ORIGINAL ARTICLE

Lactic acidosis, potassium, and the heart rate deflection point in professional road cyclists

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Objective: To determine the influence of lactic acidosis, the Bohr effect, and exercise induced hyperkalaemia on the occurrence of the heart rate deflection point (HRDP) in elite (professional) cyclists.
Methods: Sixteen professional male road cyclists (mean (SD) age 26 (1) years) performed a ramp test on a cycle ergometer (workload increases of 5 W/12 s, averaging 25 W/min). Heart rate (HR), gas exchange parameters, and blood variables (lactate, pH, P_{50} of the oxyhaemoglobin dissociation curve, and K^+) were measured during the tests.
Results: A HRDP was shown in 56% of subjects at about 88% of their maximal HR (HRDP group; n = 9) but was linear in the rest (No-HRDP group; n = 7). In the HRDP group, the slope of the HR-workload regression line above the HRDP correlated inversely with levels of K^+ at the maximal power output ($r = -0.67$; $p<0.05$).
Conclusions: The HRDP phenomenon is associated, at least partly, with exercise induced hyperkalaemia.

The response of heart rate (HR) to incremental exercise is not always linear. Fifty years ago, Wahlund[1] reported that the rate of HR increase elicited by incremental exercise tends to be lower at higher workloads. Their pioneer finding was confirmed by Brooke and coworkers in the seventies[2 3] and by extensive research conducted over the last two decades.[4 5] The point after which the slope of the HR-workload relation decreases in some people is usually known in the literature as the heart rate deflection point (HRDP).[4] The HRDP is manifested as a curvilinear response in the HR-workload relation, and usually occurs at 88–94% of maximum HR.[4] Its occurrence has been documented in humans with a wide range of fitness levels—for example, highly trained subjects (including paraplegic athletes), sedentary healthy subjects, and patients with cardiac problems or cystic fibrosis.[4 5] Furthermore, it is not dependent on age, as it may occur in children, adolescents, middle aged men, and older people.[4 5]

Although considerable controversy exists in the literature,[4] the coincidence of the HRDP with the anaerobic threshold (AT), originally reported by Conconi et al[6] during a field test in runners, has been corroborated by further studies from Conconi's team[5] and other research groups.[4 5] Moreover, the so-called "Conconi test",[5] applicable for HRDP detection in field conditions, has become one of the most commonly used exercise tests in sports medicine. Because it is easy to repeat measurements, it is commonly used by elite athletes such as European professional cyclists to establish optimum training intensity.[7]

Despite some research efforts in the field, the physiological mechanisms involved in the occurrence of the HRDP remain to be fully elucidated. Conconi et al[5 6] proposed that it is caused by activation of the anaerobic lactic acid mechanisms of ATP production, irrespective of cardiocirculatory activity and HR. Metabolic acidosis occurring at high workloads could facilitate the release of oxygen from haemoglobin (the Bohr effect) and thus improve cardiocirculatory efficiency and attenuate the increase in HR.[5] To our knowledge, however, no investigation has been specifically designed to confirm Conconi's hypothesis. The fact that some descriptive studies report a coincidence between the exercise intensities at which both HRDP and AT occur[4 5] does not necessarily imply a causal relation between the two phenomena. Studies conducted with

both healthy subjects[8 9] and cardiac patients[10 11] suggest that the HR response to exercise—that is, occurrence of HRDP *v* linear response of HR or even upward increase at high workloads—is conditioned, at least partly, by myocardial function. That is, HRDP is likely to occur in subjects with greater myocardial function (expressed as left ventricular ejection fraction (LVEF)) whereas a linear HR response or an upward HR inflection may compensate for a lower LVEF at high workloads. Pokan et al[12] suggested that HR behaviour during incremental exercise ultimately depends on individual intrinsic HR regulation, namely parasympathetic drive. Surprisingly few data exist on the possible influence of exercise induced hyperkalaemia on the HRDP phenomenon. Hyperkalaemia is known to affect heart function (through a delay in AV conduction), and myocardial electrical conditions during intense exercise depend on the interaction between raised K^+, catecholamines, and lactic acidosis.[13] Interestingly, a preliminary report by Hofmann et al,[14] who use a ramp test, has shown that the degree of the deflection in the HR-workload curve after the HRDP is associated with an exercise induced increase in blood K^+, suggesting the possible involvement of K^+ in the HRDP phenomenon.

