2$ ($p < 0.05$), $\dot{V}E/\dot{V}CO_2$ ($p < 0.01$), $PETCO_2$ ($p < 0.05$), and blood lactate ($p < 0.05$). In contrast, end exercise values for blood $[HCO_3^-]$ were lower ($p < 0.01$) than those obtained after three minutes of exercise. No significant changes were found in V_{T1} , $PETCO_2$, RER, pH, rms-EMG, and MPF values throughout the tests.

Finally, no significant correlations existed between $\Delta\dot{V}O_2$ and changes in the remaining variables ($\Delta\dot{V}E$, ΔHR , etc).

Discussion

The purpose of the present study was to analyse the $\dot{V}O_2$ slow component in professional road cyclists during intense constant load cycle ergometer exercise (above both VT_1 and lactate threshold or at about 80% $\dot{V}O_{2MAX}$). To date, few research efforts have centred on the kinetics of $\dot{V}O_2$ in such well trained subjects ($\dot{V}O_{2MAX} > 70$ ml/kg/min and outstanding racing performances) able to sustain extremely high workloads (about 400 W) for at least 20 minutes with relatively low circulating lactate levels (2–3 mM). The main novel findings of the present study were that: (a) although of small magnitude, a significant $\dot{V}O_2$ slow component was shown by the professional riders; (b) the phenomenon does not appear to be directly related to peripheral factors acting on working muscles—for example, recruitment of less efficient motor units because of neuromuscular fatigue—at least at exercise intensities up to 80% of $\dot{V}O_{2MAX}$. Additional findings were that the aetiology of the slow component seems to be multifactorial in these highly trained subjects, with no specific relation between $\Delta\dot{V}O_2$ and ΔHR , $\Delta\dot{V}E$, Δ [lactate], etc.

The value of the slow component (130 ml in 17 minutes or about 8 ml/min) was considerably lower than that reported in previous research (mean values of 330 ml in 15 minutes or 22 ml/min) using the same cycle ergometer protocol (20 minutes at 80% of $\dot{V}O_2 MAX$).¹³ Further, $\dot{V}O_2$ slow components in the range of

about 380–540 ml in nine minutes or about 40–60 ml/min have been reported in people of varying fitness levels pedalling at 80% of $\dot{V}_{O_2\text{MAX}}$.^{31,32} The low slow component observed in this investigation with respect to previous findings suggests great cycling efficiency of professional riders, which is thought to contribute to their well known ability to sustain high workloads over long periods (more than 60 minutes).²² This hypothesis seems to be confirmed if we consider our data together with the findings of a recent study by Billat *et al.*,¹⁹ who detected a slow component of 268 ml in about eight minutes or about 33 ml/min in well trained triathletes cycling at about 90% $\dot{V}_{O_2\text{MAX}}$, which was significantly higher than the value (about 3 ml/min) recorded in a treadmill test at a similar relative intensity. This difference may be partly explained by the higher mechanical efficiency of running, which relies on efficient stretch shortening movements compared with the concentric work (performed by the vastus lateralis muscle) demanded in cycling.¹⁹

METHODOLOGICAL LIMITATIONS

As racing performance in professional cycling is partly determined by the cyclist's ability to tolerate almost maximal workloads (at VT_2 or about 90% of $\dot{V}_{O_2\text{MAX}}$) during long periods of time, it would also have been interesting to analyse the \dot{V}_{O_2} kinetics of these subjects during more intense exercise—for example, tests to exhaustion at 90% of $\dot{V}_{O_2\text{MAX}}$. Indeed, some factors—for example, lactic acidosis or patterns of motor unit recruitment—which did not seem to play a major role at 80% of $\dot{V}_{O_2\text{MAX}}$, could in fact account for a higher percentage of the slow component in the same subjects exercising at more severe intensities. Further, considering both the variability of EMG recordings obtained from electrodes placed on the skin and the low value of $\Delta\dot{V}_{O_2}$, the involvement of neuromuscular factors cannot be completely ruled out even in the present experiments. Although these limitations should be taken into account when interpreting our data, they were partially compensated for by the fact that we were able to collect a large amount of novel data from top level cyclists with remarkable racing performance records, under reliable well controlled conditions.

