Oxygen uptake (VO₂) tends to slowly rise during any constant-load exercise of moderate to high intensity, that is, above the lactate threshold (LT), which is the point after which blood lactate shows a consistent, nonlinear increase compared with baseline values (6). This phenomenon, the “slow component of VO₂,” has been defined as the continued rise in VO₂ beyond the third minute of exercise (6). In accordance with this phenomenon, the VO₂:workload relationship was calculated for the following three phases: phase I (below the lactate threshold (LT)), phase II (between LT and the respiratory compensation point (RCP)), and phase III (above RCP). Results: In group P, the mean slope (∆VO₂:∆W) of the VO₂:W relationship decreased significantly (P < 0.01) across the three phases (9.9 ± 0.1, 8.9 ± 0.2, and 3.8 ± 0.6 mL O₂·watts⁻¹·min⁻¹ for phases I, II, and III, respectively). No significant differences (P > 0.05) were found between phases I and II (P > 0.05) in group A, whereas ∆VO₂:∆W significantly increased in phase III (P < 0.01), compared with phase II (10.2 ± 0.3, 9.2 ± 0.4, and 10.1 ± 1.1 mL O₂·watts⁻¹·min⁻¹ in phases I, II, and III, respectively). The mean value of ∆VO₂:∆W for phase III was significantly lower in group P than in group A (P < 0.01). Conclusion: Contrary to the case in amateur riders, the rise in VO₂ in professional cyclists is attenuated at moderate to high workloads. This is possibly an adaptation to the higher demands of their training/competition schedule. Key Words: RAMP TEST, LACTATE, VENTILATION, HEART RATE, SLOW COMPONENT OF VO₂
exercise at 80% VO$_{2\text{max}}$ was only 130 mL in 17 min or 7.6 mL·min$^{-1}$ (15), clearly below values (around 330 mL in 15 min or 22 mL·min$^{-1}$) previously reported for cyclists working at similar relative intensities (6). Despite the high power output sustained by professional cyclists (average of ~400 W), no electromyographic (EMG) evidence was found of what appears to be the main determinant of the slow component phenomenon in non-highly trained humans, that is, an increased recruitment of fast motor units because of fatigue of previously recruited slow ones. Such high resistance to fatigue of slow motor units is probably attained after years of intense training (~35,000 km·yr$^{-1}$).

Three physiological gas exchange phases can be identified during rapid incremental exercise testing (24,27): phase I, in which CO$_2$ production (VCO$_2$) comes mainly from oxidative metabolism; phase II (“isocapnic buffering”), during which VE increases in response to the rise in VCO$_2$ from buffering, with regulation of arterial partial pressure of CO$_2$ (PaCO$_2$); and phase III, in which respiratory compensation for metabolic acidosis with lowering of PaCO$_2$ (“hypocapnic hyperventilation”) occurs. The points that limit these three phases are denoted ventilatory threshold (VT) or LT (between phases I and II) and second ventilatory threshold or respiratory compensation point (RCP) (between phases II and III). The high workloads at which both VT and RCP occur in professional cyclists (~65% and ~90% of VO$_{2\text{max}}$, respectively) and the marked difference between these values and those recorded in amateur cyclists (VT ~60% of VO$_{2\text{max}}$ and RCP ~80% of VO$_{2\text{max}}$, respectively) suggest that these two variables might be an important performance factor in endurance events such as professional road races (17). Accordingly, exercise intensity is often approached as phases I, II, and III, both for exercise prescription and for estimating the physiological demands of cycling competition (17). Moreover, previous studies dealing with the kinetics of physiological variables during incremental exercise have also used this three-segmental approach (9,10,21). It would therefore be of interest to evaluate the VO$_2$ kinetics of professional road cyclists across these three phases during a rapid incremental protocol (i.e., ramp test), to determine if the VO$_2$:workload relationship exhibits an upward deflection at moderate to high workloads (above LT, or phases II and III), as appears to occur in individuals of a lower fitness level.

The main purpose of this study was twofold: 1) to analyze the kinetics of VO$_2$ in professional cyclists across the workloads (phases I, II, and III) of an incremental (ramp) protocol; and 2) to determine the influence of possible determinants of this type of kinetics, namely, central factors (cardiorespiratory work) and blood lactate. An additional goal was to compare the results with those obtained during the same protocol in a group of well-trained, amateur cyclists.

