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NEW IDEAS: Metabolic Responses To Exercise

THE VO₂ OVERSHOOT AT THE ONSET OF CONSTANT-LOAD EXERCISE IN ELITE CYCLISTS: AN UNDESCRIBED PHENOMENON

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ABSTRACT

THE VO₂ OVERSHOOT AT THE ONSET OF CONSTANT-LOAD EXERCISE IN ELITE CYCLISTS: AN UNDESCRIBED PHENOMENON. **A.R. Hoogeveen, H.A. Keizer.** *JEPonline*. 2003;6(4):34-41. In prior exercise tests in two independent laboratories we observed a VO₂ overshoot at the onset of constant-load exercise in well-trained cyclists. The purpose of this study was to investigate this previously unreported phenomenon. Fifteen male elite cyclists (age 26.3±3.3 years, height 180.7±5.3 cm, body mass 72.5±3.6 kg) underwent a constant-load exercise test (250 Watts) on a cycle ergometer. Ventilation and gas exchange kinetics were determined during this constant-load exercise. Gas exchange data were measured breath-by-breath and signal-averaged data were fit with a sixth degree polynomial function. The mean submaximal VO₂ during steady state was 3320±128 mL/min. The mean R² of the polynomial functions was 0.93±0.04. The mean maximum VO₂ at the curve maximum derived from the polynomial function was 3528±144 mL/min. The mean maximum VO₂ overshoot was reached after 76.9±13.0 s (range 65-90 s) following the onset of exercise. In three subjects we did not observe a clear VO₂ overshoot and the curve clearly showed a mono-exponential pattern. The VO₂ overshoot is in contrast with the classic exponential kinetic models in which the VO₂ is continuously increasing. The VO₂-overshoot phenomenon will highly influence the analysis of the VO₂ response with mono- or bi-exponential models and the determination of the oxygen deficit. In this article, we introduce an additional switch-on component at the onset of exercise. For analysing the short-term VO₂ response in the future we advise to add a switch-on component to the classic exponential equations. The physiological mechanism of the VO₂ overshoot has to be investigated.

Key Words: VO₂ overshoot, Oxygen deficit, VO₂ Kinetics

INTRODUCTION

Oxygen uptake kinetics has been extensively studied since the first report of the exponential nature of gas exchange responses during constant-load exercise in 1913 (1-6). The oxygen uptake (VO₂) following a step increase in work rate can be characterised as consisting of an early cardiorespiratory component with unchanged mixed-venous O₂ content (phase I) and a subsequent metabolic component (phase II) starting when venous blood from the muscle arrives at the lungs (2,3,5,6). The cardiorespiratory component plays a role in VO₂ kinetics in the first 20-30 s following a rest-exercise transition (2,6).

Xu and Rhodes (5) have recently reviewed the second phase or metabolic component of the VO₂ response at the onset of exercise. The response at exercise intensities below the lactate threshold can be described by a mono-exponential mathematical equation (see discussion). During heavy exercise the VO₂ response becomes more complex and consists of two components. The initial response still increases exponentially, however after some minutes an additional component is developed slowly causing an increase in VO₂ during the constant load exercise (VO₂ drift). The possible mechanisms underlying these fast and slow components are unclear.

Over the last few years and in two independent laboratories we observed a remarkable VO₂ overshoot phenomenon at the beginning of constant-load tests in very well trained endurance athletes. Just before reaching the level of steady state, in the first two minutes of exercise, the VO₂ peaks clearly above the steady-state level reached in the following minutes. As far as we know this observation has never been described before. This VO₂ overshoot would be in contrast with the conventional mono- or bi-exponential model, extensively described by Leyk et al. (2), Xu et al. (5), and Yoshida et al. (6).

The purpose of this study was to describe and discuss this VO₂ overshoot phenomenon at the onset of constant-load exercise. The initial VO₂ response was investigated in well-trained endurance cyclists and the characteristics and the possible mechanisms are discussed.

METHODS

Subjects

Fifteen professional and elite amateur cyclists volunteered to take part in this study (age 26.3±3.3 years, height 180.7±5.3 cm, body mass 72.5±3.6 kg) and gave their written informed consent. Percentage of body fat was estimated using the measurements of four skin folds according to Durnin and Womersley (7).

