

INFLUENCE OF A PRIOR ANAEROBIC LOAD ON THE HEART RATE KINETICS DURING INTERMITTENT EXERCISE OF PROGRESSIVE INTENSITY

by

ARVIDAS STASIULIS, LORETA DUBININKAITE,
ZANETA MACIUNIENIE, PRANAS MOCKUS

The aim of the study was to examine the influence of a prior anaerobic load (PAnL) on heart rate (HR) kinetics during intermittent exercise at varied work rate (WR). Each subject (n=15) cycled in two tests consisting of an intermittent, gradually increasing exercise (pedal frequency – 70 rpm, 1st load – 25 W, duration – 4 min, duration of other loads – 3 min). The loads were increased gradual by 25 W until the HR was above 150 b/min. The passive rest duration – 3 min. Prior to one test as PAnL the Wingate test was performed. The blood samples were taken at fifth and 20th min after PAnL. The HR kinetics was analyzed by adopting monoexponential function. The results showed that the primary change in exercise and recovery heart rate after PAnL was an increase in steady state level at the WR equal to or lower than the lactate threshold. The time courses of HR increase or decrease were almost identical under both testing conditions. This phenomenon may be explained by increased circulation of catecholamines and acidosis without changing HR kinetics.

Key words: heart rate, exercise intensity, acidosis, lactate threshold.

Introduction

Many studies have been focused on the oxygen uptake (VO_2) kinetics during on and off transitional periods of exercise. There remains considerable debate as to whether the speed of these kinetics reflects sluggishness of O_2 delivery to the muscle or, alternatively, some intramuscular limitation such as microvascular O_2 delivery- to- O_2 requirement mismatch or oxidative enzyme inertia (Linnarson 1974, Whipp and Ward 1990, Tschakovsky and Hughson 1999). The relative role of these mechanisms may vary under different conditions of exercise e.g., prior exercise (Tschakovsky and Hughson 1999).

The heart rate (HR) analysis during work and recovery may reflect O₂ transportation dynamics and is widely used in studies. HR increases during exercise in response to a combination of sympathetic activation and parasympathetic withdrawal, with the reverse occurring during recovery following exercise (Orizio et al. 1988, Perini et al. 1989, Pierpoint et al. 2000). Hormonal and intrinsic mechanisms also play a certain role, especially at the higher intensities of exercise (Christensen and Galbo 1983, Wallin et al. 1987).

Many authors have shown that La kinetics during an incremental exercise reflects the transition from aerobic to anaerobic metabolism and fits the three segmental model with two “thresholds” (Kindermann et al. 1979, Skinner and McLellan 1980, Cabrera and Chizeck, 1996). The work rate (WR) separating the first two phases is defined by different terms: aerobic threshold (Kindermann et al. 1979, Skinner and McLellan, 1980), anaerobic threshold (Wasserman et al. 1973), lactate threshold (LT) (Beaver et al. 1985). Overcoming the exercise intensity at the LT level involves significant changes in many functional and biochemical variables. Kinetics of VO₂ during dynamic muscular exercise is influenced by exercise intensity, both with respect to model order and to dynamic asymmetries between the on- and off-transient responses (Ozyener et al. 2001). HR kinetics variables at the beginning of exercise and during recovery have been shown to be dependent on exercise intensity and blood catecholamines concentration (Orizio et al. 1988, Perini et al. 1989). HR kinetics during progressively increasing intermittent exercise has not been studied.

Lactate kinetics is known to be influenced by preceding anaerobic (intensive) exercise (Cerretelli et al. 1977). Faster VO₂ kinetics during high-intensity exercise has been reported after prior exercise of high-intensity (Gerbino et al. 1996, McDonald et al. 1997, Bohnert et al. 1998). As it has been demonstrated by Burnley et al. (2000) neither prior moderate exercise nor prior heavy exercise had any effect on the VO₂ kinetics during subsequent moderate exercise. HR kinetics may be dissociated from VO₂ kinetics during transitions from mild to heavy exercise (Bearden and Moffat, 2001).

Since lactate and catecholamines are important in HR regulation it may be speculated that HR dynamics would be changed after preceding anaerobic load.

The aim of the study was to examine the influence of a prior anaerobic load (PAnL) on heart rate (HR) kinetics during intermittent exercise of progressive work rate (WR).

