Original articles

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

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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 (LVPWTd), interventricular septal wall thickness at end diastole (IVSTd), left ventricular mass index (LVMI), left atrial dimension (LAD), longitudinal left atrial (LLAD) and right atrial (LRAD) dimensions, and the ratio of early to late (E/A) diastolic flow velocity.

Results—The HR response showed a deflection point (HR$_d$) at about 85% $\dot{V}O_2MAX$ in 66.7% of subjects (D group; n = 14) and was linear in 33.3% (NoD group; n = 7). Several echocardiographic variables (LVMI, LAD, LLAD, LRAD) 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.

(*Br J Sports Med* 1999;**33**:387–392)

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[1] 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[2] 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).[2] In subsequent years, several studies conducted by Conconi *et al*[3–5] and by other authors[6–9] 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[10] or children.[11–13] With regard to the latter, Ballarin *et al*[11] 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.[14] 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.[15] 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.[16] Moreover, when identified, HR deflection tended to overestimate AT. Schmid *et al*[17] 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.[18–27]

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*[2 28] 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.[28] In this regard, it has been recently shown that stroke volume may show a linear increase up to maximal intensities in trained endurance athletes.[29] 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[3–9] does not necessarily imply a causal relation between the phenomena.

Most recently, Pokan *et al*[30] 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 (*a*) voluntarily by the subjects, (*b*) when pedalling cadence could not be maintained at 70 rpm, or (*c*) when the established criteria for test termination were met.[31] Tests were performed under similar environmental conditions (21–24°C; 45–55% relative humidity). Gas exchange data were monitored throughout the test using an automated breath by breath system (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 ($\dot{V}E/\dot{V}O_2$) and carbon dioxide ($\dot{V}E/\dot{V}CO_2$) were recorded during each test. The first ventilatory threshold (VT_1) was determined using the criterion of an increase in $\dot{V}E/\dot{V}O_2$ with no increase in $\dot{V}E/\dot{V}CO_2$ and the departure from linearity of $\dot{V}E$.[32] The second ventilatory threshold (VT_2) was determined by use of the criterion of an increase in both $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$.[32] Two independent observers detected VT_1 and VT_2. 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

Weltman and co-workers.[33] 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.[34]

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.[35] 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 HR_d) was defined as that above which the values of b started to decrease.[28] 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 HR_d).

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)[36]: 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[37]: 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 were obtained at the level of the mitral annulus from the apical four chamber view to assess left ventricular filling dynamics. For each Doppler profile, peak velocities (cm/s) 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 ($\dot{V}O_2MAX$, VT_1, 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 ($\%\dot{V}O_2MAX$) eliciting HR_d and that eliciting VT_1, VT_2, LT, and OBLA. Pearson product

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

	D (n=14)	NoD (n=7)	Confidence limits for difference between group means
Age (years)	25 (3)	24 (3)	−2.2; 3.9
$\dot{V}O_2MAX$ (ml/kg/min)	70.0 (2.0)	71.1 (6.7)	−4.3; 3.1
W_{max}	508.2 (34.0)	492.9 (13.8)	−12.9; 43.6
HR_{max} (beats/min)	196 (10)	192 (7)	−4.7; 13.2
VT_1 (%$\dot{V}O_2MAX$)	69.8 (5.6)	72.3 (2.8)	−8.9; 1.3
VT_2 (%$\dot{V}O_2MAX$)	87.7 (4.0)	88.2 (3.6)	−4.3; 3.2
LT (%$\dot{V}_2MAX$)	68.3 (8.0)	72.0 (2.7)	−2.9; 2.1
OBLA (%<$\dot{V}O_2MAX$)	88.5 (4.9)	91.5 (3.8)	−6.0; 3.2
HR_d (beats/min)*	176 (10)		
HR_d (%$\dot{V}O_2MAX$)	85.3 (8.3)		
HR_b (W)	402.6 (48.4)		