The subjects were free from serious diseases and took no drugs or medication during the course of the study. In addition, all subjects completed all testing free of acute disease or infection. The subjects had been training regularly for at least 6 years (9.1±3.6 yr.). The exact volume and intensity of training and competition for each subject was not recorded. However, the mean training duration, including competition, was 13.3±4.6 hours/week in the period of 4 months before the test.

Testing procedures

The subjects refrained from intensive exercise in the previous 24 hours, and did not compete for 72 hours prior to testing. Subjects were instructed to take a carbohydrate rich diet the day before the exercise test. Before the start of each test, a warm-up of 5 min was performed which consisted of cycling with a load of 50

Watts. After this, subjects rested for 2 min, and then the workload suddenly (delay <1 second) was increased to 250 Watts for 5 min.

All measurements took place under the same conditions of temperature (22 ° Celsius) and humidity (55%). The sitting position on the cycle-ergometer (Excalibur-sport, Lode, Groningen) was adapted individually for each subject to create the same position as in competition. An ECG-device (Jaeger BeNeLux BV, Breda, Netherlands) was used to obtain a 12-lead ECG every minute. The subjects selected their own cadence, but had to remain between 80 to 100 rev/min.

The subjects breathed through a mask, which was connected by an elastic tube to an automated expired gas analysis indirect calorimetry system (Oxycon, Jaeger BeNeLux BV, Breda, Netherlands). The mask dead space was 65 mL. Expired air was continuously sampled and analysed for O₂, CO₂ and volume. These instruments were coupled to a computer, which plotted power output against VO₂, VCO₂, minute ventilation (VE) and heart rate (Software Jaeger, Netherlands).

The volume transducer was calibrated before every test using a 3L-calibration syringe. The CO₂ and O₂ gas analysers were calibrated manual by means of a gas bag with a known calibration gas sampled via the twin tube, and automatically using a bottle of calibration gas by means of a computer controlled manoeuvre. A fully automatic pneumatic system check of the analysers and sampling system was performed every test including the tubes, valves, and the calibration factors of the analysers.

Gas-exchange data were continuously measured breath-by-breath and signal-averaged data were fit with a sixth degree polynomial function using Excel (Microsoft Corporation, Seattle, USA. 1997). The VO₂ used for sampling and calculations was the mean of every 5 seconds (Fig 1). The reason for using this data handling was that this polynomial function made it possible to describe and analyse the curves. The curve maximum, using the polynomial function, and the highest 5-second VO₂ were obtained (Fig 1). Heart rate, VE and VCO₂ were analysed the same way. Steady-state values at the different workloads were defined as the mean values in the last 120 seconds.

RESULTS

The percentage of body fat was 9.3±1.4 %. The mean VO₂, VCO₂, VE, and heart rate during steady state are shown in Table 1. The mean maximum of the VO₂ curve using the polynomial function was 3528±144 mL/min. The maximum of the VO₂ overshoot was reached at a time of 76.9±13.0 s (range 65-90 s) after the onset of exercise (Figure 1). The highest 5-second VO₂ average (Figure 1) was 3655±188 mL/min and this maximum was reached after 73.1±11.4 s (range 58-88 s). The mean R² of the polynomial functions was 0.93±0.04.

Table 1. The Mean? SD data for steady state VO₂, VCO₂, VE and heart rate.

<i>Variable</i>	<i>Mean?SD</i>
<i>VO₂ (mL/min)</i>	3320? 120
<i>VCO₂ (mL/min)</i>	3129? 113
<i>VE (L/min)</i>	75? 11
<i>HR (beats/min)</i>	134? 11

The cadence rate was 91.5±3.1 rev/min (range 85–96). The curves of VE, VCO₂ and heart rate did not show any sudden increase and fit well into a mono-exponential model. In three subjects we did not observe a clear VO₂ overshoot and the curve clearly showed a mono-exponential pattern (Figure 2).

Table 2. The resulting formulae for the different models of VO₂ kinetics during rest to steady-state exercise.