Material and methods

Subjects

A group of physically active young women (n=15), mean age 20,9±4,6 years, volunteered to participate in this study. Their mean body mass was 56,6±6,8 kg and their mean height was 1,66±0,06 m. All the subjects were involved in regular sport aerobics gymnastics. The subjects were non-smokers and none of them had a relevant medical history. They were required not to train on the testing day and not to have hard training sessions the day before. All tests for each subject were performed within one-week interval, in random order and at the same time of the day.

Intermittent exercise test

The subjects performed graded intermittent exercise test on an electrically braked ergometer, while cycling at 70 rpm. The tests consisted of repeated 3 min work and 3 min passive rest intervals. No special warm-up was performed. The duration of the first work period was 4 min, the work load was set at 25 W. Thereafter the work rate was increased by 25 W during each consecutive work period. The test was continued until the subjects' HR at the end of the work period approached 75% of age predicted maximum (220 - age).

The Wingate anaerobic test

As the prior anaerobic load the supramaximal 30 s Wingate test was performed on Monark 834E cycle ergometer (Bar-Or 1981). The test was preceded by a warm-up consisting of 5 min cycling (25-50 W) interrupted by short lasting bursts of high intensity. After this warm-up, the subjects took a 1 min rest period for blood sampling. They performed 30 s all-out cycling followed by 1 min cool-down cycling with no resistance. The bicycle ergometer mechanical resistance was set at 7,5% body mass.

Lactate threshold estimation

The LT was estimated for each subject on the basis of the relationship of HR at 3 min of recovery and WR (Stasiulis, 1997).

HR measurement and analysis

The HR was recorded continuously every 5 s with a Polar Accurex Plus heart rate monitor (Polar Electro, Kempele, Finland). The transient responses of HR during on transition and recovery periods were analyzed by adopting the mono-exponential function:

$$\text{HR} (t) = y_0 + A \times e^{-t/\tau}$$

where:

y_0 – asymptote, A – amplitude, τ – time constant, t – time.

Blood lactate samples

Arterialized fingertip blood samples were obtained before, at 5 and 20 min after the Wingate test. Using a micropipette, a blood sample of 0,1 ml was drawn and immediately analyzed by an enzymatic membrane method (Exan-G analyzer, Kulis et al. 1988). Prior to blood analyses, the analyzer was calibrated with standard solutions.

Experiment protocol

Each subject completed two exercise tests to determine the blood lactate threshold and HR kinetics without and after PANL. On one day only the intermittent exercise test was carried out. On another day the subjects performed the Wingate anaerobic 30 s test, then rested (walking and sitting) for 20 min and finished with the graded intermittent exercise test. The number of working steps was the same as in the previous intermittent test.

Statistical analysis

All the data is presented as means and SD. Comparisons of HR kinetics parameters between testing conditions and among different (relative to LT) intensities were conducted using two-way analysis of variance with repeated measures and post-hoc Tukey test. The level of statistical significance was set at $p \leq 0,05$.

Results

The mean plasma lactate concentration at the 5th and 20th min after the 30 s Wingate test was $6,97 \pm 0,81$ mmol/l and $4,38 \pm 0,73$ mmol/l respectively indicating the presence of a residual metabolic acidosis at the start of the incremental exercise test.

HR values recorded every 5 s during the intermittent increasing exercise are shown for a representative subject in Figure 1. To compare the HR parameters between testing conditions (without and after PANL) data were normalized to each individual's LT. The HR on transition parameters is given in Table 1. The HR at 3 min of exercise, the asymptotic value and amplitude of monoexponential function increased in parallel with WR. The t values of monoexponential function also increased with WR except for two lowest and two highest WR where the difference was not significant. The amplitude and τ were not different after PANL at all WR investigated. On the contrary, the y_0 and HR at 3 min of exercise were significantly larger at the WR equal or less than LT. There was no significant difference for these parameters between the two testing conditions at WR greater than LT (Fig. 2). The index of HR changes did not depend on the WR nor demonstrated any significant changes after PANL.