**METHODS**

**Subjects**

After giving their written informed consent, 12 top-level professional road cyclists (group P) and 10 well-trained, amateur cyclists (group A) were selected as subjects for this investigation. Subjects in group P were required to fulfill the following criteria at the time of the study: 1) a competition experience of at least 2 yr in the professional category of the Union Cycliste Internationale (UCI); 2) to have participated in at least two of the main 3-wk stage races (Vuelta a España, Giro d’Italia, or Tour de France); and 3) to have won at least one event of the UCI professional competition season within the last 2 yr. Several of the subjects in group P were among the best cyclists in the world (including a former world champion and winners of major professional tour races). Subjects in group A were required to 1) be enrolled in a licensed amateur team and to have a competition experience in the “sub23-elite” category of the UCI of at least 2 yr. All were highly competitive in the amateur category but had never competed in the professional category. Subjects’ mean (± SEM) age, height, and weight were 25 ± 1 yr, 181.4 ± 2.1 cm, and 70.4 ± 1.5 kg, respectively (group P); and 22 ± 1 yr, 177.0 ± 1.0 cm, and 69.9 ± 1.6 kg, respectively (group A). The study protocol was approved by the institution’s (Universidad Complutense) research ethics committee.

**Exercise Protocol**

**Exercise tests.** Subjects from each group performed an exercise test (ramp protocol) until exhaustion on a cycle ergometer (Ergometrics 900, Ergo-line, Barcelona, Spain). All the tests were performed during the months December and January under similar environmental conditions (20–24°C, 45–55% relative humidity). The subjects were cooled with a fan throughout the bouts of exercise. This type of protocol has been used for the physiological evaluation of professional cyclists in several previous studies (13,15,16,18). After a 2-min rest, sitting on the cycle ergometer, the test was started at 25 watts and the workload was increased by 25 watts·min$^{-1}$. Subjects adopted the conventional sitting position during the whole duration of the tests and were allowed to choose their preferred cadence (above LT, or phases II and III), as appears to occur in individuals of a lower fitness level.

The main purpose of this study was twofold: 1) to analyze the kinetics of VO$_2$ in professional cyclists across the workloads (phases I, II, and III) of an incremental (ramp) protocol; and 2) to determine the influence of possible determinants of this type of kinetics, namely, central factors (cardiorespiratory work) and blood lactate. An additional goal was to compare the results with those obtained during the same protocol in a group of well-trained, amateur cyclists.
An increase of at least 0.5 mM BLa concentration was observed at subsequent exercise as the highest not associated with a rise in lactate concentration-work relationship in both groups. In group P, the mean value increased significantly (P<0.01) across the three phases. On the other hand, the mean value of partial pressure of oxygen (P_{\text{ET}}O_2) and carbon dioxide (P_{\text{ET}}CO_2).

Capillary blood samples (50 μL) for the measurement of blood lactate (BLa) were taken from fingertips (sampling period of 15–20 s) at rest, every 3 min throughout the test (e.g., at the end of the stages 75 W, 150 W, 225 W, etc.), and immediately after termination of exercise. BLa was determined using an automated analyzer (YSI 1500, Yellow Springs Instruments, Yellow Springs, OH). The LT was determined using a method described by Weltman and et al. (28) (Fig. 1). This method defines the workload (W) corresponding to LT and RCP respectively, and at the maximal power output (W_{\text{max}}).

Regression equations of A and P are shown in the upper and lower part of the figure, respectively. LT, lactate threshold; RCP, respiratory compensation point; W_{\text{max}}, maximal power output; I, II, and III, phases I (below LT), II (LT to RCP), and III (above RCP), respectively.

**Data Analysis**

The regression lines of the V\dot{O}_2\text{-W}, HR\cdot\text{W}, VE\cdot\text{W}, VE\cdot V\dot{O}_2^{-1}\cdot\text{W}, and BLa\cdot\text{W} relationships were calculated in each subject for the aforementioned phases (phase I, below LT; phase II, LT to RCP; and phase III, above RCP). In each group, a one-way repeated-measures ANOVA was applied to determine if there was a significant difference between 1) phases I, II, and III in the mean slope (\Delta V\dot{O}_2:\Delta W) of the V\dot{O}_2:W relationship; and 2) mean values of mechanical efficiency at LT, RCP, and W_{\text{max}} respectively. When a significant difference was found in ANOVA tests, the post hoc Scheffé test was applied. A Student’s t-test for paired data was applied to compare the following variables in groups A and P: 1) mean values of cadence and physiological parameters (e.g., V\dot{O}_2_{\text{max}}, W_{\text{max}}); 2) mean values of \Delta V\dot{O}_2:\Delta W obtained during phases I, II, and III, respectively; and 3) mechanical efficiency at VT, RCP, and W_{\text{max}}.

