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, P50 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 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 and by extensive research conducted over the last two decades. 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). The HRDP is manifested as a curvilinear response in the HR-workload relation, and usually occurs at 88–94% of maximum HR. 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. Furthermore, the HRDP does not depend on age, as it may occur in children, adolescents, middle aged men, and older people.

Although considerable controversy exists in the literature, the coincidence of the HRDP with the anaerobic threshold (AT), originally reported by Conconi et al during a field test in runners, has been corroborated by further studies from Conconi’s team and other research groups. Moreover, the so-called “Conconi test”, 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.

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 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. 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 does not necessarily imply a causal relation between the two phenomena. Studies conducted with both healthy subjects and cardiac patients 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 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. Interestingly, a preliminary report by Hofmann et al 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. 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.

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-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, including HRDP determination.

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 pedal frequency meter was used by the subjects to maintain their preferred cadence at the range 70–90 rpm. This simulates actual cycling conditions more closely than tests at fixed 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 (VO2 MAX) 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/VO2) and carbon dioxide (Ve/VO2) and end tidal partial pressure of oxygen (PETO2) with no concomitant increase in end tidal partial pressure of carbon dioxide (PETCO2). 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. 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. 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, Po2 of the oxyhaemoglobin dissociation curve, pH, and concentration of K+.

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. The computer program that we used calculates the correlation coefficient (r), intercept of the y axis (γy), and slope of the regression lines (α) for all possible divisions of data into two contiguous groups. 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 α started to decrease. 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).

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 paired data was performed to compare the mean values of the following variables between the HRDP and No-HRDP groups: VO2 MAX; maximal power output (W); LT, OBLA, and RCP (each expressed in both W and % VO2 MAX); blood lactate, pH, Po2, and K+ at the maximal power output. A Student’s t test for paired data was performed within the HRDP group to compare the mean exercise intensity (expressed in both W and % VO2 MAX) 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, Po2, and K+ at the maximal power output; (b) the changes in blood lactate, pH, Po2, and K+ between the LT and the maximal power output.

Significance was set at p<0.05 for all statistical analyses.
Heart rate deflection point in cyclists

**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

<table>
<thead>
<tr>
<th>Variable</th>
<th>HRDP (n=9)</th>
<th>No HRDP (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25 (1)</td>
<td>26 (1)</td>
</tr>
<tr>
<td>HRDP (beats/min)</td>
<td>165 (4)</td>
<td>–</td>
</tr>
<tr>
<td>HRDP (W)</td>
<td>350 (15)</td>
<td>–</td>
</tr>
<tr>
<td>HRDP (%VO2MAX)</td>
<td>85.2 (3.0)</td>
<td>–</td>
</tr>
<tr>
<td>Variables at the maximal power output</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wmax</td>
<td>469 (10)</td>
<td>475 (24)</td>
</tr>
<tr>
<td>VO2MAX (ml/kg/min)</td>
<td>72.7 (1.2)</td>
<td>71.4 (1.3)</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>188 (2)</td>
<td>192 (3)</td>
</tr>
<tr>
<td>BlaMAX (mM)</td>
<td>9.0 (0.5)</td>
<td>9.0 (0.5)</td>
</tr>
<tr>
<td>pH</td>
<td>7.18 (0.02)</td>
<td>7.20 (0.03)</td>
</tr>
<tr>
<td>P50 (mm Hg)</td>
<td>33.4 (0.7)</td>
<td>32.1 (0.7)</td>
</tr>
<tr>
<td>K+ (mM)</td>
<td>5.2 (0.1)</td>
<td>5.0 (0.2)</td>
</tr>
<tr>
<td>LT, OBLA and RCP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LT (W)</td>
<td>283 (11)*</td>
<td>279 (13)</td>
</tr>
<tr>
<td>LT (%VO2MAX)</td>
<td>70.6 (2.9)*</td>
<td>68.6 (2.5)</td>
</tr>
<tr>
<td>OBLA (W)</td>
<td>367 (12)</td>
<td>381 (23)</td>
</tr>
<tr>
<td>OBLA (%VO2MAX)</td>
<td>87.9 (2.0)</td>
<td>86.9 (1.8)</td>
</tr>
<tr>
<td>RCP (W)</td>
<td>389 (14)</td>
<td>389 (22)</td>
</tr>
<tr>
<td>RCP (%VO2MAX)</td>
<td>91.6 (2.0)</td>
<td>88.2 (1.6)</td>
</tr>
</tbody>
</table>

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 (%VO2MAX).

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

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 %VO2MAX) eliciting the HRDP and that associated with the OBLA or RCP (p>0.05). In contrast, the workload at the HRDP (W or %VO2MAX) 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 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, 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.1,7

Following the recommendations of Conconi et al10 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.5,12

To date, few studies have analysed the physiological mechanisms involved in the HRDP. Conconi et al10 were the first to:

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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 cardiovascular 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. 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 does not necessarily imply a causal relation between the phenomena, as suggested by our findings. Agreement with a study with professional cyclists, HRDP occurred in our subjects at about 85% VO2MAX, or before the exercise intensity (about 70% VO2MAX) 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%VO2MAX) 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. No significant correlation was observed between HRDP and OBLA or RCP. Moreover, we found no correlation between pH, blood lactate, or P0, 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 may partly compensate for any hypothetical improvement in cardiocirculatory efficiency mediated by the Bohr effect. Secondly, exercise induced acidosis may decrease myocardial contractility by intracellular acidification, which in turn impairs the Ca2+ release and reuptake from the sarcoplasmic reticulum. 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. 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.

In agreement with previous research, 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 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.

**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.

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REFERENCES

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