Few data are available on the cause of the HRDP in elite athletes. In a report from our laboratory, HR kinetics of professional cyclists at the high workloads of a ramp test were partly linked to their heart dimensions—that is, HRDP occurred mainly in riders with greater heart wall thickness.[7] The aim of the present study was to determine the influence of other possible factors (lactic acidosis and Bohr effect (Conconi's hypothesis) and exercise induced hyperkalaemia) on the occurrence of the HRDP in elite (professional) cyclists. We hypothesised that the HRDP and/or the degree of the deflection in the HR-workload relation at high workloads is determined, at least partly, by exercise induced hyperkalaemia.

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Abbreviations: HR, heart rate; HRDP, heart rate deflection point; AT, anaerobic threshold; LVEF, left ventricular ejection fraction; ECG, electrocardiogram; RCP, respiratory compensation point; LT, lactate threshold; OBLA, onset of blood lactate accumulation

MATERIALS AND METHODS

Subjects

Sixteen professional male road cyclists (mean (SD) age, height, and weight, 26 (1) years, 178.2 (1.3) cm, and 67.3 (1.4) kg respectively) were enrolled in the study. All provided written consent for participation in this investigation following the regulations of the Complutense University. The institutional research ethics committee (Complutense University) approved the study. The good health of each subject was confirmed by a normal physical examination (including electrocardiogram (ECG)) within the previous month. None was taking any banned drug which could artificially improve his performance, and no exogenous banned substances were detected in any of the subjects during routine anti-blood doping analysis. Each had at least two years of competition experience in the professional category and had covered an average of 30 000–40 000 km during the last season. Some of them are among the best road cyclists in the world—for example, a world champion and winners of three week tour races.

Study protocol

Each subject performed a ramp exercise test (see below), and, on the basis of the results, was assigned to one of two groups: those showing a deflection in their HR response (HRDP group), and those showing no such deflection (linear response or upward inflection; No-HRDP group).

Exercise test

Each subject was well rested before the test and had not performed hard physical work during the preceding 24 hours. All followed a similar high carbohydrate diet during the days preceding the test, and the last meal (breakfast, with a mean intake of about 150 g carbohydrate) was eaten two to three hours before the beginning of the testing session. Any drugs such as caffeine that could influence HR were avoided on the morning of the test. All tests were performed on a cycle ergometer (Ergometrics 900; Ergo-line, Barcelona, Spain) after a five minute warm up at 50 W followed by a two minute rest. Starting at 20 W, the workload was increased by 25 W/min (5 W/s). All the subjects had previous experience with this type of protocol, which has been used often for the physiological evaluation of professional cyclists,[7 15 16] including HRDP determination.[7]

Subjects adopted the conventional (upright) cycling posture during the duration of the test. This posture was characterised by a trunk inclination of about 75% and by the cyclists placing their hands on the handlebars with elbows slightly bent (flexion about 10%). They were allowed to choose their preferred cadence within the range 70–90 rpm. This simulates actual cycling conditions more closely than tests at fixed cadence: during three week tour races, the preferred pedalling cadence of professional riders ranges between 70 rpm (hill climbs) and 90 rpm (flat terrain or individual time trials).[15] A pedal frequency meter was used by the subjects to maintain this range of cadences. The test was terminated when pedalling cadence could not be maintained at 70 rpm (at least). Verbal encouragement was given to the subjects until the end of the test.

Measurements during the tests

Gas exchange data were collected continuously using an automated breath by breath system (Mvmax 29C; Sensormedics, Yorba Linda, California, USA). The workload (expressed as W and % of maximal oxygen uptake (VO_2MAX)) corresponding to the second ventilatory threshold or respiratory compensation point (RCP) was identified using the criteria of an increase in both ventilatory equivalents of oxygen (VE/VO_2) and carbon dioxide (VE/VCO_2) and end tidal partial pressure of oxygen ($PETO_2$) with no concomitant increase in end tidal partial pressure of carbon dioxide ($PETCO_2$).[16]

