Physiol. Res. 47: 427–438, 1998

VO_2/Power Output Relationship and the Slow Component of Oxygen Uptake Kinetics During Cycling at Different Pedalling Rates: Relationship to Venous Lactate Accumulation and Blood Acid-Base Balance

J.A. ZOLADZ[1], K. DUDA[2,3], J. MAJERCZAK[1]

[1]*Department of Physiology and Biochemistry,* [2]*Institute of Rehabilitation, AWF-Krakow and* [3]*Cancer Institute, Poland*

Received April 7, 1998
Accepted July 14, 1998

Summary

In this experiment we studied the effect of different pedalling rates during cycling at a constant power output (PO) 132±31 W (mean±S.D.), corresponding to 50 % V_{O_2} max, on the oxygen uptake and the magnitude of the slow component of V_{O_2} kinetics in humans. The PO corresponded to 50 % of V_{O_2} max, established during incremental cycling at a pedalling rate of 70 rev.min^{-1}. Six healthy men aged 22.2±2.0 years with V_{O_2} max 3.89±0.92 l.min^{-1}, performed on separate days constant PO cycling exercise lasting 6 min at pedalling rates 40, 60, 80, 100 and 120 rev.min^{-1}, in random order. Antecubital blood samples for plasma lactate $[La]_{pl}$ and blood acid-base balance variables were taken at 1 min intervals. Oxygen uptake was determined breath-by-breath. The total net oxygen consumed throughout the 6 min cycling period at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1} amounted to 7.727±1.197, 7.705±1.548, 8.679±1.262, 9.945±1.435 and 13.720±1.862 l, respectively for each pedalling rate. The V_{O_2} during the 6 min of cycling only rose slowly by increasing the pedalling rate in the range of 40–100 rev.min^{-1}. This increase, was 0.142 l per 20 rev.min^{-1} on the average. Plasma lactate concentration during the sixth minute of cycling changed little within this range of pedalling rates: the values were 1.83±0.70, 1.80±0.48, 2.33±0.88 and 2.52±0.33 mmol.l^{-1}. The values of $[La]_{pl}$ reached in the 6th minute of cycling were not significantly different from the pre-exercise levels. Blood pH was also not affected by the increase of pedalling rate in the range of 40–100 rev.min^{-1}. However, an increase of pedalling rate from 100 to 120 rev.min^{-1} caused a sudden increase in the V_{O_2} amounting to 0.747 l per 20 rev.min^{-1}, accompanied by a significant increase in $[La]_{pl}$ from 1.21±0.26 mmol.l^{-1} in pre-exercise conditions to 5.92±2.46 mmol.l^{-1} reached in the 6th minute of cycling ($P<0.01$). This was also accompanied by a significant drop of blood pH, from 7.355±0.039 in the pre-exercise period to 7.296±0.060 in the 6th minute of cycling ($P<0.01$). The mechanical efficiency calculated on the basis of the net V_{O_2} reached between the 4th and the 6th minute of cycling amounted to 26.6±2.7, 26.4±2.0, 23.4±3.4, 20.3±2.6 and 14.7±2.2 %, respectively for pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}. No significant increase in the V_{O_2} from the 3rd to the 6th min (representing the magnitude of the slow component of V_{O_2} kinetics) was observed at any of the pedalling rates (−0.022±0.056, −0.009±0.029, 0.012±0.073, 0.030±0.081 and 0.122±0.176 l.min^{-1} for pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}, respectively). Thus a significant increase in $[La]_{pl}$ and a decrease in blood pH do not play a major role in the mechanism(s) responsible for the slow component of V_{O_2} kinetics in humans.

Key words
Acid-base balance – Exercise – Mechanical efficiency – Oxygen uptake kinetics – Power output

PHYSIOLOGICAL RESEARCH *ISSN 0862-8408*

Fax +420 2 293 894
E-mail physres@sun1.biomed.cas.cz
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Introduction

The relationship between oxygen uptake and pedalling rate at a given power output has fascinated many researchers since the beginning of this century (Benedict and Cathcart 1913, Dickinson 1929, Hagberg *et al.* 1981, Sargeant and Beelen 1993). The early studies suggested that minimal oxygen uptake for a given power output can be attained when cycling at a relatively low pedalling rate amounted to about 60 rev.min^{-1} (Benedict and Cathcart 1913, Dickinson 1929). However, a recent laboratory study supported by practical field observations have shown that the optimal pedalling rate for obtaining the highest efficiency depends upon the magnitude of the generated power (Seabury *et al.* 1977, Coast and Welch 1985), and that during prolonged high power output exercise this may be close to 100 rev.min^{-1} (Hagberg *et al.* 1981, Sargeant and Beelen 1993, Zoladz and Rademaker 1994).

In view of the recent studies, the mechanical efficiency seems to be related to the degree of recruitment of type II muscle fibres. It has been demonstrated that type I muscle fibres appear to be more efficient than type II during cycling at a given power output of 80 rev.min^{-1} (Coyle *et al.* 1992). Moreover, according to Sargeant and Beelen (1993) minimal recruitment of type II muscle fibres may delay the onset of fatigue and provide the highest performance.

It seems very likely that the continuous increase in oxygen uptake during constant high power output exercise, known as the slow component of the V_{O_2} kinetics (Whipp and Wasserman 1972), may be due to a gradual recruitment of type II muscle fibres, as recently suggested by Whipp (1994) .

