Heart dimensions may influence the occurrence of the heart rate deflection point in highly trained cyclists

Alejandro Lucía, Alfredo Carvajal, Araceli Boraita, Luis Serratosa, Jesús Hoyos, José L Chicharro

Abstract

Objectives—To determine whether the heart rate (HR) response to exercise in 21 highly trained cyclists (mean (SD) age 25 (3) years) was related to their heart dimensions.

Methods—Before performing an incremental exercise test involving a ramp protocol with workload increases of 25 W/min, each subject underwent echocardiographic evaluation of the following variables: left ventricular end diastolic internal diameter (LVIDd), left ventricular posterior wall thickness at end diastole (LPWTd), interventricular septal wall thickness at end diastole (IVSTD), left ventricular mass index (LVMi), left atrial dimension (LAD), longitudinal left atrial (LADD) and right atrial (RAD) dimensions, and the ratio of early to late (E/A) diastolic flow velocity.

Results—The HR response showed a deflection point (HRd) at about 85% VO₂MAX in 66.7% of subjects (D group; n = 14) and was linear in 33.3% (NoD group; n = 7). Several echocardiographic variables (LVMi, LAD, LADD, RAD) indicative of heart dimensions were similar in each group. However, mean LPWTd (p<0.01) and IVSTD (p<0.05) values were significantly higher in the D group. Finally, no significant difference between groups was found with respect to the E/A.

Conclusions—The HR response is curvilinear during incremental exercise in a considerable number of highly trained endurance athletes—that is, top level cyclists. The departure of HR increase from linearity may predominantly occur in athletes with thicker heart walls.

Keywords: heart rate; lactate threshold; ventilatory threshold; onset of blood lactate accumulation (OBLA); cycling; echocardiography

The adaptation of the cardiocirculatory system to exercise can be simply determined by evaluating the kinetics of heart rate (HR). In practice, two basic protocols are commonly used: constant exercise intensity and graded incremental exercise. Fifty years ago, Wahlund reported that the rate of HR increase elicited by incremental exercise tends to be lower at higher workloads. In agreement with this, Conconi and co-workers suggested that the HR/exercise intensity relation was linear from low to submaximal workloads during an incremental running test, but curvilinear after a certain submaximal workload was reached. In addition, the point at which HR departs from linearity coincided with the anaerobic threshold (AT). In subsequent years, several studies conducted by Conconi et al. and by other authors confirmed both the existence of a deflection point in the HR response to an incremental protocol and its coincidence with the AT in adult sportsmen and women (runners, cyclists, swimmers, cross country skiers, canoeists, etc) of different fitness levels. Indeed, the so called “Conconi test”, applicable in field conditions, has become one of the most commonly used exercise tests in sports medicine. Because of the ease of repeating measurements, it is often used by elite athletes such as European professional cyclists to establish optimum training intensity.

Although it was originally designed for athletes, the Conconi test may be also used as an alternative method to determine the AT in untrained subjects or children. With regard to the latter, Ballarin et al. suggested its potential applicability in cross sectional and longitudinal studies on changes in aerobic power during growth. In contrast, possibly because of limitations in systolic function above the AT, the HR response of the elderly to incremental exercise is highly variable. Although existing relevant data are scarce, it may be worth assessing the applicability of the Conconi test in the clinical setting. Traditional methods of AT determination, involving the measurement of blood lactate or gas exchange parameters in patients with chronic obstructive pulmonary disease, have proved inadequate for the identification of the exercise intensity at which metabolic acidosis occurs. However, a recent report showed the occurrence of a HR deflection point using Conconi’s technique in only 17 of 32 (53%) patients with cystic fibro-
sis who had mild to moderate pulmonary disease. Moreover, when identified, HR deflection tended to overestimate AT. Schmid et al recently reported that HR deflection also occurred in endurance trained paraplegics during arm cranking exercise, although no causal relation was found to the AT. Indeed, the medical sports literature reflects controversy with respect to the Conconi test, and several investigators report that the downward deflection in the HR response curve only occurs in certain subjects and if detected does not necessarily coincide with the AT.

Despite this controversy, surprisingly few research efforts have focused on the physiological mechanisms involved in the HR deflection likely to occur in some subjects during incremental tests. Conconi et al proposed that deflection is caused by activation of the anaerobic lactacid 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 so called Bohr effect) and thus improve cardiocirculatory efficiency and attenuate the HR increase. In this regard, it has been recently shown that stroke volume may show a linear increase up to maximal intensities in trained endurance athletes.