Equation	Model	Formulae
I	Monoexponential	$VO_2(t) = VO_{2baseline} + A(1 - e^{-(t-Td)/\tau})$
II	Biexponential	$VO_2(t) = VO_{2baseline} + A_1(1 - e^{-(t-Td_1)/\tau_1}) + A_2(1 - e^{-(t-Td_2)/\tau_2})$
IIIa	Overshoot	$VO_2(t) = A.t.e^{-B.t}$
IIIb	Overshoot	$VO_2(t) = A.(t-Td).e^{-(t-Td)/\tau}$

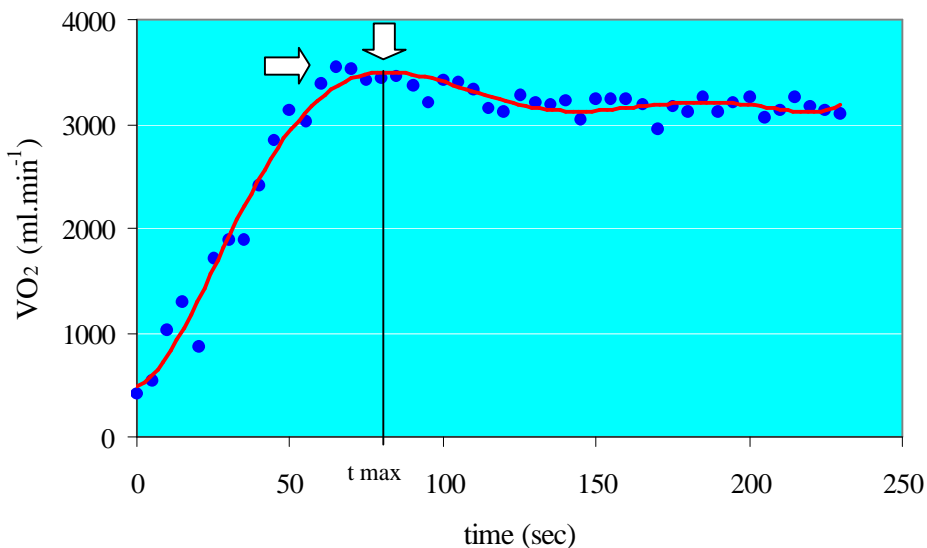


Figure 1. Typical VO₂ response of one of the subjects. The polynomial function is drawn through the measurements. The vertical arrow marks the maximum of this function. The horizontal arrow marks the highest 5 second average. The VO₂ in the first minutes can be described as follows: $VO_2 t = 2.1 E^{-9} t^6 - 1.3 E^{-6} t^5 + 3.75 E^{-4} t^4 - 4.8 E^{-2} t^3 + 2.4 t^2 + 9.5 t + 488$; $R^2 = 0.97$

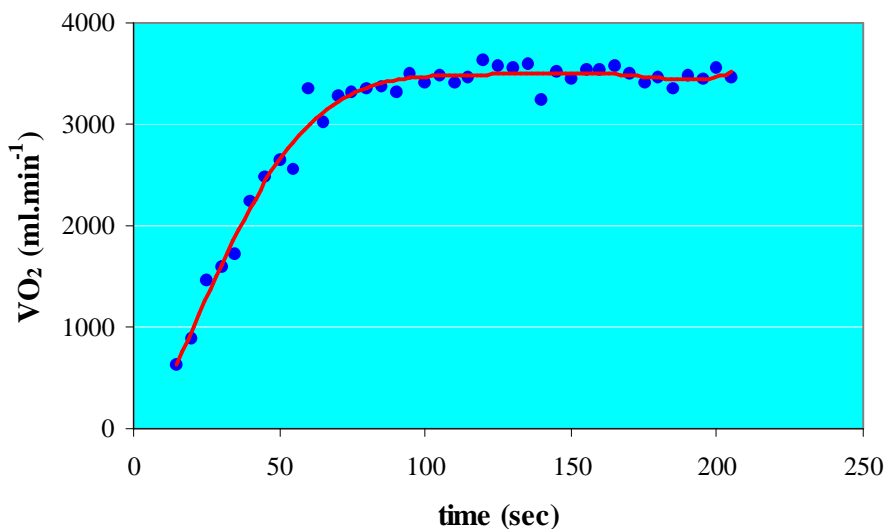


Figure 2. Data from one of the subjects who did not show a clear VO₂ overshoot.