Table 1. Parameters of HR changes during on transition periods of intermittent increasing exercise without and after preceding anaerobic load

		Work rate relative to lactate threshold									
		- 50 W		- 25 W		0		25 W		50 W	
		1		2		3		4		5	
HR3' (b/min)	PAnL	\bar{X}	S_x	\bar{X}	S_x	\bar{X}	S_x	\bar{X}	S_x	\bar{X}	S_x
	Without	*107,4	14,2	*118,1	15,2	*130,1	15,1	*145,9	19,9	163,0	13,6
	After	118,9	10,9	126,4	13,7	140,0	12,8	154,5	12,9	166,3	13,2
	p≤0,05	1-2; 1-3; 1-4; 1-5; 2-3; 2-4; 2-5; 3-4; 3-5; 4-5									
y0 (b/min)	Without	107,7	17,4	119,3	14,9	*132,0	16,5	153,7	18,0	169,0	15,6
	After	119,2	13,2	129,6	15,1	141,9	13,4	156,7	13,0	173,2	14,5
	p≤0,05	1-2; 1-3; 1-4; 1-5; 2-3; 2-4; 2-5; 3-4; 3-5; 4-5									
A (b/min)	Without	36,3	13,7	37,6	18,8	49,3	21,0	70,5	19,1	78,1	14,0
	After	33,3	13,0	37,4	13,4	50,2	20,2	65,7	17,0	75,3	20,7
	p≤0,05	1-3; 1-4; 1-5; 2-3; 2-4; 2-5; 3-4; 3-5; 4-5									
τ (s)	Without	25,1	12,6	27,6	12,5	39,4	16,2	52,1	16,6	55,5	11,6
	After	22,4	10,0	29,0	14,7	39,3	14,2	42,8	11,1	57,2	14,3
	p≤0,05	1-3; 1-4; 1-5; 2-4; 2-5; 3-5									
Index (b/s)	Without	1,03	0,39	0,96	0,51	0,87	0,34	0,91	0,29	0,91	0,20
	After	1,06	0,41	1,07	0,84	0,83	0,33	1,03	0,38	1,06	0,41

HR3' – mean HR during third min of exercise, y0, A, τ – asymptotic value, amplitude and time constant of monoexponential function, respectively, Index – $0,63 \cdot A/\tau$.
* – significant difference between testing conditions. p≤0,05 – significant difference between different WR.

Table 2. Parameters of HR changes during recovery periods of intermittent increasing exercise without and after preceding anaerobic load

		Work rate relative to lactate threshold									
		- 50 W		- 25 W		0		25 W		50 W	
		1		2		3		4		5	
HR3' (b/min)	PAnL	\bar{X}	S_x	\bar{X}	S_x	\bar{X}	S_x	\bar{X}	S_x	\bar{X}	S_x
	Without	*80,3	11,9	*83,9	10,9	*83,1	10,0	95,0	13,1	108,3	18,9
	After	92,1	9,3	94,3	10,0	93,1	11,0	101,1	13,5	108,5	13,7
	p≤0,05	1-4; 1-5; 2-4; 2-5; 4-3; 5-3; 5-4									
y0 (b/min)	Without	*78,6	12,1	*81,3	11,7	79,6	10,5	89,0	12,3	97,9	18,1
	After	88,8	10,4	92,0	13,2	86,9	15,3	92,9	16,1	98,7	19,1
	p≤0,05	1-5; 2-5; 3-4; 3-5									
A (b/min)	Without	34,3	14,1	44,9	17,4	60,8	19,2	67,9	16,1	75,6	15,3
	After	36,6	14,8	42,8	19,2	61,8	26,8	71,8	24,5	79,3	23,8
	p≤0,05	1-2; 1-3; 1-4; 1-5; 2-3; 2-4; 2-5; 3-5; 4-5									
τ (s)	Without	26,1	7,3	36,6	11,0	46,0	10,7	54,0	19,8	68,3	26,0
	After	37,6	24,4	33,0	14,6	50,6	21,4	55,2	22,2	60,0	20,1
	p≤0,05	1-4; 1-5; 2-4; 2-5; 3-5; 4-5									
Index (b/s)	Without	0,84	0,25	0,82	0,34	0,87	0,30	0,87	0,30	0,75	0,21
	After	0,77	0,38	0,87	0,28	0,78	0,24	0,84	0,16	0,85	0,09

HR3' - mean HR during third min of recovery, y0, A, τ - asymptotic value, amplitude and time constant of monoexponential function, respectively, Index - $0,63 \cdot A/\tau$.
* - significant difference between testing conditions. p≤0,05 - significant difference between different WR.

