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ORIGINAL ARTICLE

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Detection of the change point in oxygen uptake during an incremental exercise test using recursive residuals: relationship to the plasma lactate accumulation and blood acid base balance

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Abstract The purpose of this study was to develop a method to determine the power output at which oxygen uptake ($\dot{V}O_2$) during an incremental exercise test begins to rise non-linearly. A group of 26 healthy non-smoking men [mean age 22.1 (SD 1.4) years, body mass 73.6 (SD 7.4) kg, height 179.4 (SD 7.5) cm, maximal oxygen uptake ($\dot{V}O_{2\,max}$) 3.726 (SD 0.363) $l \cdot min^{-1}$], experienced in laboratory tests, were the subjects in this study. They performed an incremental exercise test on a cycle ergometer at a pedalling rate of 70 $rev \cdot min^{-1}$. The test started at a power output of 30 W, followed by increases amounting to 30 W every 3 min. At 5 min prior to the first exercise intensity, at the end of each stage of exercise protocol, blood samples (1 ml each) were taken from an antecubital vein. The samples were analysed for plasma lactate concentration $[La]_{pl,}$ partial pressure of O_2 and CO_2 and hydrogen ion concentration $[H^+]_b$. The lactate threshold (LT) in this study was defined as the highest power output above which $[La^-]_{pl}$ showed a sustained increase of more than 0.5 $mmol \cdot l^{-1} \cdot step^{-1}$. The $\dot{V}O_2$ was measured breath-by-breath. In the analysis of the change point (CP) of $\dot{V}O_2$ during the incremental exercise test, a two-phase model was assumed for the 3rd-min-data of each step of the test: $X_i = at_i + b + \varepsilon_i$ for $i = 1, 2, \ldots, T$, and $E(X_i) > at_i + b$ for $i = T+1, \ldots, n$, where $X_1, \ldots, X_n$ are independent and $\varepsilon_i \sim N(0, \sigma^2)$. In the first phase, a linear relationship between $\dot{V}O_2$ and power output was assumed, whereas in the second phase an additional increase in $\dot{V}O_2$ above the values expected from the linear model was allowed. The power output at which the first phase ended was called the change point in oxygen uptake (CP-$\dot{V}O_2$). The identification of the model consisted of two steps: testing for the existence of CP and estimating its location. Both procedures were based on suitably normalised recursive residuals. We showed that in 25 out of 26 subjects it was possible to determine the CP-$\dot{V}O_2$ as described in our model. The power output at CP-$\dot{V}O_2$ amounted to 136.8 (SD 31.3) W. It was only 11 W – non significantly – higher than the power output corresponding to LT. The $\dot{V}O_2$ at CP-$\dot{V}O_2$ amounted to 1.828 (SD 0.356) $l \cdot min^{-1}$ was [48.9 (SD 7.9)% $\dot{V}O_{2\,max}$]. The $[La^-]_{pl}$ at CP-$\dot{V}O_2$, amounting to 2.57 (SD 0.69) $mmol \cdot l^{-1}$ was significantly elevated ($P < 0.01$) above the resting level [1.85 (SD 0.46) $mmol \cdot l^{-1}$], however the $[H^+]_b$ at CP-$\dot{V}O_2$ amounting to 45.1 (SD 3.0) $nmol \cdot l^{-1}$, was not significantly different from the values at rest which amounted to 44.14 (SD 2.79) $nmol \cdot l^{-1}$. An increase of power output of 30 W above CP-$\dot{V}O_2$ was accompanied by a significant increase in $[H^+]_b$ above the resting level ($P = 0.03$).

Key words Acid-base balance · Exercise · Oxygen uptake · Power output

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Introduction

The oxygen uptake–power output relationship is one of the most often applied physiological characteristics describing exercise tolerance in humans. Lower oxygen uptake ($\dot{V}O_2$) at a given power output is considered as a sign of higher locomotory efficiency. It has generally been believed that $\dot{V}O_2$ increases linearly with power output (Astrand and Rodahl 1986; Wilmore and Costill 1994; Brooks et al. 1996). On the other hand, it has recently been shown that when performing an incremental exercise test, exceeding the power output corresponding to the lactate threshold (LT) is accompanied by a significant rise in $\dot{V}O_2$ above that expected from the linear relationship $\dot{V}O_2$–power output (Zoladz et al. 1994, 1995, 1998).

It is of interest that both the deviation from linearity in $\dot{V}O_2$ occurring during an incremental exercise test as discussed above, and the slow component of $\dot{V}O_2$ kinetics (expressed by the differences in $\dot{V}O_2$ between 6th and 3rd min of cycling at a constant power output; Whipp and Wasserman 1972) become visible just above LT. This may suggest a similarity in the physiological mechanism responsible for this phenomenon.

In recent decades, starting with the work by Wasserman and McIlory (1964) the incremental exercise tests have been most frequently used to determine the *anaerobic threshold* and maximal oxygen uptake ($\dot{V}O_{2\,max}$), but surprisingly very little attention has been paid to a careful study of the $\dot{V}O_2$–power output relationship.

As illustrated by earlier studies (Zoladz et al. 1994, 1995, 1998), an increase in power output above LT requires a substantial increase in $\dot{V}O_2$ significantly exceeding the value expected from the $\dot{V}O_2$–power output relationship below LT. Such a reduction in locomotory efficiency requires an increase in minute ventilation and greater oxygen delivery, which seems to apply particularly to patients whose exercise tolerance is mainly limited by cardio-respiratory disorders (see Poole et al. 1994b).