Pearson product-moment correlation coefficients were calculated for both groups of subjects to determine whether there was a significant relationship within each of the phases I, II, and III, between \Delta V\dot{O}_2:\Delta W and the following slopes: \Delta HR:W, \Delta VE:\Delta W, \Delta VE-V\dot{O}_2^{-1}:\Delta W, and \Delta BLa:\Delta W. These correlations were calculated to determine the possible influence of cardiorespiratory factors and BLa on V\dot{O}_2 kinetics. The level of significance was set at 0.05 for all the statistical tests and results are expressed as means ± SEM.

**RESULTS**

Mean cadence and physiological variables recorded during the tests are shown in Table 1. The workload (expressed in W and in %W_{\text{max}}) eliciting LT and RCP was significantly higher in group P than in group A (P<0.05).

Figure 1 shows the mean regression lines of the V\dot{O}_2:W relationship in both groups. In group P, \Delta V\dot{O}_2:\Delta W decreased significantly (P<0.01) across the three phases (Table 2). In group A, no significant differences (P>0.05) were found between phases I and II (P>0.05), whereas \Delta V\dot{O}_2:\Delta W significantly increased in phase III compared with phase II (P<0.01). On the other hand, the mean value of \Delta V\dot{O}_2:\Delta W for phase III was significantly lower in group P than in group A (P<0.01).
TABLE 2. Slopes (\(\Delta V_{O_2}/\Delta W\), in mL O\(_2\)/watts\(^{-1}\)/min\(^{-1}\)) of the regression lines corresponding to the tests in amateur (group A) and professional cyclists (group P).

<table>
<thead>
<tr>
<th>Phase</th>
<th>Group P</th>
<th>Group A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase I (below LT)</td>
<td>9.9 ± 0.1</td>
<td>10.2 ± 0.3</td>
</tr>
<tr>
<td>Phase II (LT to RCP)</td>
<td>8.9 ± 0.2*</td>
<td>9.2 ± 0.4*</td>
</tr>
<tr>
<td>Phase III (above RCP)</td>
<td>3.8 ± 0.6**</td>
<td>10.1 ± 1.1</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SEM. LT, lactate threshold; RCP, respiratory compensation point; NS, no statistical significance (\(P > 0.05\) for group P vs group A).

\* \(P < 0.01\) for phase II vs phase III; ** \(P < 0.01\) for phase III vs both phases I and II.

Mean values of mechanical efficiency in both groups are shown in Table 3. Briefly, mechanical efficiency increased in group P with increasing workloads (\(P < 0.01\)) in the following order: VT < RCP < \(W_{\text{max}}\). In group A, no significant differences were found between VT and RCP (\(P > 0.05\)), and mechanical efficiency significantly increased at \(W_{\text{max}}\) compared with both VT and RCP (\(P < 0.01\) and \(P < 0.05\), respectively). Mean values of mechanical efficiency at both RCP and \(W_{\text{max}}\) were significantly higher in group P than in group A (\(P < 0.05\) and \(P < 0.01\), respectively). Finally, no significant correlation was found between \(\Delta V_{O_2}/\Delta W\) and \(\Delta H_R\). \(\Delta V_{E} \cdot \Delta W\), \(\Delta V_{E} \cdot V_{O_2} \cdot \Delta W\), and \(\Delta B L_{A} \cdot \Delta W\) in groups P and A for each of the three phases.

DISCUSSION

The purpose of this study was to analyze the kinetics of \(V_{O_2}\) in professional cyclists during a ramp protocol and to determine the possible influence of cardiorespiratory factors and lactate on this phenomenon. We also analyzed \(V_{O_2}\) kinetics in a group of well-trained, amateur cyclists of a lower fitness level. In line with previous research, three physiological gas exchange phases were considered (i.e., below LT, LT to RCP, and above RCP (9,10,21)). Our main finding was that, in professional riders, the rate of the \(V_{O_2}\) rise elicited by gradual exercise (ramp protocol) decreases at moderate to high workloads (i.e., from the LT (~300 watts) to the maximal attainable power output (~500 watts)). Similarly, mechanical efficiency seemed to increase with rising exercise intensity. To the best of our knowledge, very few data from top-level endurance athletes have been generated in this field of research, and neither have findings related to \(V_{O_2}\) kinetics at such high power outputs (from ~300 to ~500 watts) been published to date. In contrast, the results of previous research on subjects of a much lower fitness level have shown a nonlinear increase in \(V_{O_2}\) at high workloads.