DISCUSSION

In this study, the initial VO₂ response to constant-load exercise was investigated in trained cyclists. A curious observation in this study was the VO₂ overshoot within the first two minutes before reaching the steady-state VO₂. The VO₂ overshoot was also demonstrated in the data of Green et al. (8) and Brunner-La Rocca et al. (9), however they did not remark on and describe this phenomenon.

The VO₂ response following a step increase in work rate can be characterised as consisting of an early cardiorespiratory component with unchanged mixed-venous O₂-content (phase I) and a subsequent metabolic component (phase II) starting when venous blood from the muscle arrives at the lungs (2,3,5,6). Yoshida et al. (6) investigated the rest-exercise transitions and observed that VO₂ rapidly increased with time constants of 6.8-7.3 s. The cardiac output (Q) also showed a similar rapid increment with time constants of 6.0-6.8 s with an apparent increase in stroke volume (SV). In Yoshida's study (6), VO₂ increased in phase I up to 30%-34% of the steady-state value and Q increased up to 58%-87% of the steady-state value. Thereafter, some 20 s after the onset of exercise, a mono-exponential increase to steady-state occurred both in VO₂ and cardiac output with time constants of 26.7-32.3 and 23.7-34.4 s, respectively. Leyk et al. (2) also observed an initial, mainly cardiovascular component over the first 30 s, followed by a second, metabolic component at constant Q. Leyk et al. (2) postulated two early features. First, a very rapid VO₂ increase during the initial breathing cycles at the onset of exercise. This was probably caused by events in the lungs or the pulmonary circulation. Second, the Q attained its steady-state level in about 10 s. This suggested a sudden increase in venous return (2).

In the current study the maximum of the VO₂ overshoot was reached between 55–90 s in to the rest to exercise transition, while the early cardiorespiratory phase is finished within 20-30 s (2,5). Furthermore the VO₂ reached during the cardiorespiratory phase is according to Yoshida et al. (6) only 30% of the finally reached steady-state VO₂. Since the VO₂ overshoot in the current study reached a maximum after 30 second and at a higher level than 30% of the steady-state VO₂, the early fast cardiorespiratory phase will probably not be responsible for the overshoot in VO₂.

The metabolic component of the VO₂ response (phase II) at the onset of exercise has been thoroughly studied and was recently extensively reviewed by Xu and Rhodes (5). The response at exercise intensity below the lactate threshold can be described by a mono-exponential mathematical equation (Formula I of Table 2). During heavy exercise the VO₂ response becomes more complex and consists of two components (Formula II of Table 2). Based on prior research and interpretation, the initial response was viewed to always increase mono-exponentially. However after some minutes an additional response is developed slowly causing an increasing VO₂ during the constant load (VO₂ drift). The possible mechanisms underlying these fast and slow components are not clear. In both formulae, the VO₂ as a function of time always increases. So this theoretical model does not fit with the observations in the current study in which a VO₂ peak occurs between 50 and 100 s after the onset of exercise, followed by a decrease to steady state. In addition, the curve fitting in the exponential models will be highly affected by the VO₂ overshoot and will influence the calculation of the O₂ deficit (8).

The dynamics and interpretation of ventilation and gas exchange during exercise have been studied thoroughly (3,5,10). These issues are very complicated and not easy to explain. Perhaps the VO₂ overshoot is a mechanism to overcome the early O₂ deficit. Since other authors already described faster VO₂ kinetics following training and in already trained subjects the VO₂ overshoot may also be an indication of an endurance-trained state (4,12-14). Based on Fick's principles, an increased oxygen uptake is the result of an increased cardiac output or arterial-venous oxygen difference ((a-v)O₂). In the current study the heart rate did not show a sudden initial increase in the first minutes so the increased oxygen uptake could be the result of an increased stroke volume, an increased (a-v)O₂, or a combination of the two. Since heart rate did not show a sudden initial increase, the oxygen pulse showed the same phenomenon as the VO₂. Thus a temporary maximum in stroke volume remains a possibility.