We postulate that the power output above which there is an increase in $\dot{V}O_2$ above that expected from the linear relationship between $\dot{V}O_2$ and power output, determined during the incremental exercise test, may be used as an additional criterion characterising human locomotory efficiency and exercise tolerance. To the best of our knowledge, no attempt has been made so far to apply quantitative methods to the problem of detecting the exercise intensity above which a further increase of power output causes a non-proportional increase in $\dot{V}O_2$.

Our experience shows that standard techniques for detecting changes in regression relationships, for example those based on recursive residuals (e.g. Brown et al. 1975), are not sensitive enough when applied to our problem. In this study, therefore, we developed a new method, also based on recursive residuals, which, in addition, exploits a special structure of the problem and is able to detect change points efficiently.

Methods

Subjects

A group of 26 healthy non-smoking men [mean age 22.1 (SD 1.4) years, body mass 73.6 (SD 7.4) kg, height 179.4 (SD 7.5) cm, body fat 10.5 (SD 3.8) percentage body mass, $\dot{V}O_{2\,max}$ 3.726 (SD 0.363) $l \cdot min^{-1}$], experienced in laboratory tests, were the subjects in this study. During an interview a medical history and physical examination were completed and basic blood tests for haemoglobin concentration ([Hb]), haematocrit value (Hct), erythrocyte count (E), leucocyte count (L), sodium (Na^+), potassium (K^+) and creatinine (Cr) were carried out (Table 1).

Exercise protocol

After 5-min rest sitting on the cycle ergometer (Ergoline 800s, The Netherlands) the subjects performed an incremental exercise test at a pedalling rate of 70 $rev \cdot min^{-1}$. The test started at a power output of 30 W, followed by increases of 30 W every 3 min. The subjects were encouraged to continue the test until they were exhausted which was taken to be the point at which they could no longer maintain the required pedalling rates. All tests were performed on the same ergometer.

Gas exchange variables

Gas exchange variables were measured continuously breath-by-breath using an Oxycon apparatus (Champion Jaeger, Germany), starting from the 5th min prior to exercise until the test was completed. Before and after each test, the gas analysers were calibrated with certified calibration gases as has been described previously by Zoladz et al. 1995.

Blood sampling and analysis

At 5 min prior to exercise, at the end of each stage of exercise (the last 15 s before increasing power output) and at the moment of ending the exercise protocol blood samples (1 ml each) were taken from an antecubital vein using catheters [Abbot Int-Catheter, Ireland (18G/1.2 × 45 mm)] inserted into the antecubital vein about 30 min prior to the onset of exercise. The catheter was connected with the extension set using a *T′* Adapter (SL Abbot, Ireland; a tube 10 cm in length). Immediately before the samples were taken for an analysis, samples 1-ml of blood were taken to eliminate the blood from the catheter and the T-set. A part of each sample (90 μl) of blood was used for the detection of blood gases (blood partial pressure of oxygen and carbondioxide, PO_2 and PCO_2) and blood hydrogen ion concentration $[H^+]_b$.

Portions of 0.5-ml blood from each sample were placed in 1.8-ml Eppendorf tubes containing 1-mg ammonium oxalate and 5-mg sodium fluoride and mixed for about 20 s. Subsequently, to separate plasma for performing measurements of lactate concentration, the blood samples were centrifuged. Samples of blood plasma (200 μl) were stored for further analysis at a temperature of minus 25°C. The PO_2 and PCO_2, as well as $[H^+]_b$ were determined using a

Table 1 Values for haematocrit (*Hct*), haemoglobin concentration (*Hb*), erythrocyte count (*E*), leukocyte count (*L*), sodium (Na^+), potassium (K^+) and creatinine concentrations [(*Cr*)] determined in blood from an antecubital vein

	Hct (vol.%)	Hb (g%)	E ($10^6 \cdot mm^{-3}$)	L ($1000 \cdot mm^{-3}$)	Na^+ ($mmol \cdot l^{-1}$)	K^+ ($mmol \cdot l^{-1}$)	[Cr] ($\mu mol \cdot l^{-1}$)
Minimal	40.4	13.9	4.47	4.5	142.5	3.61	58.3
Maximal	50.2	16.9	5.59	9.0	149.5	5.51	104.9
Mean	45.5	15.5	5.23	6.1	145.3	4.44	83.7
SD	2.7	0.9	0.6	1.2	1.8	0.50	12.1

Ciba-Corning 248 analyser (England). The blood bicarbonate concentration $[HCO_3^-]_b$ and the base excess were calculated by this unit. Plasma lactate concentration ($[La^-]_{pl}$) was measured using an automatic analyser (Ektachem XR 700, Kodak, USA). Serum Na^+ and K^+ concentrations were determined using a flame photometer (Ciba Corning Model 480, England). Blood Cr concentration was determined by a kinetics method based on the reaction with picric acid using an automatic analyser (Express 550 CBI, England). The [Hb], Hct value, E and L were determined using an automatic haematological analyser (Baker 9000, USA). The percentage of body fat was assessed according to Hassager et al. 1986.

Lactate threshold

The LT in this study was defined as the highest power output above which $[La^-]_{pl}$ showed a sustained increase of more than 0.5 mmol $\cdot l^{-1} \cdot step^{-1}$ (Zoladz et al. 1995).