Blood samples (50 μl) for the measurement of blood lactate (YSI 1500; Yellow Springs Instruments, Yellow Springs, Ohio, USA) were taken from fingertips at rest, every two minutes during the test, and immediately after termination of exercise. The lactate threshold (LT) was determined by examining the lactate concentration-workload (W) relation during the tests as described by Weltman and coworkers.[17] This method defines the workload corresponding to LT as the highest workload not associated with a rise in lactate concentration above baseline. This always occurred just before the curvilinear increase in blood lactate observed at subsequent exercise intensities. An increase of at least 0.5 mM blood lactate concentration was required for the determination of the LT. The onset of blood lactate accumulation (OBLA) was defined as the workload corresponding to a blood lactate accumulation of 4.0 mM.[18]

A 21 gauge butterfly needle was inserted into the antecubital vein of each subject before the tests. Blood samples were collected at rest, every two minutes throughout the tests, and immediately after termination of exercise to calculate the following variables with an automated blood gas analyser (ABL725; Radiometer; Copenhagen, Denmark): haemoglobin concentration, packed cell volume, P_{50} of the oxyhaemoglobin dissociation curve, pH, and concentration of K^+. Following recommendations for the adequate interpretation of changes in biochemical variables (including electrolytes) during exercise,[19] venous plasma levels of K^+ obtained at the LT and at the maximal power output were corrected for relative changes in plasma volume—that is, exercise induced haemoconcentration—using haemoglobin concentrations and packed cell volumes as previously described.[20]

Determination of the HRDP

HR (beats/min) was continuously monitored during the tests using modified 12-lead ECG tracings (EK56; Hellige, Freiburg, Germany). The HR-workload relation was plotted and analysed between the LT and the power output at maximal HR using a computer algorithm linear regression model as recommended for investigational purposes.[4] The computer program that we used calculates the correlation coefficient (r), intercept of the y axis (y_0), and slope of the regression lines (a) for all possible divisions of data into two contiguous groups.[7] The two lines yielding the least pooled residual sum of squares is chosen as the best fit. When present, the point of change from the linear phase of the HR-workload relation to the curvilinear phase (the HRDP) was defined as that above which the values of a started to decrease.[5 7] Finally, an analysis of variance was performed to determine whether there was a significant difference ($p < 0.05$) in the total sum of squares between the two regression equations (before and after HRDP).[7]

Statistical analysis

Results are expressed as mean (SEM). Once the Kolmogorov-Smirnov test was applied to show a Gaussian distribution of the results, Student's t test for unpaired data was performed to compare the mean values of the following variables between the HRDP and No-HRDP groups: VO_2MAX; maximal power output (W); LT, OBLA, and RCP (each expressed in both W and $\%VO_2MAX$); blood lactate, pH, P_{50}, and K^+ at the maximal power output. A Student's t test for paired data was applied within the HRDP group to compare the mean exercise intensity (expressed in both W and $\%VO_2MAX$) eliciting the HRDP and that eliciting LT, OBLA, and RCP. Finally, Pearson product-moment correlation coefficients were also calculated within the HRDP group to determine whether there was a significant relation between the slope of the HR regression line above the HRDP and the following variables: (a) blood lactate, pH, P_{50}, and K^+ at the maximal power output; (b) the changes in blood lactate, pH, P_{50}, and K^+ between the LT and the maximal power output.

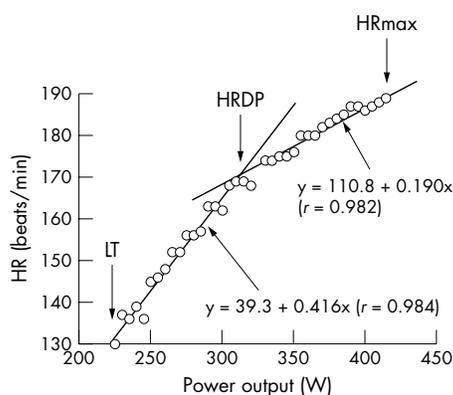Significance was set at $p < 0.05$ for all statistical analyses.

Figure 1 Example of heart rate (HR) kinetics (from the workload (W) associated with the lactate threshold (LT) to that eliciting the maximal HR (HRmax)) in one subject showing a curvilinear response (HRDP group). HRDP, heart rate deflection point.
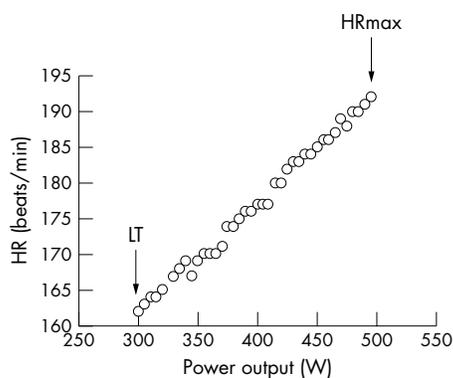

Figure 2 Example of heart rate (HR) kinetics (from the workload (W) associated with the lactate threshold (LT) to that eliciting the maximal HR (HRmax)) in one subject showing a linear response (No-HRDP group).