In order to gain more insight into the role of type II muscle fibres recruitment in the mechanical efficiency of cycling and the slow component of V_{O_2} kinetics, we attempted in the present study to evaluate the influence of gradually increasing pedalling rate up to 120 rev.min^{-1}, which requires greater recruitment of type II muscle fibres (Sargeant and Jones 1995, Sargeant 1996) on the pulmonary oxygen uptake during constant power output exercise. We hypothesised that, if the recruitment of type II muscle fibres and accompanying acidosis indeed contribute to the decrease of mechanical efficiency, then a progressive increase of the pedalling rate during constant power output exercise, which requires greater recruitment of type II muscle fibres, would be reflected by an increase of net pulmonary V_{O_2} and the magnitude of the slow component of V_{O_2} kinetics.

Methods

Subjects

Six healthy male non-smokers aged 22.2±2.0 years, body mass 75.2±5.7 kg, percentage of body fat 13±2.4 % of body mass, height 183.7±5.6 cm, body mass index 22.6±1.3, V_{O_2}max 51.9±5.3 ml.kg^{-1}, participated in this study. Their mean values of systolic and diastolic blood pressure at rest was 122±2.5 and 73.3±5.3 mm Hg, respectively, with resting heart rate of 60±5.1 beats per min.

Exercise protocols

Subjects performed constant power output exercise tests for 6 min on separate days at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}. The power output during all tests was identical, amounting to 50 % V_{O_2} max, established individually for each subject on the basis of the V_{O_2}/power output relationship determined during an incremental test performed at the pedalling rate of 70 rev.min^{-1}. Power output during the incremental tests increased by 30 W every 3 min. The calculation of individual power output corresponding to 50 % V_{O_2} max was based on the linear relationship between V_{O_2}/power output data obtained during incremental tests, taking into consideration only data below the level of sustained increase in blood lactate concentration (Zoladz *et al.* 1995, 1998a,b). Values of V_{O_2} attained during the third minute of each stage of the incremental test were used for this calculation. Starting from the 5th minute prior to the constant power output exercise, gas exchange variables were recorded breath-by-breath. One minute before starting the constant power output exercise, and at the end of each minute of cycling, antecubital blood samples were taken to determine values of blood gases and plasma lactate. All exercise tests were performed on the same ergometer (Ergo-line 800 s, Bunnik, The Netherlands). Care was taken to ensure the same cycling position on each occasion by adjusting the height of the saddle.

Gas exchange variables

These variables were measured continuously (breath-by-breath) using Oxycon Champion (Jaeger, Germany). Before and after the end of each test, gas analyzers were calibrated with certified calibration gases. The first 20 s of this calibration were used for flushing the analyzers, the last 10 s were used to measure the concentration of a sample gas. The mouth volume sensor was calibrated with a 3 l syringe. After at least six complete strokes, the average values of the last five strokes were used to calculate the inspiratory and expiratory values. The measured values had to fall within 1 % of the reference values. The repeated calibration procedures had shown very high stability for

the breath-by-breath system during the duration of the test.

Blood sampling and analysis

Under sterile conditions an Abbott Int-Catheter Ireland (18 G/1.2x45 mm) was inserted into the antecubital vein and catheter was connected with the Extension Set using a "T" Adapter SL Abbott Ireland (a tube 10 cm in length). Each time, immediately before withdrawing the blood samples for analysis (1 ml each), 1 ml samples of blood were taken in order to eliminate the blood from the catheter and the T-set. One part of each sample (90 μl) was used for immediate detection of blood gases (PO_2 and PCO_2) and pH. The second part (0.5 ml) was placed in 1.8 ml Eppendorf tubes containing 1 mg ammonium oxalate and 5 mg sodium fluoride and mixed for about 20 s. Subsequently, in order to separate the plasma for performing lactate and ammonia measurements, the blood samples were centrifuged. Samples of blood plasma (200 μl) were stored for further analysis at a temperature of −25 °C. PO_2 and PCO_2, as well as pH were determined using a Ciba-Corning analyser 238 (UK). The blood bicarbonate concentration (HCO_3^-) was also estimated by this unit. The percentage of body fat was assessed according to Hassager *et al.* (1986).

Statistical analysis

The results are expressed as the means and standard deviation (±S.D.). The significance of differences between the level of variables reached between the pre-exercise level or the 3rd and 6th minute of cycling at different pedalling rates were tested by analysis of variance for repeated measurements. The changes in blood lactate concentration, pH, HCO_3^- and NH_3 occurring between the pre-exercise level and the values reached in the 6th minute of cycling were tested using the paired Student's t-test. The significance level was set at $P<0.05$.

Table 1. Pre-exercise (0) and the net VO_2 reached during cycling at different pedalling rate.

	net VO_2 (l/min)				
Pedalling rate (rev/min)	**40**	**60**	**80**	**100**	**120**
Time (sec)					*
0	0.360±0.065	0.341±0.027	0.337±0.410	0.343±0.051	0.362±0.051
60	0.588±0.167	0.676±0.136	0.707±0.168	0.899±0.174	1.243±0.213
120	1.376±0.323	1.222±0.538	1.930±0.334	1.798±0.263	2.325±0.305
180	1.452±0.227	1.457±0.265	1.623±0.249	1.832±0.259	2.477±0.295
300	1.462±0.202	1.390±0.260	1.624±0.199	1.835±0.246	2.586±0.418
240	1.412±0.222	1.459±0.267	1.593±0.231	1.822±0.260	2.482±0.397
360	1.433±0.252	1.452±0.254	1.640±0.211	1.889±0.214	2.607±0.411

*Data are means ± S.D., * significantly different ($p<0.01$) from other pedalling rates (ANOVA for repeated measures).*

Results

The level of power output during the 6 min exercise corresponding to 50 % VO_2 max established during an incremental test performed at the pedalling rate of 70 rev.min^{-1} was 132.0±30.6 W. The mean value of power output corresponding to 50 % VO_2 max was 8.3±10.8 W below the lactate threshold (LT). With the exception of one subject whose power output during constant power output cycling exceeded the LT determined during the incremental exercise test only by 5 W, all subjects exercised just below the LT power output.