Detection of the change point in $\dot{V}O_2$

A visual inspection of the data and a previous study (see Zoladz et al. 1995) suggested the following model for the observed 3rd-min data of $\dot{V}O_2$;

$$X_i = at_i + b + \varepsilon_i \quad \text{for } i = 1, 2, \ldots, T \tag{1}$$

$$E(X_i) > at_i + b \quad \text{for } i = T+1, \ldots, n$$

where $X_1, \ldots, X_n$ are independent, $\varepsilon_i \sim N(0, \sigma^2)$, and T can possibly be equal to n, in which case all data points would be modelled by the same linear regression equation. If T is less than n then the linear regression model holds for $X_1, \ldots, X_T$ and $X_{T+1}, \ldots, X_n$ tend to lie above that linear regression line. We do not assume any specific functional form for the non-linear part of the model. The t_i's can represent minutes counted from the beginning of the test or for any linear function of time, e.g. the power output.

The above model is a form of what has been described in the literature as a change point (CP) problem (Brown et al. 1975; James et al. 1987; Antoch and Huškova 1989, and the references quoted therein). Standard procedures for testing constancy of regression relationships, described in the references given above, fail to detect CP in our model, however. This is partly because of small sample sizes, about 10–12 at best, and also because CP often occurs close to the beginning of data. What makes our CP detection problem solvable at all are very small variances σ^2 in the error terms ε_i in the linear part of the model.

Application of the model given by Eq. 1 consisted of two steps: testing for the existence of a CP and estimating its location T. We proposed to base both procedures on suitably normalised recursive residuals.

We defined the k-linear-r-ahead recursive residual V_k^r as:

$$V_k^r = \frac{kS(t_1, \ldots, t_k)}{(k+1)S(t_1, \ldots, t_k, t_{k+r})} \cdot \frac{X_{k+r} - \hat{X}_{k+r}}{\hat{S}_k}$$

where $r = 1, 2, \ldots, n-3$, $k = 3, 4, \ldots, n-r$, $\hat{X}_{k+r} = \hat{a}t_{k+r} + \hat{b}$ is the forecast for X_{k+r} based on the linear regression model $X = \hat{a}t + \hat{b}$ fitted by least squares to the first k data points (t_1, X_1), …, (t_k,X_k).

Further,

$$\hat{S}_k = \left[(k-2)^{-1} \sum_{i=1}^{k} (X_i - \hat{X}_i)^2 \right]^{1/2}$$

is the usual estimator of σ obtained from the linear fit to the first k data points, and

$$S(u_1, \ldots, u_\ell) = \left[\ell^{-1} \sum_{i=1}^{\ell} u_i^2 - \left(\ell^{-1} \sum_{i=1}^{\ell} u_i \right)^2 \right]^{1/2}$$

is a measure of dispersion of the numbers u_1. The lower limit 3 for k follows from the fact that we need at least three points to fit straight line reasonably. For a given, fixed r, we also have an upper limit $n-r$ for k, because $k-r$ must not be greater than n for V_k^r to make sense. Note an important difference between the standard procedures based on recursive residuals, as discussed for example by Brown et al. (1975), and our approach. Whereas the standard approach is to scale the residuals by the square root of the variance estimate obtained from the straight line fit to all data points, we have used the variance estimate obtained from the fit to the first k points only, thus taking advantage of small residuals, variances in the linear part of the model. This is crucial for the test's power in our specific model.

It can be shown that if $T = n$, then V_k^r is t-Student distributed with $k-2$ degrees of freedom, i.e.

$$V_k^r \sim t_{k-2} \ .$$

Let us fix r. We can then compute the recursive residuals

$$V_3^r, V_4^r, \ldots, V_{n-r}^r \ . \tag{2}$$

If there is a CP when T is less than n, one can expect at least one of the recursive residuals given by 2 to take on a value larger than expected for a linear model with no CP, i.e. for $T = n$. Thus, to test H_0: $T = n$ against H_1: $T < n$, we proposed to use the test statistic

$$W_r = \max\{V_3^r, V_4^r, \ldots, V_{n-r}^r\}$$

and to reject H_0 when W_r was greater than w_α, where w_α was selected such that, under H_0, $P(W_r > w_\alpha) \leq \alpha$ for a given significance level α. Each individual $V_k{}^r$ was t_{k-2}-distributed. They were not, however, independent. Using the Bonferroni inequality test, we produced a valid level-α test by taking w_α to be the right-hand-sided critical-value of the t_{k-2} distribution at the significance level $\alpha/(n-r-2)$. Equivalently, in terms of P-values, we computed the overall P value for our test statistic W_r as

$$(n-r-2). \min_{3 \leq k \leq n-r} P(t_{k-2} > V_k^r) \tag{3}$$

i.e. $n-r-2$ times the minimum of the individual P values corresponding to individual V_k^r.

A crucial point in the test construction was the choice of r. We had to reconcile some conflicting requirements. On the one hand, r should not be too small, because small r would have led to large $n-r-2$ and to a possible loss of the power of the test (c.f. formula 3) and because a larger r should have increased the power of the test (a departure from linearity should have been more visible when a longer-range forecast was compared with the observed X). On the other hand, however, longer range forecasts would have had larger variances, which would have made the differences $X_{k+r} - \hat{X}_{k+r}$ less significant, because $S(t_1, \ldots, t_k, t_{k+r})$ would have become larger in that case. It was also intuitively clear that an optimal r would have depended on the shape of the non-linear part of the model Eq. (1). In effect, the choice of r was somewhat arbitrary.

In the case of our data, where n ranged from 8 to 11 and, in most cases, the non-linear part of the model was exponentially shaped, setting $r = 2$ or $r = 3$ gave the best results, in the sense of most often detecting departures from linearity.