To our knowledge, however, no investigation has been specifically designed to confirm Conconi’s hypothesis. The fact that some descriptive studies report that the exercise intensity at which HR deflection occurs corresponds to that eliciting AT does not necessarily imply a causal relation between the phenomena.

Most recently, Pokan et al suggested that HR behaviour during incremental exercise is a reproducible individual response which ultimately depends on individual intrinsic HR regulation rather than sympathetic/parasympathetic activity. These authors further hypothesise that subjects who show a deflection point in the HR response to incremental exercise are those with greater heart volumes measured at rest—that is, well trained endurance subjects. From this perspective, HR deflection may represent an effort to favour diastolic filling and thus improve systolic function during high intensity exercise (above the AT).

The aim of this study was to determine the possible influence of heart dimensions—that is, volumes and muscle thickness—at rest on the HR response of highly trained cyclists during an incremental (ramp) exercise protocol. In agreement with Pokan’s original hypothesis, it is proposed that, if detected, the deflection in the HR-workload curve may be associated with larger heart volumes.

**Materials and methods**

**SUBJECTS**

Twenty one professional male road cyclists were enrolled in the study. All provided written consent to participate in this investigation following the regulations of the Complutense University. Subjects’ mean (SD) age, height, and body mass were as follows: 25 (3) years, 179.2 (5.2) cm, and 71.5 (5.5) kg. The good health of each subject was ensured by a normal physical examination (including electrocardiogram (ECG)) within the previous month. Each had competition experience of 4 (2) years in the professional category and had covered 32 000 (4000) km (including training and competitions) during the last season. The most outstanding awards obtained by the subjects in professional races were: first position in the 1995 and 1998 World Championships (road race and time trial respectively); second in the 1995 time trial World Championships; second in the 1996 Olympic Games (time trial); first and third in the 1998 “Tour of Spain”; third in the 1996 “Giro d’Italia”; fourth and eighth in the 1997 “Tour de France”.

**STUDY PROTOCOL**

Each subject performed a ramp exercise test (see below) and underwent an echocardiographic examination at the same time of year (November-December). Based on the results of the exercise test, each subject was assigned to one of two groups: subjects showing a deflection in their HR response (D group), and those showing no such deflection (linear response) (NoD group).

**Exercise test**

Subjects performed an exercise test on a bicycle ergometer (Ergometrics 900; Ergo-line, Barcelona, Spain) following a ramp protocol until exhaustion at the same time of the day (1400–1700 hours). Starting at 0 W, the workload was increased by 25 W/min (5 W/12 s) and pedalling cadence was kept constant at 70–90 rpm. The test was terminated when the established criteria for test termination were met. Tests were performed under similar environmental conditions (21–24°C; 45–55% relative humidity). Gas exchange data were monitored breath by breath (CPX; Medical Graphics, St Paul, Minnesota, USA). The measuring instruments were calibrated before each test and the necessary environmental adjustments were made. The ventilatory equivalents for oxygen (VE/VO2) and carbon dioxide (VE/VO2) were recorded during each test. The first ventilatory threshold (VT1) was determined using the criterion of an increase in VE/O2 and no increase in VE/CO2, and the second ventilatory threshold (VT2) was determined by use of the criterion of an increase in both VE/O2 and VE/CO2. Two independent observers detected VT1 and VT2. If there was disagreement, the opinion of a third investigator was sought.

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 by the methodology described by...
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Weltman and co-workers. 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 0.2 mM blood lactate concentration was required for the determination of the LT. On the other hand, the onset of blood lactate accumulation (OBLA) was defined as the workload corresponding to a blood lactate concentration of 4.0 mM.

**Determination of the point of HR deflection**

HR (beats/min) was continuously monitored during the tests using modified 12 lead ECG tracings (EK56; Hellige, Freiburg, Germany). The HR/workload (W) relation was plotted and analysed using a computer algorithm linear regression model. This program calculates the correlation coefficient (r), intercept of the y axis (a) and slope (b) of the regression lines 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/W relation to the curvilinear phase (the so called point of HR deflection or HRd) was defined as that above which the values of b 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 HRd).