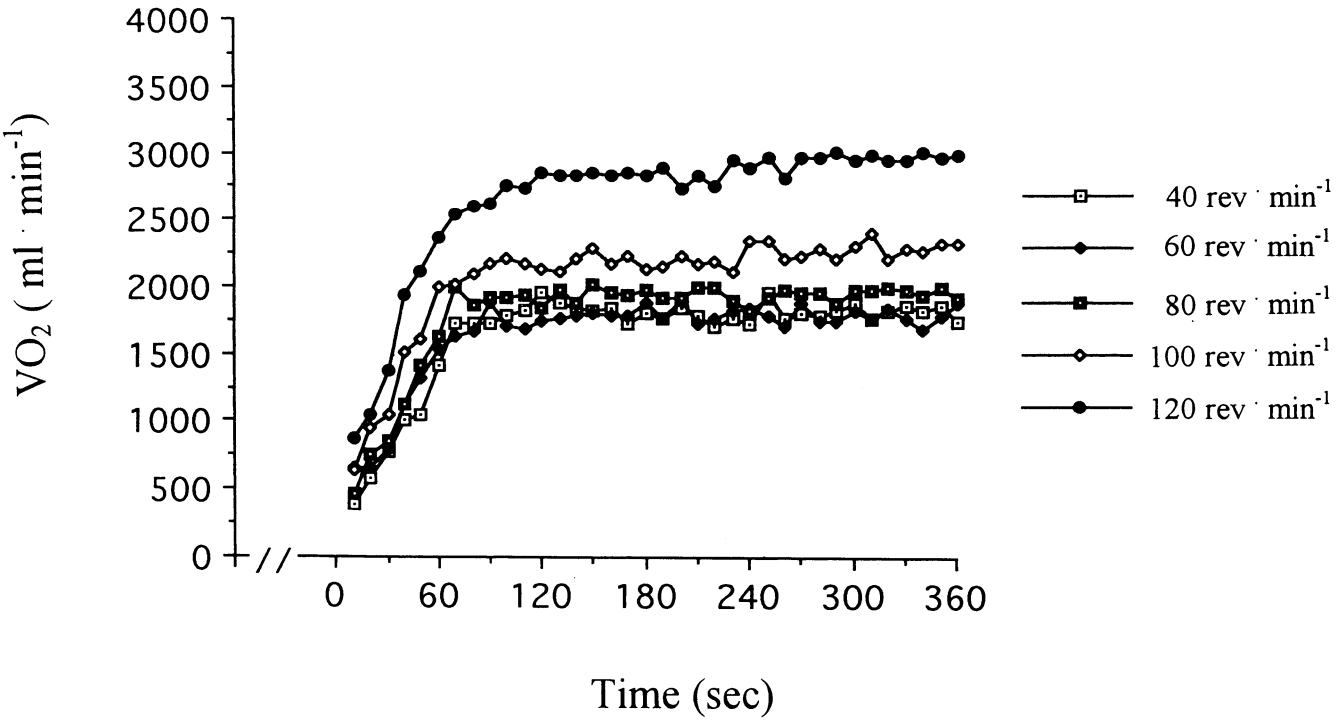

Fig. 1. *Oxygen uptake reached during cycling at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}. Data are given as mean values for 6 subjects at 37 ten-second intervals. The ANOVA test for repeated measures has shown no significant differences between the V_{O_2} reached at pedalling rates of 40, 60, 80 and 100 rev.min^{-1} but the V_{O_2} attained during cycling at 120 rev.min^{-1} was significantly higher than other values; ($P<0.01$).*

The V_{O_2} during the 6th minute of cycling at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1} amounted to 1.789±0.300, 1.793±0.270, 1.973±0.225, 2.214±0.242 and 2.971±0.448 l.min^{-1} respectively for each pedalling rate (Fig. 1). We have calculated that an average increase of V_{O_2} measured at the 6th minute of cycling in the range of pedalling rate between 40–100 rev.min^{-1} amounted to 0.142 l.min^{-1} per 20 rev.min^{-1}, whereas the difference in V_{O_2} reached during cycling at 100 rev.min^{-1} and 120 rev.min^{-1} amounted to 0.747 l.

As is illustrated by the data presented in Figure 1 and Table 1, the gradual increase of pedalling rate up to 100 rev.min^{-1} did not cause a significant increase in V_{O_2}. However, the increase of pedalling rate from 100 to 120 rev.min^{-1} was accompanied by a significant ($P<0.01$) increase in the net V_{O_2} values (Table 1). The total net oxygen consumed throughout the 6 min cycling period at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}, amounted to 7.727±1.197, 7.705±1.548, 8.679±1.262, 9.945±1.435 and 13.720±1.862 l, respectively for each pedalling rate. The net V_{O_2} values reached between the 4th and 6th minute of cycling at different pedalling rates were transformed into watts. This was done by using an appropriate O_2 equivalent of J.ml^{-1}, at a given respiratory exchange ratio (RQ) values. The RQ values during cycling at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}, amounted to 0.88±0.05, 0.89±0.04, 0.92±0.07, 0.95±0.03 and 0.99±0.08, respectively. The calculated mechanical efficiency amounted to 26.6±2.7, 26.4±2.0, 23.4±3.4, 20.3±2.6 and 14.7±2.2 %, respectively, for pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}.

The increase in the magnitude of the slow component of V_{O_2} kinetics (V_{O_2} 6th-3rd min) (see Whipp and Wasserman 1972) at any of the applied pedalling rates was not significant (Fig. 1). It amounted to −0.022±0.056, −0.009±0.029, 0.012±0.073, 0.030±0.081 and 0.122±0.176 ml.min^{-1} for pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}, respectively.