Table 1 Comparison between cyclists showing a heart rate deflection point (HRDP) and those who did not

	HRDP (n=9)	No HRDP (n=7)
Age (years)	25 (1)	26 (1)
HRDP (beats/min)	165 (4)	–
HRDP (W)	350 (15)	–
HRDP (%VO$_2$MAX)	85.2 (3.0)	–
Variables at the maximal power output		
W$_{MAX}$	469 (10)	475 (24)
VO$_2$MAX (ml/kg/min)	72.7 (1.2)	71.4 (1.3)
HR (beats/min)	188 (2)	192 (3)
BLaMAX (mM)	9.0 (0.5)	9.0 (0.5)
pH	7.18 (0.02)	7.20 (0.03)
P$_{50}$ (mm Hg)	33.4 (0.7)	32.1 (0.7)
K$^+$ (mM)	5.2 (0.1)	5.0 (0.2)
LT, OBLA and RCP		
LT (W)	283 (11)*	279 (13)
LT (%VO$_2$MAX)	70.6 (2.9)†	68.6 (2.5)
OBLA (W)	367 (12)	381 (23)
OBLA (%VO$_2$MAX)	87.9 (2.0)	86.9 (1.8)
RCP (W)	389 (14)	389 (22)
RCP (%VO$_2$MAX)	91.6 (2.0)	88.2 (1.6)

Values are expressed as mean (SEM).
*Significantly different (p<0.01) from No HRDP group (W);
†significantly different (p<0.01) from No HRDP group (%VO$_2$MAX).
VO$_2$MAX, Maximal oxygen consumption; W$_{MAX}$, maximal power output (in W); HR$_{MAX}$, maximal heart rate; BLaMAX, blood lactate at the maximal power output; LT, lactate threshold; OBLA, onset of blood lactate accumulation; RCP, respiratory compensation point; P$_{50}$, P$_{50}$ of the oxyhaemoglobin dissociation curve.

RESULTS

The HR response showed a deflection point in 56% of subjects (HRDP group; n = 9) but was linear in 44% (No-HRDP group; n = 7). Figures 1 and 2 show an example of a curvilinear (HRDP) and a linear response respectively.

Plasma loss at the end of the tests averaged −14.5 (0.9)%, and mean haemoglobin concentration and packed cell volume before exercise were 144 (2) g/l and 44.4 (0.8)% respectively. No significant differences were found between groups in any of the variables measured during the tests, including blood pH at maximal power output (table 1). In the HRDP group, the HRDP occurred at about 88% of maximal HR. No significant difference was found, on the other hand, between the workload (W or %VO$_2$MAX) eliciting the HRDP and that associated with the OBLA or RCP (p>0.05). In contrast, the workload at the HRDP (W or %VO$_2$MAX) was significantly higher than that eliciting the LT (p<0.01).

In the HRDP group, the slope of the HR-workload regression line above the HRDP correlated inversely with: (*a*) levels of K$^+$ at the maximal power output ($r = −0.67$; p<0.05) (fig 3); (*b*) the change in K$^+$ levels between the LT and the maximal power output ($r = −0.47$; p= 0.06).

DISCUSSION

The HR response of top level professional cyclists showed a HRDP (at about 88% of maximum HR) in about 56% of the subjects and was linear in the rest. The main finding of our study was that, when occurring at high workloads, this curvilinear response of HR seems to be associated, at least
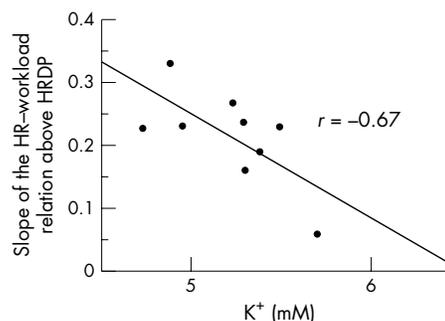

Figure 3 Correlation between the slope of the deflection of the heart rate (HR)-workload (W) relation above the heart rate deflection point (HRDP).