All values are expressed as means (SD). *Significantly different ($p<0.01$) from both VT_1 and LT. Abbreviations: $\dot{V}_2max$, maximal oxygen consumption; W_{max}, maximal power output (in W); HR_{max}, maximal heart rate; VT_1, first ventilatory threshold; VT_2, second ventilatory threshold; LT, lactate threshold; OBLA, onset of blood lactate accumulation; HR_d, heart rate deflection point.
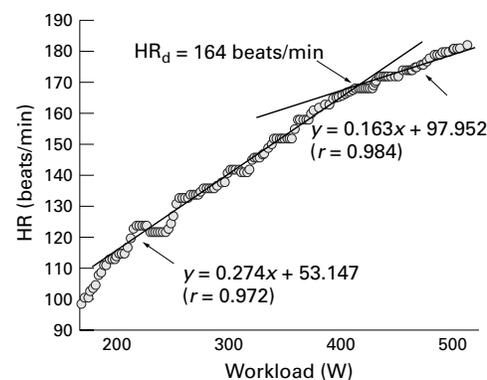

Figure 1 Determination of heart rate deflection point (HR_d) in one subject from the HR/workload (W) relation. At times (such as in this example), the initial data points—that is, below 100–110 beats/min—do not fall on the straight line and should be ignored according to the methodology of Conconi and co-workers.[28] This phenomenon may be attributed to the fact that HR adapts initially (at the lowest workloads) to the increasing intensity by increasing stroke volume more than rate.[28]

Table 2 Echocardiographic variables

	D (n=14)	NoD (n=7)	Confidence limits for difference between group means
LVIDd (mm)	60.9 (2.9)	61.9 (2.6)	−3.8; 1.7
LVPWTd (mm)	9.0 (0.6)	8.4 (0.6)*	0.1; 1.2
IVSTd (mm)	9.1 (0.5)	8.3 (0.7)**	0.2; 1.3
LVMI (g/min^2)	117.1 (9.9)	112.8 (11.9)	−5.9; 14.6
LAD (mm)	39.1 (4.3)	38.6 (3.5)	−3.6; 4.3
LLAD (mm)	58.8 (3.8)	54.8 (7.3)	−2.8; 10.9
LRAD (mm)	58.2 (2.6)	56.7 (2.1)	−0.3; 4.7
E/A	2.4 (0.8)	2.1 (0.9)	−0.6; 1.1

All values are expressed as means (SD). *$p<0.01$; **$p<0.05$ compared with D group.
Abbreviations: LVIDd, left ventricular end diastolic internal diameter; LVPWTd, left ventricular posterior wall thickness at end diastole; IVSTd, interventricular septal wall thickness at end diastole; LVMI, left ventricular mass index; LAD, left atrial dimension; LLAD, longitudinal left atrial dimension; LRAD, longitudinal right atrial dimension; E/A, diastolic flow velocity.

moment correlation coefficients were also calculated to determine whether there was a significant relation between HR_d (%$\dot{V}o_2MAX$) and the above variables.

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

Results

EXERCISE TEST

Table 1 shows data obtained in the exercise tests. The HR response showed a deflection point in 66.7% of subjects (D group; n = 14) but was linear in 33.3% (NoD group; n = 7). Figure 1 shows an example of HR_d determination in one of the D group subjects.

No significant differences were found between groups. In the D group, HR_d occurred at higher intensities than those eliciting VT_1 or LT ($p<0.01$). In contrast, no significant differences were found in this group when HR_d was compared with VT_2 on the one hand, and OBLA on the other. Finally, no significant correlation was observed between HR_d and the aforementioned variables.

ECHOCARDIOGRAPHIC VARIABLES

Table 2 shows data corresponding to the test and echocardiographic variables. The echocardiographic variables LVIDd, LVMI, LAD, LLAD, LRAD indicative of heart dimensions were similar in both groups. Mean LPWTd ($p<0.01$) and IVSTd ($p<0.05$) values, however, were significantly different between the groups, indicating a greater heart wall thickness in the D group subjects. Finally, no significant differences were found in E/A values between groups.

Discussion

The HR response showed a deflection point in most subjects (about two thirds) and was linear in the rest (about one third). These findings are in agreement with those of several studies which report that most young healthy subjects may exhibit a curvilinear HR response to incremental exercise with a lower HR increase rate during highly intense exercise—that is, involving mainly anaerobic metabolism.[8 38 39] Following the most recent recommendations made by Conconi et al[28] for HR_d determination, a ramp protocol (increases of 25 W/min) was selected for this investigation. According to Conconi's team, the fact that some authors have previously failed to detect HR_d 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) are indeed more appropriate for the detection of HR_d in subjects showing a curvilinear HR response.[28]

To date, little attention has been paid to the physiological mechanisms involved in the HR deflection that is likely to occur in some individuals during incremental tests. Conconi et al[28] proposed that the deflection is caused by activation of the anaerobic lactacid mechanisms of ATP production irrespective of cardiocirculatory activity and HR. However, as far as we are aware, no investigation has been specifically designed to confirm such a hypothesis. The fact that the exercise intensity at which HR_d occurs and that eliciting AT[3–9] coincide does not necessarily imply a causal relation between the phenomena.