Having detected non-linearity, we were interested in estimating the location T of CP. We were thus interested in locating the first point significantly above the regression line fitted to all the previous points, and we based the procedure on the k-linear-l-ahead recursive residuals

$$V_3^1, V_4^1, \ldots, V_{n-1}^1 \tag{4}$$

It was natural to set $T = k$, where V_k^1 was the first recursive residual satisfying $P(t_{k-2} > V_k^1) < \alpha$, where α was a fixed threshold value. Since the residuals given by Eq. were strongly correlated, it was difficult to interpret the threshold value α precisely and quantitatively. It was clear, however, that the choice of α balanced the relative loss we assigned to two possible errors: selecting T too small and selecting T too large. If one considers the first kind of error more serious than the second, then α should be selected smaller than in the opposite case. In our study, we used $\alpha = 0.10$.

One important remark is in order here. Having detected CP at $T = k_o$ with a fixed α, we might have been surprised that the P-values computed for $k > k_o$ were often much higher than the assumed α. This could not be interpreted, however, as a sign that those points still belonged to the linear part of the model. This was due to the fact that, for the points above CP, the P-values could not be computed from the t_{k-2}-distribution, as the model assumptions were not fulfilled in that region. V_k^r were t_{k-2}-distributed *only* if all k points $X_1, \ldots, X_k$ belonged to the linear part of the model. This was why CP was set at the power output at which the P-value dropped below α for the first time.

Results

The power output at CP-$\dot{V}O_2$ and at LT

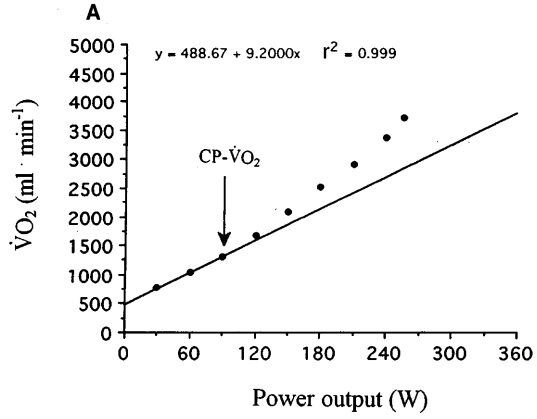
In the case of 25 out of 26 subjects tested, it was possible to determine the CP in $\dot{V}O_2$ (CP-$\dot{V}O_2$) during the incremental exercise test, in the way described above. The mean values of power output at the CP-$\dot{V}O_2$ and at LT amounted to 137 (SD 31) and 126 (SD 28) W, respectively (Table 2). Typical examples of detection of CP-$\dot{V}O_2$ in subjects with low, average and high levels of power output at the CP-$\dot{V}O_2$ are presented by Fig. 1, panels A, B and C, respectively. Figure 2 gives the only case (subject 3) in which CP-$\dot{V}O_2$ was not detectable by our method. Individual values of the power output and $\dot{V}O_2$ determined at rest, at CP-$\dot{V}O_2$, at LT and at the maximal power output (P_{max}) reached during the incremental exercise test, are presented in Table 2. Tables 3 and 4 give the individual values of $[La^-]_{pl}$, $[H^+]_b$, and $[HCO_3^-]_b$, determined at rest, at CP-$\dot{V}O_2$, at LT and at P_{max} reached during the incremental exercise test.

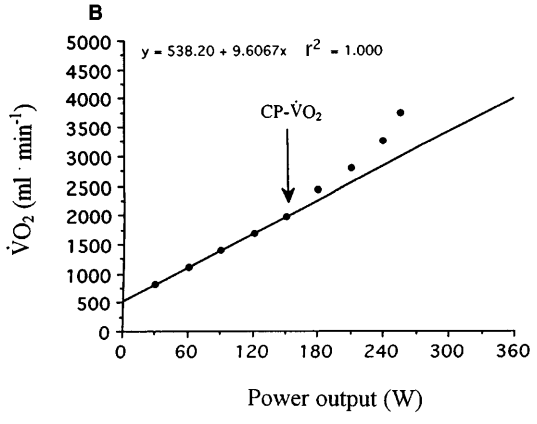
Comparison of the power output at CP-$\dot{V}O_2$ and at LT

The power output and the differences of power outputs at the detected CP-$\dot{V}O_2$ and at LT are by design discrete variables: multiples of 30. In the 25 cases in which the CP-$\dot{V}O_2$ was detectable, those differences took the values of - 60 (2 cases) - 30 (3 cases), 0 (9 cases), 30 (7 cases) and 60 (4 cases). To test the hypothesis that, on average, CP-$\dot{V}O_2$ and LT coincide, we applied the standard χ^2 test. With the aim of obtaining acceptable bin counts, we formed three classes: negative (5 cases), 0 (9 cases) and positive (11) cases. Standard χ^2 statistic for testing the null hypothesis – probabilities of positive and negative differences are equal – took the value of 2.25, which meant that the null hypothesis was not rejected up to the test significance level 0.13 (χ^2 statistic with one degree of freedom). Thus, on average, there was no significant difference between power outputs at the detected CP-$\dot{V}O_2$ and at LT. Notice, however, that out sample size was rather small ($n = 25$), and apparently the power outputs at CP tended to take on values higher than at LT. It is plausible that the differences at the power output would have become significant if the sample size had been larger.