The HR on transition parameters is given in Table 2. The amplitude of monoexponential function increased in parallel with WR. The HR at 3 min of recovery and the asymptotic value demonstrated little changes at WR equal or less than LT but were significantly increased above LT. The τ values of monoexponential function also increased with WR except for two lowest WR where the difference was not significant. The amplitude and τ were not different after PAnL at all WR investigated. Similarly to on transition, the y_0 and HR at 3 min of recovery were significantly larger at the WR equal to or less than LT. There was no significant difference for these parameters between two testing conditions at WR greater than LT (Fig. 2). The index of HR changes did not depend on the WR nor demonstrated any significant changes after PAnL.

Discussion

We have demonstrated in this study that HR was elevated at the end of work and recovery of exercise at the intensities lower than LT after PAnL. There was no influence of PAnL on the HR mean response times or rates of HR changes. In addition we observed the increase of HR mean response times during on and off transitions in parallel with the exercise intensity under both conditions.

The elevated HR steady state level during work and recovery is consistent with the results reported for HR and VO_2 after prior heavy exercise. Thus, the HR at 5th min of submaximal exercise was elevated following heavy strength leg exercise (Crawford et al. 1991). It has been shown that prior running exercise of high-intensity induced a significant increase in VO_2 of a subsequent 6 min running exercise of low and moderate intensity (Zavorsky et al. 1998). Oxygen uptake during an incremental cycling exercise test to exhaustion was increased at the low-intensity levels when the test was repeated after a 5 min rest period (Davis and Gass 1981). Similarly, the ventilatory equivalent for O_2 was elevated at low intensities (15-60 W) during incremental cycling performed after high-intensity exercise (Schneider and Berwick 1998). However, the study by Yoshida et al. (1995) has shown that VO_2 and cardiac output kinetics may be dissociated during repeated exercise with the other leg. On the other hand, Hughson and Morrissey (1983) have observed coincident variation of HR and VO_2 during the transition from 40% to 80% of anaerobic threshold in comparison with that in the transition from rest to 40% of anaerobic threshold and have suggested that VO_2 kinetics were controlled by oxygen transport.

In this study we did not find any significant influence of PAnL on HR kinetics during on and off transitions to exercise intensities below and

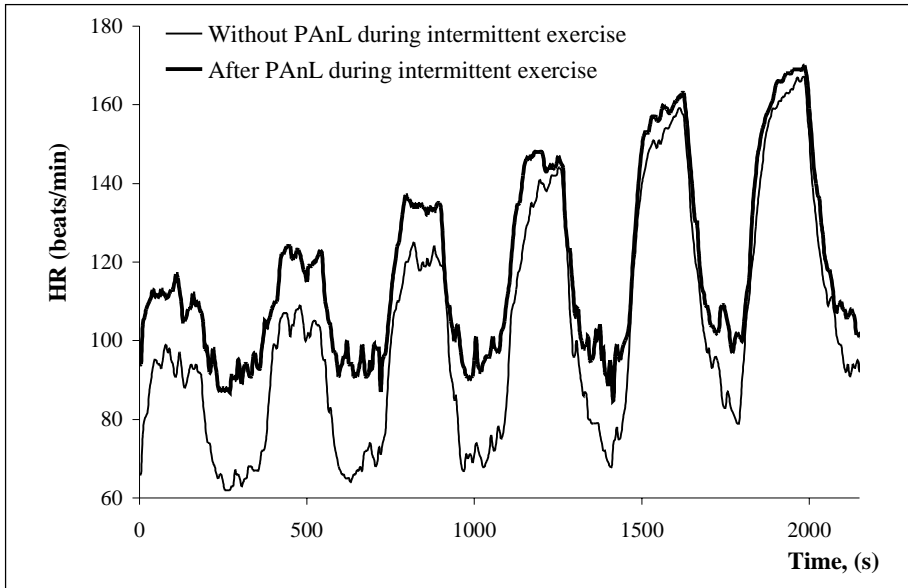


Fig. 1. Time course of HR in representative subject in two intermittent tests with-out and after a preceding anaerobic load