There was no significant accumulation of blood lactate above the resting value during the 6th minute of cycling at pedalling rates of 40–100 rev.min^{-1}. The antecubital plasma lactate concentration in the 6th min of cycling was 1.83±0.48, 1.80±0.48, 2.33±0.88 and 2.52±0.33 mmol.l^{-1}· respectively, for pedalling rate of 40, 60, 80 and 100 rev.min^{-1} (Fig. 2). However, an increase of pedalling rate from 100 to 120 rev.min^{-1} was accompanied by a significant rise in blood lactate concentration ($P<0.01$) from 1.31±0.26 mmol.l^{-1} measured under pre-exercise conditions to 5.92±2.46 mmol.l^{-1} reached in the 6th minute of cycling at 120 rev.min^{-1} (Table 2).

Table 2. Antecubital plasma lactate concentration and blood pH during cycling at different pedalling rate.

	$[La]_{pl}$ (mmol · l^{-1})					pH				
Pedalling rate (rev · min^{-1})	**40**	**60**	**80**	**100**	**120**	**40**	**60**	**80**	**100**	**120**
Time (sec)					*					*
0	1.63 ± 0.37	1.36 ± 0.36	1.47 ± 0.29	1.35 ± 0.25	1.31 ± 0.26	7.353 ± 0.027	7.348 ± 0.035	7.364 ± 0.048	7.348 ± 0.029	7.355 ± 0.039
60	1.42 ± 0.26	1.32 ± 0.35	1.32 ± 0.26	1.27 ± 0.21	1.40 ± 0.30	7.351 ± 0.017	7.343 ± 0.024	7.359 ± 0.029	7.339 ± 0.026	7.355 ± 0.031
120	1.58 ± 0.30	1.42 ± 0.33	1.50 ± 0.33	1.52 ± 0.23	2.50 ± 1.29	7.347 ± 0.022	7.339 ± 0.027	7.351 ± 0.026	7.330 ± 0.027	7.320 ± 0.049
180	1.73 ± 0.41	1.57 ± 0.34	1.72 ± 0.44	1.88 ± 0.26	3.63 ± 1.84	7.340 ± 0.027	7.329 ± 0.26	7.341 ± 0.030	7.319 ± 0.028	7.300 ± 0.061
240	1.85 ± 0.55	1.75 ± 0.41	1.82 ± 0.58	2.18 ± 0.25	4.53 ± 2.19	7.341 ± 0.026	7.325 ± 0.029	7.336 ± 0.028	7.312 ± 0.026	7.298 ± 0.062
300	1.90 ± 0.64	1.83 ± 0.48	2.23 ± 0.74	2.43 ± 0.30	5.18 ± 2.16	7.340 ± 0.024	7.327 ± 0.026	7.338 ± 0.029	7.317 ± 0.027	7.291 ± 0.059
360	1.83 ± 0.70	1.80 ± 0.48	2.33 ± 0.88	2.52 ± 0.33	5.92 ± 2.46	7.342 ± 0.027	7. 332 ± 0.027	7.340 ± 0.023	7.319 ± 0.028	7.296 ± 0.060

*Data are means ± S.D., * significantly different ($p < 0.01$) from other pedalling rates (ANOVA for repeated measures).*

Table 3. Antecubital blood bicarbonate and ammonia concentration during cycling at different pedalling rate.

	$[HCO_3^-]$ (mmol · l^{-1})					$[NH_3]$ (μmol · l^{-1})				
Pedalling rate (rev · min^{-1})	**40**	**60**	**80**	**100**	**120**	**40**	**60**	**80**	**100**	**120**
Time (sec)					*					
0	25.2 ± 1.1	24.8 ± 1.1	25.4 ± 0.6	25.9 ± 0.5	26.3 ± 1.3	34.3 ± 3.8	34.2 ± 5.7	39.5 ± 6.6	34.5 ± 5.0	35.7 ± 3.7
60	25.8 ± 1.3	25.1 ± 0.9	25.7 ± 0.9	26.4 ± 0.3	26.5 ± 1.4	33.2 ± 4.3	34.0 ± 8.1	37.8 ± 5.7	31.7 ± 4.6	36.2 ± 5.8
120	25.8 ± 1.3	24.9 ± 1.3	25.7 ± 0.8	25.9 ± 0.4	25.4 ± 1.7	35.2 ± 4.9	33.2 ± 7.0	36.2 ± 5.6	33.7 ± 4.5	34.8 ± 4.7
180	25.5 ± 1.2	24.7 ± 0.7	25.4 ± 0.7	25.5 ± 0.5	24.3 ± 2.3	34.3 ± 4.1	33.3 ± 7.0	36.7 ± 8.0	34.8 ± 3.2	38.3 ± 4.3
240	25.4 ± 1.3	24.0 ± 1.4	25.3 ± 0.7	25.0 ± 0.5	23.6 ± 2.4	35.2 ± 5.9	33.3 ± 5.5	39.2 ± 7.8	34.3 ± 4.1	41.8 ± 7.0
300	25.2 ± 1.3	24.0 ± 0.8	25.0 ± 0.8	24.7 ± 0.5	23.0 ± 2.3	36.0 ± 7.3	35.3 ± 5.5	37.7 ± 4.7	42.5 ± 12.2	58.2 ± 19.1#
360	25.0 ± 1.6	24.0 ± 1.0	24.5 ± 1.2	24.6 ± 0.5	22.6 ± 2.7	37.2 ± 6.0	35.3 ± 4.7	39.8 ± 7.3	41.3 ± 6.4	55.8 ± 17.8

*Data are means ± S.D., # n = 5, * significantly different ($p < 0.01$) from other pedalling rates (ANOVA for repeated measures).*