CARDIORESPIRATORY WORK

In our investigation, pulmonary ventilation significantly increased during the tests (from about 110 litres/min at the third minute to about 120 litres/min at the end of the test). The latter was achieved mainly by increasing breathing frequency (up to about 40 breaths/min), as tidal volume showed no change during exercise. Ventilatory work may have contributed to the rise in \dot{V}_{O_2} , given that \dot{V}_E/\dot{V}_{O_2} had significantly increased by the end of exercise. Indeed, previous research has shown that the \dot{V}_{O_2} cost of respiratory muscle work needed to increase \dot{V}_E partly contributes to the slow component seen during exercise above the lactate threshold, with increases of about 20–60 litres during heavy and severe exercise.^{1,3}

Nevertheless, the relation between the slow component of \dot{V}_{O_2} and the change in ventilation is not fully established to date. According to some authors, ventilatory work accounts for rather a small percentage of the slow component.^{3,12} For example, Aaron *et al.*³³ suggest that the O_2 cost of ventilation may be 2.85 ml/l within the range of \dot{V}_E values 117–147 litres/min, which is comparable with the range observed in our experiments (about 110–120 litres/min). Using the aforementioned value of 2.85 ml/l, we could estimate that the 12 litre increase observed by us in \dot{V}_E from the third minute to the end of exercise required an additional O_2 uptake of about 34 ml in 17 minutes or about 2 ml/min. This represents 26% of the \dot{V}_{O_2} slow component. This value is in agreement with those (18–23%) reported by others,^{3,12} and suggests that ventilation is not the main determinant of the slow component. In this regard, it must also be kept in mind that, at least during incremental exercise, the ventilatory pattern of professional cyclists is particularly efficient from both the mechanical and metabolic perspective—that is, compared with amateur well trained riders—as recently shown in our laboratory.²¹ In contrast, other reports have suggested a greater contribution of ventilatory work during cycle ergometer tests at 80% of $\dot{V}_{O_2\text{MAX}}$,^{13,31} which could account for as much as 81% of the observed slow component.¹³ Although further research is necessary, our findings suggest an appreciable involvement of ventilatory work to sustain \dot{V}_E values above 100 litres/min at a constant tidal volume of about 3 litres. Further, both the values of \dot{V}_E/\dot{V}_{O_2} and \dot{V}_E/\dot{V}_{CO_2} were: (a) relatively high (about 25.0 and about 27.0 respectively) and similar to those previously reported for professional riders at the workload eliciting VT_2 during incremental exercise²⁰; and (b) significantly increased at the end of the tests. Furthermore, the disproportionate rise in \dot{V}_E with respect to \dot{V}_{CO_2} —that is, significant increase in \dot{V}_E/\dot{V}_{CO_2} —shown by our subjects during exercise suggests there is an additional ventilatory stimulus. In fact, several factors able to stimulate peripheral chemoreceptors such as blood $[K^+]$, adenosine, osmolarity, catecholamines, or temperature are known to show a substantial increase during intense exercise.³⁴ However, as we did not estimate such factors, we propose that future trials should examine the effects of K^+ , catecholamines, and temperature on the \dot{V}_{O_2} slow component in elite endurance athletes.

Mean HR values showed appreciable changes throughout the tests (from 157 to 173 beats/min). The relation between the change in HR and the slow component of \dot{V}_{O_2} has been scarcely reported. Westerlind *et al.*³⁵ estimated that, because of the small size of the heart (0.33 g average), the percentage O_2 cost corresponding to the work performed by the heart would be no greater than 20% of the total \dot{V}_{O_2} . Similarly, in a recent report,³¹ the O_2 cost attributable to the change in rate pressure product was estimated at a mere 10–15% of the total slow component of \dot{V}_{O_2} , in subjects of varying fitness levels. However, Hamilton *et al.*³⁶ showed that,

when the $\dot{V}O_2$ slow component is prevented—for example, with glucose infusion and fluid replacement—HR drift is almost totally eliminated. Although our findings are limited by the fact that we did not measure cardiac output, they suggest that, at least in elite endurance cyclists, the contribution of central factors (cardiac work coupled to that of respiratory muscles) to the slow component is not negligible. Moreover, it has been suggested that high ventilation rates (above 100 litres/min) increase cardiac work through both (a) the pumping action of the lungs, which in turn augments venous return, and (b) the increased metabolic needs of the respiratory muscles.³⁷