In our study, HR_d occurred at about 85% $\dot{V}o_2MAX$, or before the exercise intensity (about 70% $\dot{V}o_2MAX$) corresponding to the LT or VT_1, in which anaerobic metabolism is partly involved. In contrast, HR_d was detected at a comparable workload to that corresponding to the situation in which lactate production exceeds clearance with subsequent blood lactate accumulation (OBLA) and ventilatory compensation (VT_2) (about 90% $\dot{V}o_2MAX$). No significant correlation was observed between HR_d and OBLA or VT_2. We propose that future research protocols should mainly focus on establishing whether or not there is a cause-effect relation between the involvement of anaerobic metabolism and the occurrence of HR_d. On the other hand, the regulation of HR during exercise is achieved by reduced parasympathetic drive at lower workloads and by increased sympathetic activity at higher intensities.[40] In this perspective, the downward deflection of the HR-workload relation may also be, at least in part, attributed to the fact that parasympathetic control of HR may be maintained even at highly intense exercise levels. Recent findings by Pokan et al,[30] however, suggest that the HR response to gradual exercise and its regulation is an individual and reproducible characteristic and may not be dependent on sympathetic/parasympathetic activity. They further hypothesise that subjects who show a deflection point in their HR response to incremental exercise are those with greater heart dimensions measured at rest—that is, well trained endurance subjects. Thus HR deflection could represent an effort to favour diastolic filling and consequently systolic function during high intensity exercise.

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.[41–44] The present subjects indeed showed a cardiac profile expected of endurance athletes[44] with predominantly eccentric LV hypertrophy (enlarged LVIDd

and proportional increase in wall thickness). Few previous studies have reported echocardiographic variables in professional cyclists.[45–46] Missault *et al*[45] and Rodríguez Reguero *et al*[46] showed the existence of significant non-pathological cardiac hypertrophy in professional riders of comparable characteristics ($\dot{V}O_2MAX$, training, competition experience) to those of the present subjects. However, our results are not in agreement with the original hypothesis of Pokan *et al*.[30] 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*[29] 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 *v* curvilinear) does not seem to affect maximal exercise performance in endurance athletes, as indicative parameters such as $\dot{V}O_2MAX$ were similar in the two groups. Future studies involving other population groups such as sedentary subjects or cardiopulmonary 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.

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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.

British Association of Sport and Medicine in association with the National Sports Medicine Institute

Education programme 2000

IAB/SEM Diploma Preparation Course   **Late March**
Exact date and venue to be confirmed
PGEA and CME will be sort

General Sports Medicine Course   **9–14 April**
Lilleshall Hall National Sports Centre, Shropshire (residential)
PGEA and CME will be given

Intermediate Sports Injury Course—Part 1   **9–14 July**
Lilleshall Hall National Sports Centre, Shropshire (residential)
PGEA and CME will be given

General Sports Medicine Course   **24–29 September**
Lilleshall Hall National Sports Centre, Shropshire (residential)
PGEA and CME will be given

Practical Sport and Medicine Meeting   **5–12 October**
Club La Santa, Lanzarote (residential)

Advanced Sports Medicine Course   **8–13 October**
Lilleshall Hall National Sports Centre, Shropshire (residential)
PGEA and CME will be sort

BASM National Congress: (West Midlands)   **3–5 November**
Stakis Luxury Puckrup Hall Hotel, Tewkesbury

Intermediate Sports Injury Course—Part 2   **19–14 November**
Lilleshall Hall National Sports Centre, Shropshire (residential)
PGEA and CME will be given

For further details of these courses please contact Mr Barry Hill, The National Sports Medicine Institute, c/o Medical College of St Bartholomew's Hospital, Charterhouse Square, London EC1M 6BQ.
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