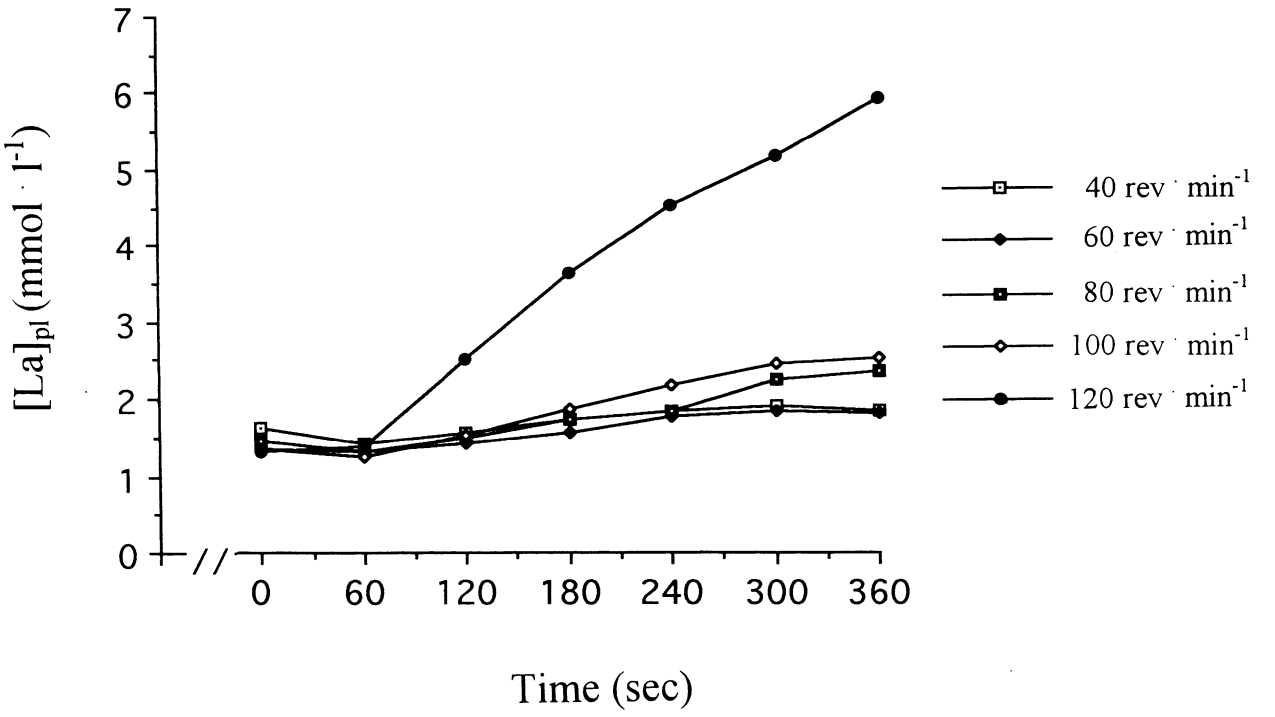

Fig. 2. *Antecubital venous plasma lactate concentration $[La]_{pl}$ reached during cycling at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1}. Data are given as mean values for 6 subjects, at one-minute intervals. No significant differences between $[La]_{pl}$ values occurred at pedalling rates of 40, 60, 80 and 100 rev.min^{-1} but the $[La]_{pl}$ attained during cycling at 120 rev.min^{-1} was significantly higher ($P<0.01$) than other values (ANOVA for repeated measures).*

Blood pH underwent a small change during cycling at pedalling rates between 40 to 100 rev.min^{-1}. In the 6th minute of cycling it amounted to 7.342±0.027, 7.332±0.027, 7.340±0.023 and 7.319±0.028, respectively, for pedalling rates of 40, 60 80 and 100 rev.min^{-1}. An increase of the pedalling rate from 100 to 120 rev.min^{-1} caused a significant decrease in blood pH ($P<0.01$), from 7.355±0.039 in the pre-exercise period to 7.296±0.060 in the 6th minute of cycling (Table 2).

Blood bicarbonate concentrations (HCO_3^-) during cycling at pedalling rates between 40–100 rev.min^{-1} were very similar. The values of HCO_3^- reached at the 6th minute of cycling were not significantly different from the pre-exercise values (Table 3). However, during cycling at the pedalling rate of 120 rev.min^{-1} there was a continuous decrease in HCO_3^-, from 26.3±1.3 mmol.l^{-1} in the pre-exercise period (Table 3) to 22.6±2.7 mmol.l^{-1} in the 6th minute of cycling ($P<0.01$).

The plasma ammonia concentration during cycling at pedalling rates of 40–120 rev.min^{-1} did not significantly differ from pre-exercise values. Cycling at the pedalling rate of 100 and 120 rev.min^{-1} was accompanied by a trend towards increased values of NH_3 from the pre-exercise level. However, this difference was not statistically significant (Table 3).

We also correlated the individual results for Δ $[La]_{pl}$ (between the pre-exercise level and the values reached in the 6th minute of cycling at the pedalling rate of 120 rev.min^{-1}) with the magnitude of the slow component of V_{O_2} kinetics reached at this pedalling rate. The correlation coefficient of this relationship was relatively low (r_{xy} = 0.54, P = 0.10).

Minute ventilation (V_E) during the 6th minute of cycling at pedalling rates of 40, 60, 80, 100 and 120 rev.min^{-1} was 41.5±10.8, 36.8±9.4, 44.8±8.6, 51.5±11.0 and 72.5±22.9 l.min^{-1}, respectively (Fig. 3). A gradual increase of pedalling rate in the range between 40–100 rev.min^{-1} was accompanied by only a small increase of V_E, but an increase of pedalling rate from 100 to 120 rev.min^{-1} was accompanied by a sharp increase in V_E, amounting to 21.0 l.min^{-1}.