partly, with blood levels of K$^+$. In contrast, lactic acidosis and its subsequent effect on the oxyhaemoglobin dissociation curve (through the Bohr effect) seems to play a minor role, as discussed below. Although there is considerable controversy surrounding this area of research,[4] the occurrence of HRDP in 56% of the subjects is in agreement with several studies that report that a good number of young, healthy subjects may exhibit a curvilinear HR response to incremental exercise with a lower rate of increase of HR during highly intense exercise.[4 5 7] Following the recommendations of Conconi *et al*[5] for HRDP determination, we selected a ramp protocol (mean increases of 25 W/min) for this investigation. According to Conconi's team, the fact that some authors have previously failed to detect the HRDP may be explained by the protocol used—that is, step-like workload increases rather than the more gradual ramp method. Ramp protocols such as the present, which allow gradual increases in HR (<8 beats/min per minute of exercise), may be more appropriate for the detection of HRDP in subjects showing a curvilinear HR response.[5]

To date, few studies have analysed the physiological mechanisms involved in the HRDP. Conconi *et al*[6] were the first to

provide an explanatory hypothesis for the phenomenon. On the basis of the reported coincidence between the workloads at which both the AT and the HRDP occur, they proposed that the deflection is caused by activation of the anaerobic lactic acid mechanisms of ATP production irrespective of cardiocirculatory activity and HR. Metabolic acidosis occurring at high workloads could indeed facilitate the release of oxygen from haemoglobin (the Bohr effect) and thus improve cardiocirculatory efficiency and attenuate the increase in HR.[5] However, no investigation has been specifically designed to confirm Conconi's original hypothesis. The fact that the exercise intensity at which HRDP occurs and that eliciting AT may coincide[4 5 7] does not necessarily imply a causal relation between the phenomena, as suggested by our findings. In agreement with a study with professional cyclists,[7] HRDP occurred in our subjects at about 85% V_{O_2MAX}, or before the exercise intensity (about 70% V_{O_2MAX}) corresponding to the LT, in which a first breakpoint in blood lactate and ventilation (the ventilatory threshold) is evident and above which anaerobic metabolism is partly involved. In contrast, HRDP was detected at a comparable workload (85–90% V_{O_2MAX}) to that eliciting the AT—that is, the second breakpoint in lactate (OBLA) and ventilation (second ventilatory threshold or RCP). Above the AT (the so-called Phase III), lactate production exceeds clearance, with subsequent blood lactate accumulation and ventilatory compensation.[16] No significant correlation was observed between HRDP and OBLA or RCP. Moreover, we found no correlation between pH, blood lactate, or P_{50} of the oxyhaemoglobin dissociation curve and the degree of HR deflection. Thus our results do not support the idea that lactic acidosis occurring at high workloads is involved in the HRDP phenomenon—that is, through facilitation of oxygen release from haemoglobin—at least in elite endurance athletes. Other physiological mechanisms do not support a possible association between lactic acidosis at high workloads and the deflection in HR rise. Firstly, it must be kept in mind that the increased recruitment of less efficient type II fibres (particularly the type IIX subtype) that occurs in Phase III[21 22] may partly compensate for any hypothetical improvement in cardiocirculatory efficiency mediated by the Bohr effect. Secondly, exercise induced acidosis can decrease myocardial contractility by intracellular acidification, which in turn impairs the Ca^{2+} release and reuptake from the sarcoplasmic reticulum.[13 23] Thus, one would expect a further increase in the catecholamine outflow (and thus in HR) to occur in an attempt to maintain cardiac output, especially in highly trained athletes, as the present ones in which the oxygen demands of working muscles are likely to be exceptionally high. Previous animal studies have indeed shown that the negative cardiac effects of acidosis can be ameliorated by raised extracellular concentrations of catecholamines or by direct stimulation of cardiac sympathetic nerves.[13] Future research protocols may determine whether there is a cause-effect relation between the involvement of anaerobic metabolism and the occurrence of HRDP in subjects of lower fitness levels.