VO\(_2\) KINETICS IN PROFESSIONAL CYCLISTS

VO\(_2\) kinetics in groups P and A differ from one another. In group P, the slope of the \(V_{O_2}\) rise below the LT (~10 mL O\(_2\)/watts\(^{-1}\)/min\(^{-1}\)) was within “typical” limits for healthy, non-highly trained adults during incremental cycle ergometer tests (i.e., ~9–11 mL O\(_2\)/watts\(^{-1}\)/min\(^{-1}\) (22)), whereas it was below these limits after the LT was reached (mean \(V_{O_2}\) rise of 8.9 and 3.8 mL O\(_2\)/watts\(^{-1}\)/min\(^{-1}\) in phases II and III, respectively). In fact, the \(V_{O_2}\) kinetics recorded at high workloads in our group of amateur cyclists was closer to that expected to occur in healthy, non-highly trained subjects than to that shown by professional riders; amateur cyclists showed a mean \(V_{O_2}\) rise of > 9 mL O\(_2\)/watts\(^{-1}\)/min\(^{-1}\) at each workload, and \(\Delta V_{O_2}/\Delta W\) significantly increased above the RCP. A further finding was that \(V_{O_2}\) kinetics in both groups were not significantly influenced by lactate or cardiorespiratory work.

As mentioned above, the occurrence of a deflection in the \(V_{O_2}/W\) relationship above the LT in group P differs from that previously described during gradual exercise in subjects of a lower fitness level (2,12,29,30). The ramp protocol chosen for the present investigation (workload increases of 25 watts-min\(^{-1}\)) and also for numerous studies performed on elite cyclists (13,15,16,18) differs from that used in previous research in the area of \(V_{O_2}\) kinetics (i.e., workload increases every 3–4 min (3,12,29,30)). Nevertheless, in a recent experiment in our laboratory (unpublished data), we also noted a significant “excess” of \(V_{O_2}\) in sedentary, young men during high ramp protocol workloads comparable to those applied here (workload increases of 5 watts-15 s\(^{-1}\), averaging 25 watts-min\(^{-1}\)). The results recorded for group P reflect one of the many adaptations to professional road cycling compared with amateur cycling—that is, a greater mechanical efficiency at high workloads—which may explain their higher performance level. Indeed, the mean \(V_{O_2\text{max}}\) values corresponding to groups A and P were very similar (~70 mL·kg\(^{-1}\)·min\(^{-1}\)) despite the greater performance of the latter (i.e., considerably higher values of both maximal power output and power output at the RCP). We recently determined \(V_{O_2}\) kinetics in a group of professional riders of similar characteristics during a 20-min constant-load cycle ergometer test at 80% \(V_{O_2\text{max}}\) (15). Despite the high average power output sustained by the professional riders (~400 watts), the average magnitude of the slow component (130 mL in 17 min or 7.6 mL·min\(^{-1}\)) was considerably lower than that reported by previous research (22 mL·min\(^{-1}\)) on the basis of constant-load cycle ergometer tests performed at similar relative intensities (6). This finding is also suggestive of a great cycling efficiency of professional riders, which is thought to contribute to their renowned ability to sustain extremely high workloads over long periods, as shown by previous field research (e.g., average power output > 400 watts during the 1-h record in a velodrome over the past decade (17)). Professional riders show considerable resistance to fatigue of recruited motor...
units, at least at high, submaximal intensities (17,18). Such an adaptation is probably attained after years of highly demanding training and competition (i.e., ~35,000 km·yr⁻¹ and ~90 competition days) as suggested in a previous study in which the physiological response of professional cyclists was compared with that of their elite, amateur counterparts (18).