Table 2 Individual values of power output and oxygen uptake ($\dot{V}O_2$) determined at rest, at the change point in oxygen uptake (CP-$\dot{V}O_2$), at the lactate threshold (LT) and at the maximal power output (P_{max}) reached during the incremental exercise test ($n = 26$ subjects)

Subject	Power output (W)			$\dot{V}O_2$ (ml · l^{-1})			
	CP-$\dot{V}O_2$	LT	max	Rest	CP-$\dot{V}O_2$	LT	P_{max}
1	210	180	330	318	2575	2258	4542
2	150	120	290	292	2070	1739	3818
3	–	90	270	301	–	1370	3387
4	150	90	280	344	2050	1418	4042
5	120	120	225	340	1682	1682	3219
6	120	120	315	340	1577	1577	4450
7	150	90	256	388	1976	1413	3751
8	90	90	275	327	1384	1384	3689
9	180	120	320	332	2299	1658	4360
10	180	120	320	371	2247	1689	4146
11	120	120	265	325	1588	1588	3517
12	120	120	290	435	1818	1818	3790
13	120	90	270	462	1648	1298	3778
14	180	150	284	456	2197	1970	3679
15	150	150	263	428	1849	1849	3467
16	120	120	270	402	1705	1705	3481
17	120	120	260	305	1636	1636	3340
18	90	120	255	308	1321	1675	3709
19	120	90	244	363	1594	1302	3284
20	120	150	267	353	1592	1929	3630
21	150	120	250	354	2243	1882	3558
22	90	150	270	339	1203	1970	3189
23	120	150	270	540	1610	1923	3406
24	180	180	310	489	2313	2313	3799
25	150	120	300	486	2048	1720	3842
26	120	180	310	264	1464	2112	4001
Mean	136.8	125.8	279.2	371.6	1828	1726	3726
SD	31.3	28.2	26.3	70.3	356	274	363





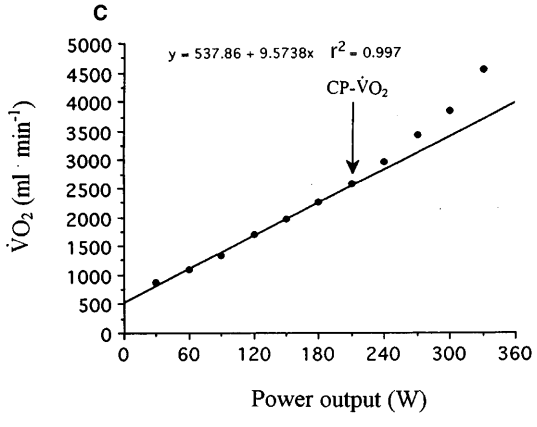


Fig. 1 Typical examples of detection of the change point in oxygen uptake ($CP\text{-}\dot{V}O_2$) in subjects with low (panel A), average (panel B) and high (panel C) levels of power output at $CP\text{-}\dot{V}O_2$. The linear regression is based on the data below $CP\text{-}\dot{V}O_2$

Comparison of the $\dot{V}O_2$, $[La^-]_{pl}$, $[H^+]_b$ and $[HCO_3^-]_b$ at rest, at CP-$\dot{V}O_2$ and at the power output 30 W above CP-$\dot{V}O_2$

The following differences were tested at the significance level 0.05 by means of the standard Student's t test for having expected values equal to 0:

A. The magnitude of $\dot{V}O_2$ millilitres per minute reached at CP-$\dot{V}O_2$ was not significantly different ($P = 0.21$) from the value reached at LT (Table 2). The

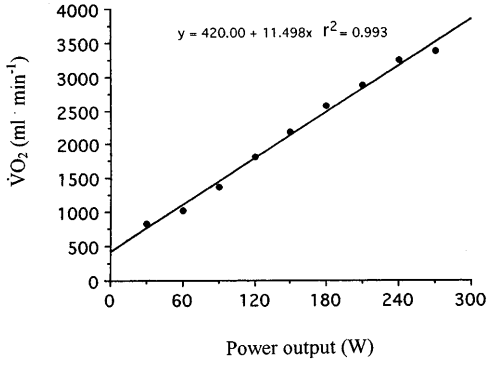


Fig. 2 Oxygen uptake ($\dot{V}O_2$) during the incremental exercise test in subject no. 3, in whom the change point in $\dot{V}O_2$ was not detectable

$\dot{V}O_2$ reached at CP-$\dot{V}O_2$ amounted to 1.828 (SD 0.356) $l \cdot min^{-1}$, corresponding to 48.9 (SD 7.9)% $\dot{V}O_{2\,max}$.

The $[La^-]_{pl}$ at the power output corresponding to CP-$\dot{V}O_2$ amounting to 2.57 (SD 0.69) $mmol \cdot l^{-1}$ was significantly ($P < 1 \cdot 10^{-4}$) elevated above the resting level, amounting to (1.85 SD 0.46) $mmol \cdot l^{-1}$.

The $[H^+]_b$ at CP-$\dot{V}O_2$ was higher when compared to the pre-exercise level [45.10 (SD 3.03) vs 44.13 (SD 2.79) $nmol \cdot l^{-1}$, respectively], however, this increase did not reach the level of significance ($p = 0.07$).

The $[HCO_3^-]_b$ at CP-$\dot{V}O_2$ amounting to 24.88 (SD 1.44) $mmol \cdot l^{-1}$ was significantly ($p = 0.02$) lower than before the test [25.38 (SD 1.12) $mmol \cdot l^{-1}$; (Table 3).