**Echocardiographic study**

Transthoracic M mode, two dimensional, and Doppler echocardiographic examinations were performed using a Toshiba SSH-140A (Toshiba Medical Systems, Madrid, Spain) instrument equipped with a 2.5 MHz transducer. Athletes were positioned in the left lateral position (45°) with the head slightly tilted. Image location and gain settings were adjusted to yield optimal definition of endocardial and epicardial limits. An ECG tracing was simultaneously displayed on the screen. All examinations were recorded on a Panasonic AG-7330 videocassette recorder for subsequent analysis of three to five frozen high quality frames. All measurements were performed by the same experienced investigator who ensured that the subject and transducer positions were similar in each evaluation.

Two dimensionally guided M mode echocardiograms were obtained from the left parasternal long axis view. The following variables were measured according to the recommendations of the American Society of Echocardiography (ASE): left ventricular end diastolic internal diameter (LVIDd), left ventricular posterior wall thickness at end diastole (LVPWTd), interventricular septal wall thickness at end diastole (IVSTd), and left atrial dimension (LAD). Left ventricular mass was calculated using Devereux’s formula: LV mass = 0.80 (ASE – cube LV mass) + 0.6 g. Left ventricular mass index (LVMI) was calculated with correction for body surface area. Left atrial (LLAD) and right atrial (LRAD) longitudinal dimensions were also measured from the two dimensional images obtained from the apical four chamber view at end systole. Finally, two dimensionally guided pulse Doppler recordings of left ventricular inflow in early (E) and late (A) diastole, and the ratio of early to late (E/A) diastolic flow velocity were calculated.

**Statistical analysis**

Results are expressed as mean (SD). Once the Kolmogorov-Smirnov test was applied to demonstrate a Gaussian distribution of the results, a Student’s t test for unpaired data was performed to compare the mean values of both the exercise test variables (VO2MAX, VT1, etc.) and echocardiographic variables obtained for the D and NoD groups. Confidence limits for the mean differences between the two groups are also reported. A one way repeated measures analysis of variance was applied within the D group to compare the exercise intensity (%VO2MAX) eliciting HRd and that eliciting VT1, VT2, LT, and OBLA. Pearson product

### Table 1 Demographic characteristics (age) and incremental exercise test results

<table>
<thead>
<tr>
<th></th>
<th>D (n=14)</th>
<th>NoD (n=7)</th>
<th>Confidence limits for difference between group mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25 (3)</td>
<td>24 (3)</td>
<td>2.2; 3.9</td>
</tr>
<tr>
<td>VO2MAX (ml/kg/min)</td>
<td>70.0 (2.0)</td>
<td>71.1 (6.7)</td>
<td>4.3; 3.1</td>
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<tr>
<td>Wmax (W)</td>
<td>508.2 (34.0)</td>
<td>492.9 (13.8)</td>
<td>12.9; 43.6</td>
</tr>
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<td>HRd (beats/min)</td>
<td>199 (10)</td>
<td>192 (7)</td>
<td>4.7; 13.2</td>
</tr>
<tr>
<td>V T1 (%VO2MAX)</td>
<td>69.8 (5.6)</td>
<td>73.2 (2.8)</td>
<td>8.3; 1.3</td>
</tr>
<tr>
<td>V T2 (%VO2MAX)</td>
<td>87.7 (4.0)</td>
<td>88.2 (3.6)</td>
<td>3.3; 2.3</td>
</tr>
<tr>
<td>LT (%VO2MAX)</td>
<td>68.3 (8.0)</td>
<td>72.0 (2.7)</td>
<td>2.9; 2.1</td>
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<tr>
<td>OBLA (%&lt;VO2MAX)</td>
<td>88.5 (4.9)</td>
<td>91.5 (3.8)</td>
<td>6.0; 3.2</td>
</tr>
<tr>
<td>HRd (%VO2MAX)</td>
<td>176 (10)</td>
<td>85.3 (8.3)</td>
<td>2.4; 2.1</td>
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<tr>
<td>HRd (W)</td>
<td>402.6 (48.4)</td>
<td></td>
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</table>

All values are expressed as means (SD). **Significantly different (p<0.01) from both VT1 and LT.** Abbreviations: VO2MAX, maximal oxygen consumption; Wmax, maximal power output (w); HRmax, maximal heart rate; VT1, first ventilatory threshold; VT2, second ventilatory threshold; LT, lactate threshold; OBLA, onset of blood lactate accumulation; HRd, heart rate deflection point.