Discussion

In this study we evaluated the effect of increasing the pedalling rate during cycling at a constant power output (50 % V_{O_2} max established at 70 rev.min^{-1}) on the pulmonary V_{O_2} and the

magnitude of the slow component of V_{O_2} kinetics in human subjects.

Our study has shown that, during cycling at a constant power output corresponding to 50 % of V_{O_2} max, an increase of pedalling rate in the range of 40–60 rev.min^{-1} had no effect on the oxygen uptake and the mechanical efficiency. However, as can be seen in Figure 1, an increase of the pedalling rate between 60 and 100 rev.min^{-1} was accompanied by a tendency towards a gradual (but not statistically significant) increase of net oxygen uptake and a decrease of mechanical efficiency.

The most important finding of our study concerns the fact that an increase of pedalling rate from 100 to 120 rev.min^{-1} caused a sudden increase in the net oxygen uptake (see Fig. 1, Table 1) and a significant decrease in mechanical efficiency from 20.3±2.6 % to 14.7±2.2 % ($P<0.01$). This drop in mechanical efficiency was accompanied by a significant rise in the plasma lactate concentration and significant changes in the acid-base balance towards acidosis (see Fig. 1 and Tables 2 and 3). Surprisingly, however, the pronounced progressively increasing blood lactate accumulation and acidosis occurring during cycling at the pedalling rate of 120 rev.min^{-1} was not accompanied by a significant increase of the slow component of V_{O_2} kinetics (Fig. 1, Table 1).

One may argue the sharp increase of net oxygen uptake observed during cycling at the pedalling rate of 120 rev.min^{-1} may simply be caused by a greater recruitment of additional muscle groups involved in the stabilisation of posture of the subjects during cycling at this velocity. However, if this were indeed the case, one would expect to see significantly higher values of V_{O_2} max during cycling at pedalling rate of 120 rev.min^{-1}. However, our previous studies (Zoladz and Rademaker 1994, Zoladz *et al.* 1995) have shown that the V_{O_2} max reached during maximal incremental tests was not different from those during cycling at pedalling rates of 60, 80, 100 or 120 rev.min^{-1}. But it should be mentioned that the power output reached at V_{O_2} max was lower by 60 W on the average ($P<0.01$) than during cycling at the lower pedalling rates mentioned above. This suggests that the significant increase in net V_{O_2} observed in the present study during cycling at the pedalling rate of 120 rev.min^{-1} represents a significant drop in the efficiency of leg muscles contracting at such a high velocity.

The increase in net oxygen uptake observed at high pedalling rates, representing a decrease in mechanical efficiency, may partly be due to the increase of mechanical work output needed to keep the limbs in motion defined as *internal work*. The effect of increasing pedalling rate on the oxygen cost of *internal work* during cycling has been evaluated in several studies (Wells *et al.* 1986, Widrick *et al.* 1992, Francescato *et al.* 1995).

Perhaps the most comprehensive analysis of the relationship between the pedalling rates and oxygen cost of *internal work* during cycling is presented in the study by Francescato *et al.* (1995). These authors calculated that *internal work* during cycling at pedalling rates of 40, 60, 80, and 100 rev.min^{-1} amounted to 4.9, 26.0, 86.1 and 218 W, respectively, for each pedalling rate. These results closely confirm those of the study by Gaesser and Brooks (1975). However, in none of these studies was the magnitude of *internal work* performed during exercise evaluated when cycling at pedalling rates amounting to 120 rev.min^{-1}.

Interestingly, the calculation of *internal work* during cycling in the study by Francescato *et al.* (1995) was performed using the equation of Di Prampero *et al.* (1979). This was based on the theoretical analysis of lower limb function during cycling, which provides significantly lower values at high pedalling frequencies amounting to 8.3, 28.0, 66.4 and 130 W at pedalling rates of 40, 60, 80 and 100 rev.min^{-1}, respectively. Moreover, van Ingen-Schenau *et al.* (1990) have calculated that the lost energy (*internal work*) may only amount to ~ 28 W during cycling at 88 rev.min^{-1} and 320 W of the power output.

Using the equation of Francescato *et al.* (1995), we have estimated the *internal work* performed by our subjects. It amounted to 4.4, 24.2, 79.9 and 202.4 W for pedalling rates of 40, 60, 80, and 100 rev.min^{-1}, respectively. If one includes the *internal work* into the equation for calculation the mechanical efficiency, then its level increases significantly reaching the values 27.0, 28.0, 27.9 and 31.0 %, respectively, for pedalling rates of 40, 60, 80 and 100 rev.min^{-1}.

The above discussed data illustrate that an increase of pedalling rate contributes to the increase of the *internal work* which has to be taken into consideration when studying the mechanical efficiency of human muscles during cycling at different pedalling rates. However, as was discussed above the magnitude of *internal work* determined by different methods may vary significantly. We postulate that the magnitude of *internal work* may modify the degree of recruitment of type II muscle fibres. Assuming that the performance of fast movements requires greater recruitment of type II muscle fibers, which appear to be less efficient than type I (Crow and Kushmerick 1982, Coyle *et al.* 1992), then the *internal work* performed to keep the limbs in motion may increase as the need for recruitment of type II muscle fibres increases. However, to our knowledge, no study has been performed to evaluate the magnitude of *internal work* during cycling at different pedalling rates in relation to the muscle fibre recruitment.

Moreover, the increase of *internal work* during cycling at a pedalling rate of 120 rev.min^{-1} may be at last partly caused by less optimally directed forces at such a high velocity of movements (Patterson and Moreno 1990, Bauer *et al.* 1994).