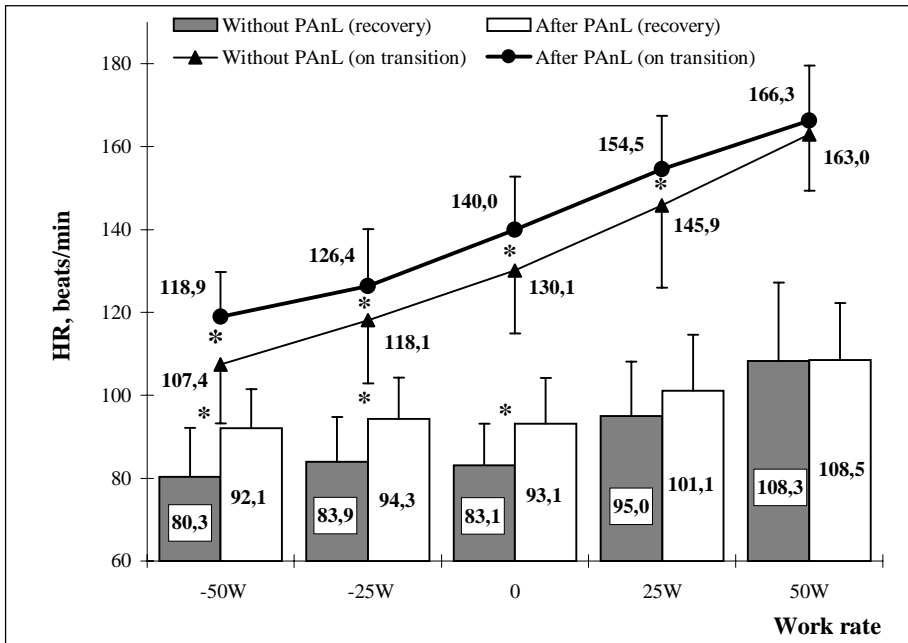


Fig. 2. Comparison of mean values of HR during 3 min of work and recovery at the same work rates relative to LT under different testing conditions (* - $p \leq 0,05$ significant difference between testing conditions)

above LT. In contrast to the present study Yoshida et al. (1995) have found that VO_2 and cardiac output kinetics were accelerated during repeated exercise with the same leg. When the exercise was changed to the other leg, VO_2 kinetics were significantly slower, although cardiac output kinetics continued to be faster. HR kinetics was slowed when the transition from elevated baseline to heavy WR was performed. The initial rise in HR was not different in moderate and two successive bouts of heavy exercise, but the time constant of slow component was slower during the repeated bout of heavy exercise (Bearden and Moffatt 2001). Pendergast et al. (1983) have found that the kinetics of VO_2 was accelerated during intensive (90% of $\text{VO}_{2\text{max}}$) exercise when lactate concentration was increased prior to exercise. Intermittent running at $\text{VO}_{2\text{max}}$ accelerated VO_2 kinetics in a subsequent run (Billat et al. 2000).

Several studies have demonstrated a reduction in VO_2 slow component after prior high-intensity exercise (Gerbino et al. 1996, Bohnert et al. 1998). A more detailed analysis has shown that the prior high-intensity exercise decreased only the VO_2 slow component but had no effect on the fast one (Burnley et al. 2000, Koppo and Bouckaert, 2000). McDonald et al. (2001) have found that muscle VO_2 was significantly elevated during the first minute of the second exercise bout compared with the first one.

The present study has also demonstrated the influence of exercise intensity on the HR rate kinetics during on and off transitions. We observed nonlinear relationship between HR at the end of recovery and the WR of exercise performed. This confirms our previous findings that HR during recovery is elevated when the WR exceeds LT (Stasiulis 1997). Time constants of HR kinetics during transitional periods were related to WR. In general, they increased with WR. It is of interest to note that the HR changing rate (63% percent of amplitude divided by time constant) remained practically unchanged during WR investigated. Our results are in agreement with findings by Orizio et al. (1988) and Perini et al. (1989), who observed increase of time constants during three different exercise intensities, both, during at beginning of exercise and during recovery.

The mean response time of HR, Ve and VO_2 was significantly slower in the transition from 40% to 80% of anaerobic threshold than that in the transition from rest to 40% of anaerobic threshold (Hughson and Morrissey, 1983). The phase I increase of VO_2 , HR and VO_2/HR was related to exercise intensity. At very low WR VO_2 and HR exceeded their steady state levels in the phase I. For higher WR mean response times were faster for VO_2/HR than for VO_2 and HR, suggesting that arteriovenous O_2 difference reached a steady state before cardiac output did (Sietsema et al. 1989).