An inverse correlation was found between the slope of the HR-workload curve above the HRDP and both maximal levels of K^+ ($r = -0.67$; $p<0.05$) and the change in K^+ between the LT and the maximal power output ($r = -0.47$; $p = 0.06$). Our results are not surprising when one considers the physiological role of K^+ during exercise (see below); they suggest a certain involvement of exercise induced hyperkalaemia in the HRDP phenomenon, or at least in the degree of the HR deflection in those subjects showing an HRDP. It has been well documented that stimulation of skeletal muscle cells causes the efflux of K^+, which results in hyperkalaemia.[13] During the low to moderate workloads (below about 60% V_{O_2MAX}) of an incremental exercise, K^+ increases proportionally to V_{O_2}; thereafter a disproportionate increase is usually reported.[13 24] The loss of K^+ from working muscles occurs mainly through

Take home message

The response of heart rate to incremental (ramp) exercise is curvilinear in a good number of professional cyclists. This response is not associated with lactic acidosis, but blood K^+ released from working muscles may play a role.

the delayed rectifier K^+ channel during the repolarisation phase of skeletal muscle action potential,[13 25] and incomplete reuptake of this ion by the Na^+ pump results in a rise in extracellular K^+.[26] From the Nernst equation, a considerable rise in extracellular K^+, as often observed in maximal exercise, can reduce the potential of all excitable cells.[13] In the heart, hyperkalaemia can decrease upstroke velocity and shortens the plateau phase of the ventricular action potential.[27] Induced hyperkalaemia in venous blood can alter the HR response—that is, delaying AV conduction[28] or inducing ECG changes such as widening of the QRS complex or a greater amplitude of the T wave.[29] In fact, hyperkalaemia, metabolic acidosis, and increased catecholamine levels interplay during intense exercise (*a*) to compensate for the harmful effects that each of them separately is known to have in the resting heart and (*b*) to maintain cardiac function. For instance, K^+ plays an important role in maintaining myocardial stability during high sympathetic tone.[13] Although we did not evaluate catecholamine activity and thus cannot evaluate the relative contribution of the three factors to the HR deflection phenomenon, we speculate that K^+ plays a relevant role (more than acidosis, for example).

Whichever the case, the shape of the HR response curve (linear *v* curvilinear) does not seem to affect performance in elite endurance cyclists, as indicative variables of road cycling performance such as V_{O_2MAX} or the workload eliciting the RCP[16] were similar in both HRDP and No-HRDP groups. Taken together, the findings of this study and those of previous research suggest that, when it does occur, the HRDP appears to be a multifactorial phenomenon in which each of the following factors may play a certain role in humans with a wide range of fitness levels: myocardial function (expressed as LVEF),[8–11] individual variations in autonomic HR regulation,[12] heart dimensions (thickness of both left ventricular posterior and interventricular walls),[7] and blood K^+.[14] Future research may determine the relative contribution of each of the proposed factors.

In agreement with recent research,[7] HR may show a curvilinear response during incremental (ramp) exercise in a considerable number of highly trained endurance athletes at about 88% of their maximum heart rate, or about 85% of V_{O_2MAX}. Although further research is needed, it may be suggested that increased blood K^+ released from working muscles is involved in the phenomenon. Future nondescriptive protocols—for example, using pharmacological manipulation to alter blood pH or K^+ levels—may confirm the exact mechanisms responsible for the occurrence of the HRDP phenomenon in elite endurance athletes.