In view of the early reports showing that the appearance of the slow component of VO_2 kinetics is always accompanied by an increase in blood lactate concentration (Casaburi *et al.* 1987, 1989, Roston *et al.* 1987, Koike *et al.* 1990), it seems to be surprising that we do see significant blood lactate accumulation accompanied by a significant acidosis without the appearance of the slow component of oxygen uptake kinetics in our study during cycling at the pedalling rate of 120 rev.min^{-1}. It has been shown that the magnitude of the slow component of the VO_2 kinetics positively correlates with the degree of blood lactate accumulation (Casaburi *et al.* 1987, 1989, Roston *et al.* 1987, Koike *et al.* 1990). However, we did not find a significant correlation between the individual values of the increase in $[La]_{pl}$ (between the pre-exercise level and the values reached in the 6th minute of cycling at the pedalling rate of 120 rev.min^{-1}) with the magnitude of the slow component of VO_2 kinetics reached at this pedalling rate. The correlation coefficient in this relationship was rather poor (r_{xy} = 0.54, P = 0.10).

Our study has shown that a significant increase of the net VO_2 occurring during cycling at 120 rev.min^{-1} (Fig. 1, Table 1) was indeed accompanied by a significant blood lactate accumulation and acidosis (Fig. 2, Table 2). This suggests that even in the case of such a rapid frequency of movements there is a link between exercise-induced metabolic acidosis and the drop in muscle efficiency. More evidence for the close relation between the magnitude of the slow component of VO_2 kinetics and $[La]_{pl}$ may be provided by endurance training which decreased blood lactate concentrations accompanied by a subsequent decrease in the magnitude of the slow component of VO_2 kinetics, as reported in several independent reports (Casaburi *et al.* 1987, Roston *et al.* 1987, Womack *et al.* 1995). Moreover, it has recently been proposed that blood lactate and hydrogen ion accumulation play an important regulatory role in the slow component of VO_2 kinetics. For example Stringer *et al.* (1994) have suggested that the slow component of VO_2 kinetics is simply caused by lactic acid accumulation causing a shift of the oxyhaemoglobin dissociation curve to the right and increasing oxygen transport into the muscle. According to Capelli *et al.* (1993), acidosis may contribute to the upward drift in oxygen uptake by intensification of the rate of mitochondrial respiration in the muscle, due to an increase in free creatine, as was already suggested by Mahler (1985). The rise in the free creatine may be caused by a shift of the equilibrium of the creatine kinase reaction caused by the accumulation of H^+ (Harris *et al.* 1977). However, the creatine production does not stimulate mitochondrial respiration by itself, but probably *via* phosphate production. However, the shift of the creatine kinase reaction towards creatine synthesis also increases then ATP/ADP ratio, which can be expected to slow down oxygen consumption. For this reason, the relevance of the mechanism proposed by Capelli *et al.* (1993) is not so obvious.

It should be noted that we have recently evaluated the influence of pre-exercise acidification induced by ingestion of 3 mmol.kg BW^{-1} of ammonium chloride on oxygen expenditure of cycling and the slow component of VO_2 kinetics. We have shown (Zoladz *et al.* 1997) that a significant down-ward shift of blood pH, ($P<0.01$), had no significant effect on the oxygen cost of cycling during 6 min of exercise performed at 75 % VO_2, but we observed that enhanced acidosis, consistently in the case of each individual subject (n = 5), was accompanied by a higher value of the slow component of VO_2 kinetics. Moreover, as reported by Zoladz *et al.* (1995), when an incremental exercise test is performed, the increase of the power output above the onset of blood lactate accumulation is accompanied by an increase in the VO_2/power output ratio, leading to a positive curvilinear VO_2-power output relationship. The true oxygen cost of the final power output attained in the incremental test was 14–17 % higher ($P<0.01$) than expected from the initial linear relationship occurring only at the low power output (below the onset of blood lactate accumulation). This illustrates that blood lactate accumulation and acidosis are indeed associated with decreased muscle efficiency (Zoladz *et al.* 1995, Zoladz *et al.* 1998a,b).

On the other hand, there is good evidence from animal experiments (Poole *et al.* 1994) as well as from human studies (Gaesser *et al.* 1994, Womack *et al.* 1995, Zoladz *et al.* 1996) that blood lactate *per se* does not mediate the magnitude of the slow component of VO_2 kinetics. Moreover, Zoladz *et al.* (1996) have shown that significant alkalosis accompanied by enhanced lactacidaemia induced by ingestion of 3 mmol.kg BW^{-1} of $NaHCO_3$ did not affect the rate of VO_2 at the onset of exercise or the magnitude of the slow component of VO_2 kinetics in man. Moreover, the results of the present study provide more evidence showing that a significant increase in blood lactate concentration and H^+ accumulation, as illustrated by the data from cycling at 120 rev.min^{-1}, do not correspond with the magnitude of the slow component of VO_2 kinetics. Moreover, as reported by Gaesser *et al.* (1994), the epinephrine-induced increase in the blood lactate concentration did not affect the magnitude of the slow component of VO_2 kinetics.