Kinetics of VO_2 during dynamic muscular exercise was influenced by the exercise intensity, both with respect to model order and to dynamic asymmetries between the on- and off-transient responses (Ozyener et al. 2001). Barstow et al. (1993) have shown that, when the exercise response was described with a monoexponential term, the time constant was systematically slowed as the power output was increased above the LT. However, this slowing of VO_2 kinetics above the LT was not related to the slowing of the phase II VO_2 kinetics but rather to the inclusion of the slow component term in the monoexponential model. When the VO_2 response was mathematically modeled using discrete exponential terms to describe the phase II and slow component responses, the phase II VO_2 kinetics was invariant during exercise bouts ranging from 35% to 100% $\text{VO}_{2\text{max}}$. On the contrary, time constant for phase II O_2 kinetics during cycle ergometer exercise was slowed in transition from one power output to a higher one in the upper reaches of the moderate intensity domain compared to the same work rate increment in lower reaches (Brittain et al. 2001).

Despite the 15 min rest period separating the anaerobic prior load from the intermittent exercise test subjects still had a high blood lactate levels. The sympathetic activity has been shown to be persistent after the cessation of moderate and heavy exercise (Dimsdale et al. 1984, Perini et al. 1989).

The primary effect of PAnL on HR observed in this study was an increase in the asymptotic values during work and recovery when WR was lower or equal to LT. This is possibly due to an increase in circulating catecholamines, resulting from spillover of the greater sympathetic nerve activation that has been reported to take place under anaerobic conditions (Dimsdale et al. 1984). This might have also been due to peripheral reflexes that stimulate cardiac output when the pH decreases. Indeed, the mechanisms of HR elevation during recovery are not quite clear. For passive recovery in the seated position, the existence of muscle metaboreflex – sustained elevation of HR should be considered. The reflex control of HR might be due to sympathetic nerve activation elicited by afferents of muscle metaboreceptors within the lower limbs at post-exercise in which an amount of venous blood is pooled. Alternatively, there is a possibility that the elevation of resting HR was modulated by arterial baroreflex, which might be related to post-exercise hypotension (Floras et al. 1992). Thus, in response to 30 s bout of maximal cycling a cardiac (and possibly respiratory) compensation of metabolic acidosis occurred during a subsequent incremental exercise test in an attempt to return arterial pH to normal level. The possibility exists that lactate actually begins to accumulate in the muscle at WR below the LT because of elevated catecholamines levels and pyruvate con-

centration in the recovery from the prior supramaximal exercise or because of muscle fatigue resulting in earlier recruitment of type II motor units (Carter et al. 1999). Davis and Gass, (1981) have shown that since the incremental portion of the lactate minimum test commenced while the subject had high levels of blood lactate (~8 mM), the incremental test produced a “U-shaped” blood lactate profile. It has been considered that in the early stages of the incremental test there was a net lactate clearance which caused blood lactate to decrease, while in the latter stages of the test there was a net lactate production which caused blood lactate to increase (Carter et al. 1999). Despite the relative role of oxygen transport and the oxygen utilization for the control of VO_2 kinetics is not well understood. Some mechanisms that cause VO_2 alterations may have influence on HR as well. The effect of the prior high-intensity exercise on VO_2 is often related to the subsequences of induced metabolic acidosis such as vasodilatation and improved blood flow, and a rightward shift of the O_2 -haemoglobin dissociation curve increasing the O_2 diffusion gradient between the capillary blood and the mitochondrial, leading to an improvement in O_2 availability at the start of the second period of exercise (Gerbino et. al. 1996).

This increase in oxygen uptake could be related to an increase in body temperature, a change in substrate use, an increase in catecholamines level and/or lactate metabolism. It is known that heavy loads markedly increase the temperature not only in the working muscle but in other muscles as well (Knuttgen et al. 1982). However, recently, Koga et al. (1997) have demonstrated that elevated temperature did not contribute to the slow component and did not accelerate oxygen kinetics.

Our data and the analysis by Bearden and Moffatt (2001) suggest no effect of PAnL on HR kinetics during transitional phases of exercise. These observations for intensive exercise are somewhat surprising given that, under anaerobic conditions, increasing cardiac frequency at higher HR is primarily achieved by sympathetic activation (Robinson et al. 1966), a slower process than parasympathetic removal, which is the likely mechanism for initial cardiac acceleration at lower HR (Fagraeus et al. 1976). The data on heart rate kinetics obtained suggest that direct neural cardiac control (sympathetic and parasympathetic) during exercise may be unaltered above LT compared with that below LT and that the primary change in exercise and recovery heart rate after PAnL is one of a shift in steady state level, presumably due to increased circulating catecholamines rather than to changes in kinetic control.