Ramp tests involving gradual, constant workload increases such as the one used here may be the most suitable for determining the possible influence of motor unit recruitment patterns on VO₂ kinetics, since they can reproduce the hierarchy of muscle fiber recruitment that is likely to occur with rising exercise intensity in humans, that is, type I (phase I) → type IIA (phase II) → type IIX (phase III), as shown by previous EMG (25) and biopsy studies (26). Given that 1) the nonlinear increase in VO₂ after the LT is mainly linked to the recruitment of type II fibers in non-highly trained humans (6) and that 2) subjects in group P failed to show this response, one may speculate that the latter may have a greater proportion of type I fibers in the main muscles involved in cycling than their amateur counterparts. Previous research performed on amateur, well-trained cyclists has indeed shown that cycling efficiency during heavy exercise (above LT) is positively related to the percentage of type I fibers in the vastus lateralis muscle (11). As mentioned before, a higher resistance to fatigue of slow motor units in group P could have also been involved, and would allow these professional riders to reach moderate to high workloads (i.e., between LT and RCP, or ~70–90% VO₂max) before significant recruitment of the less-efficient type II fibers. However, it must be also kept in mind that the relationship between the rise in VO₂ and the rise in power output described here and in previous research involving gradual exercise tests (2,12,29,30) does not directly reflect the efficiency of muscle contraction (i.e., myosin cross-bridge cycling, which is directly related to force production) but rather the increase in aerobic metabolism as muscle power output increases (2). Indeed, other ATP-dependent, non-crossbridge activities termed “activation heat” (i.e., calcium uptake and release from the sarcoplasmic reticulum) could also affect VO₂ kinetics during gradual exercise. Nevertheless, it remains unclear whether activation heat varies or remains relatively constant during fatiguing exercise (1). Thus, it is not possible to determine to what extent the attenuation of the VO₂ rise exhibited by group P at high intensities solely reflects a higher efficiency of muscle contraction, a smaller contribution of activation heat compared with subjects of a lower fitness level, or both.

Several potential contributors to VO₂ kinetics in non-highly trained humans (i.e., cardiorespiratory work, and lactate and/or lactic acidosis) seemed to play a minor role in our subjects. In non-highly trained individuals, the VO₂ cost of cardiorespiratory work might partially contribute to the slow component seen during exercise above the LT (4,7). Pulmonary ventilation and HR significantly increased in group P to reach high values at the end of the tests (VE ~200 L·min⁻¹ and HR ~190 beats·min⁻¹), but such high cardiorespiratory work did not seem to influence VO₂ kinetics. Indeed, we found no correlation between ΔVO₂:ΔW and any of the variables related to cardiorespiratory work (ΔHR: ΔW, ΔVE:ΔW, or ΔVE·VO₂⁻¹·ΔW). It may be speculated that the deflection of the VO₂ rise recorded in group P as opposed to both amateur riders and nontrained subjects is partly attributable to more efficient cardiac function during moderate- to high-intensity exercise (above LT). Indeed, it was recently shown that approximately two thirds of professional cyclists exhibit a deflection in the HR:W relationship at high intensities (~RCP) during a ramp protocol, which is partly determined by their great myocardial wall thickness (14). Professional riders, on the other hand, have been shown to exhibit a characteristic breathing pattern at the high workloads of a ramp protocol (i.e., lack of “tachypneic shift”) (13). This breathing adaptation might allow their respiratory muscles to work more efficiently in both mechanical and metabolic terms (13), and could thus at least partly account for the VO₂ kinetics shown by these subjects. In fact, the oxygen cost of breathing in highly fit individuals is not negligible (close to 15% VO₂max) (8). Furthermore, the work of breathing during heavy exercise compromises leg blood flow to working limb muscles, possibly because of sympathetically mediated vasoconstriction originating in the respiratory musculature (8). In the event of a more efficient breathing pattern (i.e., subjects in group P), blood flow to working limbs may not be significantly reduced by the aforementioned mechanism. This in turn would lead to a later recruitment of type II fibers, which predominantly undertake anaerobic metabolism and are less efficient than type I fibers (5). Finally, we found no correlation between VO₂ and lactate kinetics in any of the three phases. These results suggest that lactate per se does not enhance whole-body O₂ in highly fit endurance athletes during gradual exercise. In contrast, findings related to non-highly trained humans provide evidence for the involvement of lactate and/or lactic acidosis in the VO₂ slow component that occurs at high exercise intensities (above LT) in both constant-load (23) and incremental protocols (12).

In conclusion, the rate of the VO₂ rise elicited by gradual exercise (ramp protocol) decreases at moderate to high workloads (from LT to maximal power output) in professional cyclists. This response is different to that shown by most individuals (including well-trained cyclists of a lower performance level) and probably reflects one of the main adaptations to professional road cycling, that is, a great ability to tolerate high workloads (at or above the RCP) over long periods of time (>30 min) before fatigue occurs. Our findings suggest the possibility of using VO₂ kinetics during “routine” incremental testing of elite endurance athletes, as an indicator of both performance and training adaptation (11).

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