One may argue that if the $NaHCO_3$ or epinephrine-induced changes in blood pH and blood lactate concentration do really reflect the metabolic changes induced during normal muscle activity. However, in this study we have followed the changes in blood lactate concentration and H^+ accumulation representing the rate of lactate elimination from working muscle cells. As illustrated by the data presented in Figure 1 a progressive increase in blood lactate and H^+ accumulation is not accompanied by a

further increase in V_{O_2} throughout the 6 min of cycling at the pedalling rate of 120 rev.min^{-1}. Thus, the participation of blood lactate and H^+ accumulation in the mechanisms responsible for the slow component of V_{O_2} kinetics, as suggested by Capelli *et al.* (1993) and Stringer *et al.* (1994), seems to be at least questionable. As is shown in Figures 1 and 2 we could demonstrate that during cycling at the pedalling rate of 120 rev.min^{-1}, the significant level of plasma lactate accumulation was not accompanied by an appearance of the slow component of V_{O_2} kinetics. This illustrates that even in case of blood sampling from a rather disadvantageous site, the antecubital vein (where the level of lactate accumulation is lower than in the artery) (Poortmans *et al.* 1978, Yoshida *et al.* 1982, Granier *et al.* 1996), we were able to demonstrate a clear dissociation between the appearance of the slow component of V_{O_2} kinetics and blood lactate accumulation. The obtained results question the role of blood lactate accumulation in the mechanism responsible for the slow component of V_{O_2} kinetics postulated by several investigators (Casaburi *et al.* 1987, Koike *et al.* 1990, Stringer *et al.* 1994). It could be expected that using arterial or arterialized venous blood samples during cycling at a pedalling rate of 120 rev.min^{-1}, would demonstrate even greater dissociation between the degree of lactacidaemia and the slow component of V_{O_2} kinetics than that found in our study. This is why we have postulated that such a study should be performed.

The significant increase in the net V_{O_2} observed at pedalling rate of 120 rev.min^{-1} may also be due to an increase of minute ventilation and the cost of respiratory muscle activity. In order to evaluate the potential contribution of the increased cost of minute ventilation to the increase in oxygen uptake observed during cycling at the pedalling rate of 120 rev.min^{-1}, we used the calculation proposed by Aaron *et al.* (1992) and we estimated the additional V_{O_2} caused by an increase in minute ventilation during cycling at the pedalling rate of 120 rev.min^{-1} which amounted to about 40 ml. Thus, the difference in V_E between cycling at 100 and 120 rev.min^{-1} would account for only about 5 % of the difference in V_{O_2}, amounting to ~ 750 ml.min^{-1}.

It is still puzzling what is the link between the rise of blood lactate concentration accompanied by acidosis and the observed increase in the oxygen cost during high intensity exercise. Our study has shown that significant increase of the net V_{O_2} (see Fig. 1) and the drop in mechanical efficiency occurring during cycling at 120 rev.min^{-1} was indeed accompanied by a significant blood lactate accumulation and acidosis (see Fig. 2 and Table 2). However, as mentioned above, it seems to be rather unlikely that the increase in blood lactate accumulation and/or H^+ concentration is the main cause of the increase in the oxygen cost of cycling. We do believe that the most likely explanation of the increased net V_{O_2} occurring during cycling at the pedalling rate of 120 rev.min^{-1} is due to a greater recruitment of type II muscle fibres, and the accompanying acidosis is only secondary to their activity, caused by an intensification of anaerobic glycolysis. Moreover, during cycling at the pedalling rate of 120 rev.min^{-1} type I muscle fibres may be operating at a velocity exceeding their optimum for mechanical efficiency and utilised more oxygen at this velocity of movement (Suzuki 1979, Sargeant 1996).

It has been shown that type I muscle fibres are more efficient in terms of oxygen cost of cycling at a given power output than type II (Coyle *et al.* 1992). Thus minimum recruitment of type II muscle fibres is required to obtain the highest efficiency. It seems likely that, in order to generate the required power output during cycling at the pedalling rate of 120 rev.min^{-1}, it is necessary for the system to recruit a substantial number of type II muscle fibres (for review see Rome 1993, Sargeant and Jones 1995). If this is indeed the case that the observed increase in net oxygen uptake and the decrease in mechanical efficiency are caused by recruitment of type II muscle fibres, then the absence of the slow component of V_{O_2} kinetics accompanied by progressive acidosis during cycling at the pedalling rate of 120 rev.min^{-1} suggests that type II muscle fibres are already recruited rapidly in a great majority from the start of exercise performed at the pedalling rate of 120 rev.min^{-1}.

Our study confirms the earlier findings (Hagberg *et al.* 1981, Zoladz and Rademaker 1994, Sargeant 1996) showing that the pedalling rate up to 105 rev.min^{-1} is the upper limit for sustained high power output exercise, above which there is a drop in muscle efficiency with an accompanying increase of metabolic acidosis, which will clearly be disadvantageous for prolonged high intensity exercise. We believe that the increase in the net V_{O_2} accompanied by acidosis observed at a high velocity of movements, as illustrated during cycling at 120 rev.min^{-1} has the same origin (greater recruitment of type II muscle fibres) as the non-linear increase in oxygen uptake occurring at high power output incremental exercise performed above the lactate threshold, as recently described by Zoladz *et al.* (1995, 1998a,b).

We have demonstrated in the present study that cycling even at a low power output, exceeding a critical threshold value of velocity of cycling (100 rev.min^{-1} in our subjects) causes a significant drop in mechanical efficiency, illustrated by an increase of net V_{O_2}. This is accompanied by a significantly elevated level of plasma lactate concentration and a significant drop in blood pH. However, as was shown during cycling at the pedalling rate of 120 rev.min^{-1}, a pronounced increase in blood lactate and ammonia concentration accompanied by a significant drop in blood pH is not necessarily accompanied by the slow

component of the V_{O_2} kinetics. We have therefore concluded that metabolic acidosis does not play a major regulatory role in the slow component of V_{O_2} kinetics.

Acknowledgment

This study was supported by a grant from AWF-Krakow to Dr. Jerzy A. Zoladz. Local ethical committee approval was obtained for this study.

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Reprint requests

Dr. Jerzy A. Zoladz, Department of Physiology and Biochemistry, AWF-Krakow, Al. Jana Pawla II 78, 31-571 Krakow, Poland, e-mail: wfzoladz@cyf-kr.edu.pl.