B. The increase of power output by 30 W above the change point in $\dot{V}O_2$ (CP-$\dot{V}O_2$ + 30) was accompanied by more pronounced acidosis when compared to the CP-$\dot{V}O_2$ level. The $[H^+]_b$ at this power output was significantly elevated ($P = 0.03$) compared to resting conditions. Moreover, the increase of power output by only 30 W above CP-$\dot{V}O_2$ was accompanied by a further significant increase ($P < 1 \cdot 10^{-6}$) of $[La^-]_{pl}$, and a significant ($P < 1 \cdot 10^{-4}$) in $[HCO_3^-]$ in relation to the level reached at CP-$\dot{V}O_2$.

The individual values of $\dot{V}O_2$, $[La^-]_{pl}$, $[H^+]_b$ and $[HCO_3^-]_b$ at rest, at CP-$\dot{V}O_2$ and at LT as well as at CP-$\dot{V}O_2$ + 30, are given in Table 3.

All variables involved tested positively for normality using the Shapiro-Wilk test at the significance level of 0.05.

Discussion

The present study confirmed the suitability of using recursive residuals to determine the power output above which $\dot{V}O_2$ during the incremental exercise test increases more than proportionately with the increase of power output. This characteristic exercise intensity (power output) we have called CP-$\dot{V}O_2$ (see Fig. 1). As illustrated by the data given in Table 2, the power output at which CP-$\dot{V}O_2$ occurred was very close – and not significantly different from the power output at which LT

Table 3 Individual values of plasma lactate ($[La^-]_p$), blood hydrogen ion ($[H^+]_b$), and blood bicarbonate ($[HCO_3^-]_b$), concentrations determined at rest, at the change point in oxygen uptake (*CP*-$\dot{V}O_2$), at the lactate threshold (*LT*) and at a power output 30 W above the change during point (*CP*-$\dot{V}O_2$ + 30) reached during the incremental exercise test (n = 26 subjects)

Subject	$[La^-]_{pl}$ (nmol · l^{-1})				$[H^+]_b$ (nmol · l^{-1})				$[HCO_3^-]_b$ (mmol · l^{-1})			
	Rest	CP-$\dot{V}O_2$	LT	CP-$\dot{V}O_2$ + 30	Rest	CP-$\dot{V}O_2$	LT	CP-$\dot{V}O_2$ + 30	Rest	CP-$\dot{V}O_2$	LT	CP-$\dot{V}O_2$ + 30
1	2.3	2.9	2.4	3.6	41.3	41.5	42.4	42.5	25.2	25.3	25.1	24.7
2	1.4	3.1	2.3	3.9	41.8	43.0	44.9	44.3	25.4	23.3	24.6	22.9
3	1.3	–	1.7	–	43.4	–	44.5	–	25.1	–	25.0	–
4	1.5	2.7	1.7	4.0	43.9	45.5	43.8	45.5	24.3	23.9	24.6	22.6
5	1.7	1.8	1.8	3.1	45.0	47.5	47.5	46.7	22.5	22.3	22.3	22.4
6	1.6	1.8	1.8	2.4	42.9	46.0	46.0	44.5	25.5	25.0	25.0	24.8
7	1.1	3.2	1.6	3.2	43.9	46.8	45.4	46.8	24.8	23.6	24.9	23.6
8	2.0	2.3	2.3	2.8	44.7	46.3	46.3	47.6	24.2	24.4	24.4	23.8
9	2.8	4.6	3.0	5.9	45.5	48.8	48.1	46.2	27.3	26.4	27.2	25.9
10	1.7	2.9	1.8	3.6	40.5	43.7	44.5	44.0	25.9	25.8	26.1	24.6
11	1.9	2.1	2.1	2.8	45.1	43.3	43.3	43.8	25.3	26.3	26.3	25.4
12	1.9	2.1	2.1	2.6	41.8	40.1	40.1	40.6	25.9	26.0	26.0	25.7
13	1.7	2.9	2.3	4.0	48.7	46.2	45.1	46.2	25.1	25.6	26.1	25.5
14	2.3	3.3	2.2	4.2	48.0	43.7	45.6	45.8	25.4	24.9	25.7	23.8
15	1.8	3.4	3.4	4.5	42.5	44.3	44.3	44.7	26.9	25.4	25.4	24.4
16	1.7	1.9	1.9	2.5	43.2	42.0	42.0	41.3	26.8	26.5	26.5	26.2
17	1.4	2.1	2.1	2.6	40.5	43.8	43.8	44.9	26.5	26.9	26.9	25.5
18	2.0	1.5	1.5	1.5	44.9	41.2	43.4	43.4	26.9	26.3	26.7	26.7
19	2.3	2.6	2.1	3.4	42.7	46.5	45.3	46.2	25.0	25.7	25.7	25.3
20	2.3	2.7	3.1	3.1	40.6	41.5	40.5	40.5	25.3	25.7	25.0	25.0
21	2.1	2.8	2.3	4.0	45.4	46.9	45.7	49.1	25.9	25.1	25.4	24.5
22	1.5	1.9	2.8	2.4	44.8	46.0	44.6	45.0	25.8	26.2	25.7	25.6
23	1.5	1.8	2.2	2.2	43.8	44.4	43.7	43.7	26.0	22.5	22.7	22.7
24	1.6	2.1	2.1	3.1	50.0	47.3	47.3	48.4	25.0	23.9	23.9	23.6
25	1.6	3.1	2.5	4.2	50.3	54.4	54.5	51.5	23.6	22.0	22.2	21.3
26	3.1	2.6	3.1	2.8	41.5	47.9	49.0	48.8	23.9	23.0	22.7	23.3
Mean	1.85	2.57	2.24	3.30	44.1	45.1	45.1	45.3	25.4	24.9	25.1	24.4
SD	0.46	0.69	0.50	0.92	2.8	3.0	2.9	2.65	1.1	1.4	1.4	1.4