REFERENCES

- Bar-Or O. 1981. *Le tests anaerobic de Wingate*. Symbioses 13: 157-172.
- Barstow T.J., Casaburi R., Wasserman K. 1993. *O₂ uptake kinetics and the O₂ deficit as related to exercise intensity and blood lactate*. J. Appl. Physiol. 75: 755-762.
- Bearden S.E., Moffatt R.J. 2001. *Leg electromyography and the VO₂-power output relationship during incremental cycle ergometry*. Med Sci Sports Exerc, In press.
- Beaver W.L., Wasserman K., Whipp B.J. 1985. *Improved detection of lactate threshold during exercise using a log-log transformation*. J. Appl. Physiol., 59: 1936-1940.
- Billat V.L., Bocquet V., Slawinski J., Laffite L., Demarle A., Chassaing P., Koralsztein J.P. 2000. *Effect of a prior intermittent run at VO_{2max} on oxygen kinetics during an all-out severe run in humans*. J. Sports Med. Phys. Fitness 40: 185-194.
- Bohnert B., Ward S.A., Whipp B.J. 1998. *Effects of prior arm exercise on pulmonary gas exchange kinetics during high-intensity leg exercise in humans*. Exp. Physiol. 83: 557-570.
- Brittain C.J., Rossiter H.B., Kowalchuk J.M. 2001. *Effect of prior metabolic rate on the kinetics of oxygen uptake during moderate-intensity exercise*. Eur. J. Appl. Physiol. 86: 125-134.
- Burnley M., Jones A.M., Carter H., Doust J.H. 2000. *Effects of previous heavy exercise on phase II pulmonary oxygen uptake kinetics during heavy exercise*. J. Appl. Physiol. 89: 1387-1396.
- Carter H., Jones A.M., Doust J.H. 1999. *Effect of incremental test protocol on the lactate minimum speed*. Med. Sci. Sports Exerc. 31: 837-845.
- Cabrera M., Chizeck H. 1996. *On the existence of a lactate threshold during incremental exercise: a systems analysis*. J Appl Physiol, 80(5): 1819-1828.
- Cerretelli P., Shindell D., Pendergast D., di Prampero P.E., Rennie D.W. 1977. *Oxygen uptake transients at the onset and offset of arm and leg work*. Respir. Physiol. 30: 81-97.
- Christensen N.J., Galbo H. 1983. *Sympathetic nervous activity during exercise*. Ann. Rev. Physiol. 45: 139-153.
- Crawford W.W., Loy S.F., Nelson A.G., Conlee R.K., Fisher A.G., Allsen P.E. 1991. *Effects of prior strength exercise on the heart rate oxygen*

- uptake relationship during submaximal exercise. J. Sports Med. Phys. Fitness, 31(4): 505-509.*
- Davis H.A., Gass G.C. 1981. *The anaerobic threshold as determined before and during lactic acidosis. Eur. J. Appl. Physiol. 47: 141-9.*
- Dimsdale J.E., Hartley L.H., Guiney T., Ruskin J.N., Greenblatt D. 1984. *Post exercise peril. Plasma catecholamines and exercise. JAMA 251: 630-632.*
- Fagraeus L. and Linnarsson D. 1976. *Autonomic origin of heart rate fluctuations at the onset of muscular exercise. J. Appl. Physiol. 40: 679-682.*
- Floras J.S., Wesche J. 1992. *Haemodynamic contributions to post-exercise hypotension in young adults with hypertension and rapid resting heart rates. J. Hum. Hypertens. 6(4): 265-269.*
- Gerbino A., Ward S.A., Whipp B.J. 1996. *Effects of prior exercise on pulmonary gas-exchange kinetics during high-intensity exercise in humans. J. Appl. Physiol. 80: 99-107.*
- Hughson R.L., Morrissey M.A. 1983. *Delayed kinetics of VO_2 in the transition from prior exercise. Evidence for O_2 transport limitation of VO_2 kinetics: a review. Int. J. Sports. Med. 4: 31-39.*
- Yoshida T., Kamiya J., Hishimoto K. 1995. *Are oxygen uptake kinetics at the onset of exercise speeded up by local metabolic status in active muscles. Eur. J. Appl. Physiol. 70: 482-486.*
- Kindermann W., Simon G. & Keul J. 1979. *The significance of the aerobic-anaerobic transition for the determination of work load intensities during endurance training. Eur. J. Appl. Physiol., 42(1): 25-34.*
- Knuttgen H.G., Nadel E.R., Pandolf K.B., Patton J.F. 1982. *Effects of training with eccentric muscle contraction on exercise performance, energy expenditure and body temperature. Int. J. Sports Med. 3: 13-17.*
- Koga S., Shiojiri T., Konda N., Barstow T.J. 1997. *Effect of increased muscle temperature on oxygen uptake kinetics during exercise. J. Appl. Physiol. 83: 1333-1338.*
- Koppo K., Bouckaert K. 2000. *In humans the oxygen uptake slow component is reduced by prior exercise of high as well as low intensity. Eur. J. Appl. Physiol. 83: 559-565.*
- Kulis Y.Y., Laurinavichyus V.S.A., Firantas S.G.A., Kurtinaitienė B.S. 1988. *Determination of lactic acid with an Exan-G analyser. J. Anal. Chem. of the USSR 43(7): 1521-1523.*
- Linnarson D. 1974. *Dynamics of pulmonary gas exchange and heart rate changes at start and of exercise. Acta. Physiol. Scand. [Suppl.] 415: 1-68.*