Another possibility is that during the sudden increase in exercise intensity, the near immediate increases in ventilation may be exaggerated based on physiological needs and thus mismatched to cardiac output. This would cause a slight increase in the lung volume at end tidal volume and an inflation of whole body VO₂.

Another explanation may be that a sudden opening of arterioles and pre-capillary sphincters would enhance muscle perfusion resulting in faster and/or more oxygen extraction. Experiments in humans indeed have shown a sudden increase in perfusion of exercising muscles at the onset of work intensity (15,16). Therefore, muscle perfusion is a good possibility to explain the new-described overshoot phenomenon. In addition, a temporary peak of catecholamines or the activation of the muscle pump itself may play a role in the enhanced peripheral perfusion.

Finally, an explanation may be the extra VO₂ that is needed for the legs to start moving and for accelerating the pedals. However this counts only for the first seconds. Furthermore rest-exercise transitions are sometimes associated with contractions of trunk or arm muscles, which may induce effects on ventilation and circulation.

Apart from the question concerning the possible mechanism, it should be pointed out that the classic mono- and bi-exponential models do not agree with our observations. Our method of analyses using a polynomial function can be an issue of discussion but this mathematical function certainly fits well in the observed curves and made the analysis of the curves more easily.

To describe the VO₂ response and VO₂ kinetics to constant-load exercise, we introduce an additional component. The behaviour of this third component can be viewed as a switch-on phenomenon as a result of the start of a linear dynamic system. A possible mathematical equation is shown in formula IIIa (Table 2) and an example of the curve of this component is shown in Figure 3. In this equation a delay time can be inserted and a time constant can be used instead of the constant factor B (Formula IIIb, Table 2). In Figure 4 this component is added to the fast component of the classic mono-exponential model (Formula 1, Table 2). This results in the typical VO₂ overshoot observed in the current study. At higher loads, the classic slow component can be added (second part of Formula 2). It is obvious that our possible model with two or three components fits better than the classic mono- and bi-exponential model, since in the conventional model, the VO₂ is continuously increasing as function of the time. For analysing VO₂ kinetics in the future, we advise to

use our theoretical model. Since computer programs to analyse this three-component model are not yet available, curve fitting with a higher degree polynomial function is an alternative.

In summary, in this study we described the VO₂ response to the rest to steady state exercise transition. We have observed a previously unreported VO₂ overshoot during the first minutes before reaching the steady-state VO₂. The possible physiological explanations of this phenomenon have been discussed but are not clear. Further research will be necessary to hypothesize and test the physiological determinants of the VO₂ overshoot phenomenon.

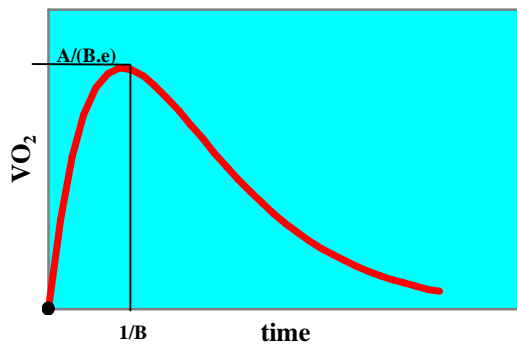


Figure 3. An example of a curve expressed by Formula IIIa or IIIb (see text).

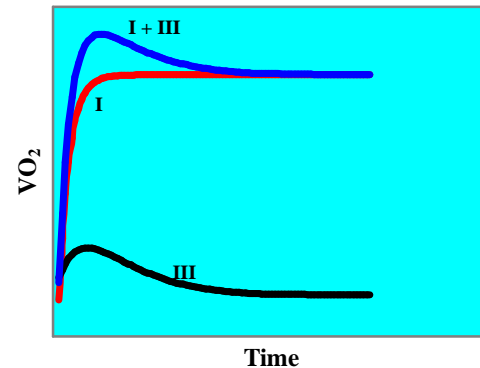


Figure 4. The third component (III) component is added to the fast component of the classic mono-exponential model (I). This results in the typical VO₂ overshoot observed in this study (I+III).

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