Table 4 Individual values of $[La^-]_{pl}$, $[H^+]_b$, and $[HCO_3^-]_b$ determined at the maximal power output reached during the incremental test ($n = 26$). For definitions see Table 3

Subject	$[La^-]_{pl}$ (mmol · l^{-1})	$[H^+]_b$ (nmol · l^{-1})	$[HCO_3^-]_b$ (mmol · l^{-1})
1	11.7	49.2	19.2
2	11.6	54.4	18.1
3	10.1	54.7	17.3
4	10.7	58.0	17.2
5	10.8	52.2	18.0
6	12.3	55.8	17.0
7	13.5	59.4	16.3
8	15.4	57.4	15.5
9	15.5	56.4	17.7
10	14.1	57.5	18.0
11	15.0	55.3	16.6
12	9.1	51.4	21.0
13	9.3	54.6	18.2
14	9.6	54.3	20.1
15	9.8	51.3	20.2
16	5.6	45.3	23.7
17	9.2	53.3	17.6
18	8.8	49.1	20.8
19	10.7	55.0	19.8
20	15.3	49.4	18.0
21	7.2	48.8	21.3
22	7.6	48.8	21.0
23	6.8	46.3	18.8
24	8.0	58.3	18.0
25	12.0	56.1	14.6
26	9.8	54.8	17.7
Mean	10.75	53.35	18.53
SD	2.79	3.84	2.02

was found. The mean value of $\dot{V}O_2$ at CP-$\dot{V}O_2$ amounted to 49 (SD 8)% $\dot{V}O_{2max}$. It is of interest that the $[La^-]_b$ determined at CP-$\dot{V}O_2$ was already significantly elevated when compared to the pre-exercise level, but $[H^+]_b$ determined at CP-$\dot{V}O_2$ was not significantly different from the pre-exercise value. However, an increase of only 30 W in power output above CP-$\dot{V}O_2$ was already accompanied by a significant increase in $[H^+]_b$ in relation to the resting level (see Table 3). These results suggested that the onset of the more than proportionate increase in $\dot{V}O_2$ observed during the incremental exercise test was accompanied by the onset of acidosis (see Table 3, Fig. 3). On the other hand, as illustrated by the individual data, in some subjects CP-$\dot{V}O_2$ may have occurred at lower or higher power outputs than LT.

The physiological mechanism responsible for CP-$\dot{V}O_2$ remains unclear. We believe that the physiological background of CP-$\dot{V}O_2$ has the same origin as the slow component of $\dot{V}O_2$ kinetics (see Whipp and Wasserman 1972; Whipp 1994, 1996). A number of factors have been suggested as a possible cause of the slow component of $\dot{V}O_2$ kinetics, which include an increase in concentration of circulating catecholamines, an increase in muscle and body temperature, an increase in the cost of respiratory muscle work due to the intensification of hyperpnoea, an increase in the involvement of additional muscle groups, and recruitment of the less efficient type II muscle fibres (for review see Whipp 1994).