- MacDonald M., Maureen J., Heather L., Naylor M., Tschakovsky E., Hughson R.L. 2001. *Peripheral circulatory factors limit rate of increase in muscle O₂ uptake at onset of heavy exercise*. J. Appl. Physiol. 90: 83-89.
- Orizio C., Perini R., Comande A., Castellano M., Beschi M., Veicsteinas A. 1988. *Plasma catecholamines and heart rate at the beginning of muscular exercise in man*. Eur. J. Appl. Physiol. 57: 644-651.
- Ozyener F., Rossiter H.B., Ward S.A., Whipp B.J. 2001. *Influence of exercise intensity on the on- and off- transient kinetics of pulmonary oxygen uptake in humans*. J. Physiol., 533(3): 891-902.
- Pendergast D., Leibowitz R., Wilson D., Cerretelli P. 1983. *The effect of preceding anaerobic exercise on aerobic and anaerobic work*. Eur. J. Appl. Physiol. 52: 29-35.
- Perini R., Orizio C., Comande A., Castellano M., Beschi M., Veicsteinas A. 1989. *Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in men*. Eur. J. Appl. Physiol. 58: 879-883.
- Pierpoint M.D., Stolpman M.D., Gornick M.D. 2000. *Heart rate recovery post-exercise as an index of parasympathetic activity*. J. Auton. Nerv. Syst. 80: 169-174.
- Robinson B.F., Epstein S.E., Beiser G.D., Braunwald E. 1966. *Control of heart rate by the autonomic nervous system*. Circ. Res. 19: 400-411.
- Schneider D., Berwick J. 1998. *VE and VCO₂ remain tightly coupled during incremental cycling performed after a bout of high-intensity exercise*. Eur. J. Appl. Physiol. Occup. Physiol., 77(1-2): 72-76.
- Sietsema K.E., Daly J.A., Wasserman K. 1989. *Early dynamics of O₂ uptake and heart rate as affected by exercise work rate*. J. Appl. Physiol. 67(6): 2535-2541.
- Skinner J.S., McLellan H. 1980. *The transition from aerobic to anaerobic metabolism*. Res. Q. Exerc. Sport, 51(1): 234-248.
- Stasiulis A. 1997. *The relationship between heart rate recovery level and lactate threshold during intermittent exercise*. Pflügers. Archiv: Eur. J. Appl. Physiol., 433: 131.
- Tschakovsky M.E., Hughson R.L. 1999. *Interaction of factors determining oxygen uptake at the onset of exercise*. J. Appl. Physiol. 86(4): 1101-1113.
- Wallin B., Morlin C., Hjiemdahl P. 1987. *Muscle sympathetic activity and venous plasma noradrenaline concentrations during static exercise*

- in normotensive and hypertensive subjects. Asta. Physiol. Scand.* 129: 489-497.
- Whipp B.J., Ward S.A. 1990. *Physiological determinants of pulmonary gas exchange kinetics during exercise.* Med. Sci. Sports Exerc. 22: 62-71.
- Wasserman K., Whipp B.J., Koyal S.N., Beaver W.L. 1973. *Anaerobic threshold and respiratory gas exchange during exercise.* J. Appl. Physiol. 35: 236.
- Zavorsky G.S, Montgomery D.L, Pearsall D.J. 1998. *Effect of intense interval workouts on running economy using three recovery durations.* Eur. J. Appl. Physiol., 77: 224-230.