METABOLIC FACTORS: LACTATE AND pH

Several reports provide evidence for the involvement of lactate and/or lactic acidosis in the slow component.^{2 4 8 9} Increased lactate levels may lead to increased metabolic activity in the liver and possibly skeletal muscle through stimulation of glycogenolysis.³ Furthermore, some authors⁹ speculate that the reduction in pH associated with lactic acidosis causes a rightward shift in the oxyhaemoglobin dissociation curve as the result of the Bohr effect and permits the continued $\dot{V}O_2$ increase. Here, lactate levels showed a significant increase throughout the constant load tests. However, if present, the contribution of lactate to the slow component in our subjects was possibly of a small magnitude because the lactate concentration was relatively low (mean values of 2–3 mM) and blood pH values remained unchanged throughout the tests.

NEUROMUSCULAR FACTORS

Previous studies have shown that most of the slow component arises from working limbs in both euhydrated³ and dehydrated subjects.¹⁸ Thus the mechanisms responsible for this phenomenon may have their main effect on the exercising skeletal muscles.⁷ In this regard, it has been hypothesised that the slow component reflects, at least partly, a progressive recruitment of less efficient motor units—that is, those composed of Type II fibres—with exercise duration.^{15–17} Previous research has indeed shown the lower efficiency of Type II than Type I fibres.³⁸ To our knowledge, only one previous study, that by Shinohara and Moritani,¹⁶ reports EMG data to corroborate the involvement of neuromuscular factors—for example, patterns of motor unit recruitment and/or firing frequency—in the aetiology of the $\dot{V}O_2$ slow component. These authors found a significant increase in the iEMG of the quadriceps muscle in untrained subjects during constant load cycle ergometer testing at a relative intensity (above the ventilatory threshold) comparable with that used in this study. They concluded that, because increased iEMG reflects changes in motor unit recruitment, the slow component of $\dot{V}O_2$ may have been due to the progressive recruitment of more motor units during exercise, particularly fast ones, to compensate for the reduced power output of fatigued motor units. However, as indicators of both motor unit recruitment (rms-EMG) and

firing rate (MPF) remained almost constant throughout the tests, we cannot claim evidence of neuromuscular fatigue in our subjects. One would indeed expect a decline in MPF³⁹ and a concomitant increase in rms-EMG^{28 40} in the event of neuromuscular fatigue. In this regard, our previous research has shown that rms-EMG kinetics are closely linked to changes in the recruitment patterns of motor units in the participating exercising muscles—for example, vastus lateralis and/or rectus femoris—of professional cyclists and are valid indicators of neuromuscular fatigue in these subjects.^{20 23} Thus our EMG data suggest a considerable resistance to fatigue of recruited motor units, at least at submaximal intensities up to 80% of $\dot{V}O_{2MAX}$. Such adaptation is probably attained after years of highly demanding training (about 35 000 km are covered a year by professional riders), as we suggested in a previous study which compared the physiological response of professional cyclists with that of their elite amateur counterparts.²⁰

In conclusion, a significant but small $\dot{V}O_2$ slow component was shown by professional cyclists during intense constant load exercise. Bearing in mind that (a) the exact mechanism(s) underlying this phenomenon is (are) yet to be established and (b) our findings are limited in that certain possibly related variables, such as leg $\dot{V}O_2$, temperature, K^+ , and catecholamines, were not determined, our results suggest that, in these highly trained athletes, the primary origin of the slow component does not seem to involve neuromuscular factors (changes in fibre recruitment patterns) at workloads up to 80% of $\dot{V}O_{2MAX}$.

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Contributors: A L initiated the research, analysed the data, discussed core ideas and edited the manuscript. J L C designed the main part of the exercise protocol and contributed to the paper. J H coordinated the study (subject selection, testing sessions, etc) and also contributed to the paper. Each of them participated in data collection during the tests. M Pérez, A Carvajal, J Pardo, J R Echevarria and G Toledo assisted the authors in collecting blood and/or gas exchange variables.

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Take home message

Professional cyclists exhibit a small $\dot{V}O_2$ slow component during intense (80% of $\dot{V}O_{2MAX}$) constant load exercise, which does not seem to primarily originate from neuromuscular factors. Although more exhaustive research is required, a multifactorial aetiology involving both central and peripheral factors is proposed.