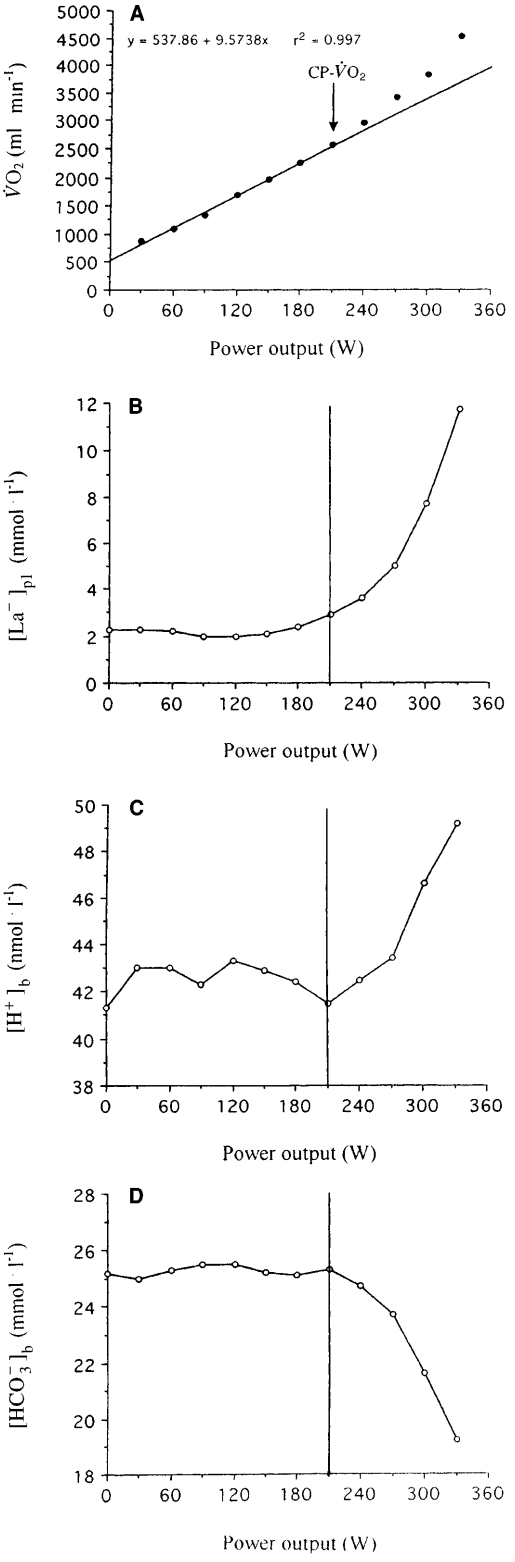


Fig. 3 Typical changes in oxygen uptake ($\dot{V}O_2$; *panel A*), plasma lactate concentration ($[La^-]_{pl}$; *panel B*), blood hydrogen ion concentration ($[H^+]_b$; *panel C*) and blood bicarbonate concentration ($[HCO_3^-]_b$; *panel D*) below and above the change point in oxygen uptake (*CP-*$\dot{V}O_2$). The *vertical line* illustrates the power output corresponding to *CP-*$\dot{V}O_2$

One may argue as to how far the pulmonary $\dot{V}O_2$ reflects $\dot{V}O_2$ at the level of working locomotory muscle. According to Poole et al. (1991), who have simultaneously measured the pulmonary $\dot{V}O_2$ and the leg muscle $\dot{V}O_2$ during constant high power exercise, 86% of the increase in the pulmonary $\dot{V}O_2$ observed between 3rd and 21st min of cycling was attributable to the exercising muscles in the legs. This suggests that indeed most of the additional increase in $\dot{V}O_2$ represents the fall in efficiency of the working locomotory muscle cells. On the other hand, studies by Saltin et al. (personal communication) have not supported the data by Poole et al. (1991) showing the lack of appearance of the slow component of the $\dot{V}O_2$ kinetics at the level of working leg muscle.

Since the slow component of $\dot{V}O_2$ kinetics is always accompanied by an accelerated rate of increase in $[La^-]_{pl}$, the study of the relationship between the degree of acidosis and the magnitude of the slow component of $\dot{V}O_2$ kinetics has attracted the attention of many researchers in the last decade. It has been shown that the magnitude of the slow component of $\dot{V}O_2$ kinetics positively correlates with the rate of increase of $[La^-]_{pl}$ (Casaburi et al. 1987; Roston et al. 1987; Koike et al. 1990). This suggests that acidosis may play a regulatory role in the slow component of the $\dot{V}O_2$ kinetics. On the other hand, neither infusion of lactate into working dog gastrocnemius muscle (Poole et al. 1994a), nor adrenaline-infusion-induced increase of blood lactate concentration (Gasser et al. 1994), nor bicarbonate-ingestion-induced increase of $[La^-]_{pl}$ (Zoladz et al. 1997a) influenced the magnitude of the slow component of $\dot{V}O_2$ kinetics. On the other hand, Zoladz et al. (1997b) have recently shown that pre-exercise acidification induced by ingestion of NH_4Cl did increase the magnitude of the slow component of $\dot{V}O_2$ kinetics in each individual subject.

One may also consider an intensification of the exercise hyperpnoea and an increase in the cost of breathing as a possible cause of the non-linear increase in $\dot{V}O_2$ during an incremental exercise test. In our previous study (Zoladz et al. 1998) the increase of power output from LT to P_{max} was accompanied by an increase in the minute ventilation from 49.6 (SD 7.3) to 116.8 (SD 8.9) $l \cdot min^{-1}$. On the basis of the data reported by Aaron et al. (1992), one may calculate that such an elevation of minute ventilation would increase the respiratory muscle $\dot{V}O_2$ by about 200 ml. This estimation would suggest that in our study about one-third of the additional $\dot{V}O_2$ [569 (SD 269) ml] observed at P_{max} could be due to intensification of the exercise hyperpnoea. However, taking into account the curvilinear increase of minute ventilation above LT, the true cost of hyperventilation at $\dot{V}O_{2\,max}$ is presumably smaller than one-third of the additional $\dot{V}O_2$.

It has been suggested that the most likely explanation of a major part of the slow component of $\dot{V}O_2$ kinetics is recruitment of type II muscle fibres (Whipp 1994; Barstow et al. 1996). Indeed it has been shown that individuals with a high percentage of type II muscle fibres utilise substantially more oxygen at a given power output than those with a higher proportion of type I (see Coyle et al. 1992). Moreover, experiments in which selective blocking of type I muscle fibres was obtained by administration of a low dosage of tubocurarine have shown a significantly higher oxygen cost of cycling at a given power output at a pedalling rate of 60 rev $\cdot$ min^{-1}, when compared to control conditions (Galbo et al. 1987). This supports the suggestions that type II muscle fibres are indeed significantly less efficient than type I.

Little is known about the strategy of recruitment of different types of muscle fibres; the available data suggest that increasing power output at a given velocity of muscle contraction causes a progressive recruitment of type IIa and type IIb muscle fibres (see Sargeant and Beelen 1993; Sargeant 1996). The reason why it requires more oxygen for type II muscle fibres to generate a given power output than type I remains unclear. It has been postulated that the efficiency (the high energy phosphate produced per oxygen molecule consumed) of mitochondria in type II muscle fibres appears to be lower than that of mitochondria from type I muscle fibres (see Willis and Jackman 1994).

In conclusion, the CP-$\dot{V}O_2$ representing the highest exercise intensity above which a further increase of power output requires a more than proportionate increase in $\dot{V}O_2$, can be detected by the method described above. This characteristic exercise intensity indicates the onset of a fall in human locomotory efficiency. This is why we suggest that CP-$\dot{V}O_2$ may be used as an additional physiological index when studying human exercise tolerance.

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