![Figure 1 Determination of heart rate deflection point](http://bjsm.bmj.com/33.6.387)
Table 2  Echocardiographic variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>D (n=14)</th>
<th>NoD (n=7)</th>
<th>Confidence limits for difference between group means</th>
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<tr>
<td>LVIDd (mm)</td>
<td>60.9 (2.9)</td>
<td>61.9 (2.6)</td>
<td>-3.8; 1.7</td>
</tr>
<tr>
<td>LVPWTd (mm)</td>
<td>9.0 (0.6)</td>
<td>8.4 (0.6)*</td>
<td>0.1; 1.2</td>
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<tr>
<td>IVSTd (mm)</td>
<td>9.1 (0.5)</td>
<td>8.3 (0.7)**</td>
<td>0.2; 1.3</td>
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<td>LVMI (g/min²)</td>
<td>117.1 (9.9)</td>
<td>112.8 (11.9)</td>
<td>-5.9; 14.6</td>
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<tr>
<td>LAD (mm)</td>
<td>39.1 (4.3)</td>
<td>38.6 (3.5)</td>
<td>-3.6; 4.3</td>
</tr>
<tr>
<td>LLAD (mm)</td>
<td>58.2 (2.6)</td>
<td>56.7 (2.1)</td>
<td>-0.3; 4.7</td>
</tr>
<tr>
<td>LRAD (mm)</td>
<td>2.4 (0.8)</td>
<td>2.1 (0.9)</td>
<td>-0.6; 1.1</td>
</tr>
</tbody>
</table>

All values are expressed as means (SD). *p<0.01; **p<0.05 compared with D group.

Echocardiographic variables

The echocardiographically derived cardiac dimensions in our subjects (both D and NoD groups) were similar or slightly higher (in the case of LVIDd) than those reported in previous studies for highly trained endurance athletes.43 The present subjects indeed showed a cardiac profile expected of endurance athletes44 with predominantly eccentric LV hypertrophy (enlarged LVIDd).
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and proportional increase in wall thickness). Few previous studies have reported echocardiographic variables in professional cyclists. M.-J. Missault et al. and Rodriguez Reguero et al. showed the existence of significant non-pathological cardiac hypertrophy in professional riders of comparable characteristics (V0MAX, training, competition experience) to those of the present subjects. However, our results are not in agreement with the original hypothesis of Pokan et al. Left ventricular internal dimensions (LVIDd) were similar in the two groups, while mean LPWTd and IVSTd values were significantly higher in the D group (p<0.01 and p<0.05 respectively). The present findings suggest that athletes who show a deflection point in their HR response to incremental exercise are those with greater wall thickness rather than greater cavity dimensions. It may be speculated that HR deflection is due to more efficient cardiac function during high intensity exercise in cyclists who have a thicker myocardial wall. Further research—for example, measuring diastolic and systolic ventricular function during exercise—is required to corroborate this hypothesis. Gledhill et al. found that stroke volume does not plateau at high exercise intensities during an incremental test in well-trained endurance athletes. This latter phenomenon would be especially remarkable in elite endurance athletes showing a point of deflection in HR—that is, the D group. Whatever the case, the shape of the HR response curve (linear vs. curvilinear) does not seem to affect maximal exercise performance in endurance athletes, as indicative parameters such as V0MAX were similar in the two groups. Future studies involving other population groups such as sedentary subjects or cardiovascular patients would serve to confirm our findings.

In conclusion, HR may show a curvilinear response during incremental exercise in a considerable number of highly trained endurance athletes such as those in the present investigation (top level cyclists with excellent racing performance history). It may also be suggested that the deflection in HR increase may occur predominantly in subjects with thicker heart walls and does not seem to bear any relation to heart volume.

We wish to thank Ana Burton for her linguistic assistance. We gratefully acknowledge Dr. José López-Calbet (University of Las Palmas) for assisting us with the use of the computer algorithm linear regression model and Dr. Ignacio Busturia (Departamento de Matemáticas, Universidad Europea de Madrid) for his help in the statistical analyses.


**Take home message**

Heart rate shows a curvilinear (downward) response to incremental exercise in a considerable number of top level (professional) cyclists. Such a deflection in HR increase may predominantly occur in those with thicker heart walls and is not related to heart volume.

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