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REFERENCES

1 **Wahlund H**. Determination of the physical working capacity. *Acta Med Scand* 1948;**215**(suppl):1–78.
2 **Brooke JD**, Hamley EJ. The heart-rate-physical work curve analysis for the prediction of exhausting work ability. *Med Sci Sports Exerc* 1972;**4**:23–6.
3 **Brooke JD**, Hamley EJ, Thomson H. The relationship of heart rate to physical work. *J Physiol (Lond)* 1968;**167**:61–3.
4 **Bodner ME**, Rhodes EC. A review of the concept of the heart rate deflection point *Sports Med* 2000;**30**:31–46.
5 **Conconi F**, Grazzi G, Casoni I, *et al.* The Conconi test: methodology after 12 years of application. *Int J Sports Med* 1996;**17**:509–19.
6 **Conconi F**, Ferrari M, Ziglio PG, *et al.* Determination of the anaerobic threshold by a noninvasive field test in runners. *J Appl Physiol* 1982;**152**:869–73.
7 **Lucía A**, Carvajal A, Boraita A, *et al.* Heart dimensions may influence the occurrence of the heart rate deflection point in highly trained cyclists. *Br J Sports Med* 1999; **33**:387–92.
8 **Hofmann P**, Pokan R, Preidler K, *et al.* Relationship between heart rate threshold, lactate turn point and myocardial function. *Int J Sports Med* 1994;**15**:232–7.
9 **Pokan R**, Hofmann P, Preidler K, *et al.* Correlation between inflection of heart/rate work performance curve and myocardial function in exhausting cycle ergometer exercise. *Eur J Appl Physiol* 1993; **67**:385–8.
10 **Foster C**, Spatz P, Georgakopoulos N. Left ventricular function in relation to the heart rate performance curve. *Clin Exer Physiol* 1999;**1**:29–32.
11 **Pokan R**, Hofmann P, Von Duvillard SP, *et al.* The heart rate performance curve and left ventricular function during exercise in patients after myocardial infarction. *Med Sci Sports Exerc* 1998;**30**:1475–80.
12 **Pokan R**, Hofmann P, Von Duvillard SP. Parasympathetic receptor blockade and the heart rate performance curve. *Med Sci Sports Exerc* 1998;**30**:229–33.
13 **Paterson DJ**. Role of potassium in the regulation of systemic physiological function during exercise. *Acta Physiol Scand* 1996;**156**:287–94.
14 **Hofmann P**, Seibert F-J, Pokan R, *et al.* Relationship between blood pH, potassium, and the heart rate performance curve [Abstract]. *Med Sci Sports Exerc* 1999;**31**(suppl):628.
15 **Lucia A**, Hoyos J, Chicharro JL. Preferred pedalling cadence in professional cycling. *Med Sci Sports Exerc* 2001;**33**:1361–6.
16 **Lucia A**, Hoyos J, Pérez M, Chicharro JL. Heart rate and performance parameters in elite cyclists: a longitudinal study. *Med Sci Sports Exerc* 2000;**32**:1777–82.
17 **Weltman A**, Snead D, Stein P, *et al.* Reliability and validity of a continuous incremental treadmill protocol for the determination of lactate threshold, fixed blood lactate concentrations, and VO_2max. *Int J Sports Med* 1990;**11**:26–32.
18 **Heck H**, Mader A, Hess G, *et al.* Justification of the 4-mmol/l lactate threshold. *Int J Sports Med* 1985;**6**:117–30.
19 **Kargotich S**, Goodman C, Keast D, *et al.* The influence of exercise-induced plasma volume changes on the interpretation of biochemical parameters used for monitoring exercise, training and sport. *Sports Med* 1998;**26**:101–17.
20 **Dill DB**, Costill DL. Calculation of percentage change in volumes of blood, plasma and red cells in rehydration. *J Appl Physiol* 1979;**37**:247–8.
21 **Crow MT**, Kushmerick MJ. Chemical energetics of slow- and fast-twitch muscles of the mouse. *J Gen Physiol* 1982;**79**:147–66.
22 **Jones AM**, Carter H, Doust JH. A disproportionate increase in VO_2 coincident with lactate threshold during treadmill exercise. *Med Sci Sports Exerc* 1999;**31**:1299–306.
23 **Fabiato A**, Fabiato F. Effects of pH on the myofilaments and the sarcoplasmic reticulum of the skinned cells from cardiac and skeletal muscles. *J Physiol (Lond)* 1978; **276**:233–55.
24 **Vollestad NK**, Hallen J, Sejersted OM. Effect of exercise intensity on potassium balance in muscle and blood of man. *J Physiol (Lond)* 1994;**475**:359–68.
25 **Kolb H**. Potassium channels in excitable and non-excitable cells. *Rev Physiol Biochem Pharmacol* 1995;**115**:51–91.
26 **Clausen T**, Nielsen OB. The Na-K pump and muscle contractility. *Acta Physiol. Scand* 1994;**152**:365–73.
27 **Patterson DJ**, Rogers J, Powell T. Effect of catecholamines on the ventricular myocyte action potential in raised extracellular potassium. *Acta Physiol Scand* 1993;**148**:177–86.
28 **Ellinger PO**, Regan TJ, Oldewartel HA. Hyperkalaemia, cardiac conduction, and the electrocardiogram: a review. *Am Heart J* 1974;**88**:360–71.
29 **Winkler AW**, Hoff HE, Smith PK. Electrocardiographic changes and concentrations of potassium in serum following intravenous injection of potassium chloride. *Am J Physiol* 1938;**124**:478–83.

Note to readers

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