Previous research conducted by Conconi’s group [1, 2] and by other authors [3–5] on young or middle-aged subjects of varying fitness levels has suggested: (1) the existence of a deflection point (HRd) in the heart rate (HR) response to an incremental exercise protocol after which the previously linear HR/workload relationship becomes curvilinear, and (2) its association with the anaerobic threshold (AT) [1, 2]. However, medical sports literature reflects controversy with respect to the Conconi hypothesis. Several investigators report that the HRd only occurs in certain subjects, and if detected, does not necessarily coincide with the AT [6–8]. In a recent report from our laboratory, we found the existence of HRd in the majority of a group of professional road cyclists (70% of a total of 21 subjects) [9].

Despite this controversy, surprisingly few research efforts have focused on the physiological mechanisms involved in HR deflection which is likely to occur in some individuals during incremental tests. It was originally proposed that HR deflection might be attributed, at least in part, to activation of the anaerobic lactacid mechanisms of ATP production, irrespective of cardiocirculatory activity and HR [2]. However, no investigation has been specifically designed to confirm such a hypothesis. More recently, Pokan et al. [10] have hypothesized that subjects who exhibit a deflection point in the HR response to incremental exercise are those with greater heart volumes measured at rest (i.e., well-trained endurance subjects). From this perspective, HR deflection could represent an effort to favor diastolic filling, and thus improve systolic function during high-intensity exercise (above the AT) [10]. However, we recently found that, in elite athletes (i.e., professional road cyclists), the downward deflection in HR response to exercise predominantly occurs in those whose hearts have greater wall thickness [9].

On the other hand, surprisingly no data exists concerning the possible existence of a HRd in the HR response of elderly. The aim of this study was therefore to analyze the kinetics of HR response (i.e., existence of HRd) during incremental exercise in a group of master runners. A secondary goal was to assess the possible influence of heart dimensions measured at rest with echocardiographic evaluation (i.e., diastolic diameter of left ventricular cavity and wall thickness measured) in such response.

To be eligible for this investigation, subjects were required to meet the following criteria: (1) >55 yr of age; (2) in good health, as determined by blood analysis (i.e., undetectable serum levels of a cardiac damage indicator such as cardiac troponin I), spirometry.

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Abstract: We analyzed the kinetics of heart rate (HR) response during incremental treadmill exercise in thirteen master runners (62 ± 1 yr). The HR/running speed (HR/S) relationship showed the existence of a point of downward deflection (HRd) in only ~31% of the subjects. Resting echocardiographic evaluations showed similar heart dimensions in all of the subjects. In conclusion, HR does not seem to show a curvilinear response (downward deflection) in most aged athletes. [Japanese Journal of Physiology, 50, 155–158, 2000]

Key words: heart rate, echocardiography, aging, endurance exercise.
tests and cardiac examination (including 12-lead electrocardiogram and M-mode, two-dimensional and Doppler echocardiograms) within the previous month; (3) no history of cardiopulmonary disease; and (4) no history of use of cardiovascular drugs.

Thirteen marathoners (11 male and 2 female) were enrolled in the study. Values of mean (±SEM) age, height, weight and body mass index (BMI) were the following: 62±1 yr (range 55–70), 167.5±3.5 cm, 70.6±3.6 kg and 25.2±4.0. Their mean training experience was 13±3 yr during which they had completed 15±3 marathon races. Their average training load during the last two months previous to the study was of 65±6 km·week⁻¹. Informed consent was obtained from each subject in accordance with the regulations of Complutense University.

Each subject performed a ramp exercise test (see below) and underwent an echocardiographic examination within the same week. The results of the exercise test permitted the division of subjects into two groups: those showing a HRd (D group), and those showing no such downward deflection (linear response or upward deflection) (NoD group).

Each subject performed a ramp protocol to exhaustion. According to Conconi et al. [2], gradual ramp protocols (i.e., eliciting increases of HR <8 beats·min⁻¹ per min of test) are most suitable to detect HRd. Starting at 6 km·h⁻¹, running velocity was increased by 0.3 km·h⁻¹ at each 20-s interval. Treadmill (LE-6000; Erich Jaeger; Wuerzburg, Germany) inclination was kept constant at 1.0%. All exercise tests were terminated voluntarily by the subjects or when two of the following criteria of test termination were met [11]: plateau of \( \dot{V}O_{2\text{max}} \) with increasing work rate, heart rate exceeding 95% of age-predicted maximum, or respiratory exchange ratio exceeding 1.15. Expired gases were analysed with the use of an automated breath-by-breath system (CPX; Medical Graphics; St. Paul, MN, USA). The first ventilatory threshold \( (V_{T1}) \) was determined using the criterion of an increase in the ventilatory equivalent for oxygen \( (\dot{V}E·\dot{V}O_2) \) with no increase in the ventilatory equivalent for carbon dioxide \( (\dot{V}E·\dot{V}CO_2) \) and departure from the linearity of \( \dot{V}E \), whereas the second ventilatory threshold \( (V_{T2}) \) was determined by use of the criterion of an increase in both \( \dot{V}E·\dot{V}O_2 \) and \( \dot{V}E·\dot{V}CO_2 \) [12]. Two independent observers detected \( V_{T1} \) and \( V_{T2} \) [12]. If there was disagreement, the opinion of a third investigator was sought.

Heart rate (HR, in beats·min⁻¹) was continuously monitored during the tests from modified 12-lead ECG tracings (EK56; Hellige; Freiburg, Germany). The HR/running speed (HR/S) relationship was graphed and analysed using a computer algorithm linear regression model which allows detection of the point of passage from the linear phase of the HR/S relationship to the curvilinear phase (so called “point of HR deflection,” or HRd) [2].

Transthoracic M-mode, two-dimensional and Doppler echocardiographic examinations were performed by the same experienced investigator with a Toshiba SSH-140A (Toshiba Medical Systems, Madrid, Spain) using a 2.5 MHz transducer. The following parameters were measured according to the recommendations of the American Society of Echocardiography [13]: left ventricular end-diastolic internal diameter (LVIDd), left ventricular posterior wall thickness at end-diastole (LVPWd), interventricular septal wall thickness at end diastole (IVSd), left atrial dimension (LAD), left atrial (LLAD) and right atrial (LRAD) longitudinal dimensions, and left ventricular mass index (LVMi). For each Doppler profile, peak velocities (cm·s⁻¹) of left ventricular inflow in early (E) and late (A) diastole, and the ratio of early to late (E/A) diastolic flow velocity were also calculated.

Results are expressed as means±SEM. A Student’s \( t \)-test for unpaired data was performed to compare the mean values obtained in the D and NoD groups of (1) the physiological parameters measured during the exercise tests and (2) the echocardiographic parameters. Finally, a Student’s \( t \)-test for paired data was applied to compare the exercise intensity corresponding to HRd and that corresponding to \( V_{T1} \) and \( V_{T2} \) within the D group. The level of significance was set at 0.05.

The HR/S relationship showed a downward deflection in only ~31% of the subjects (D group; \( n=4 \)). In these subjects, HRd (148±5 beats·min⁻¹) occurred at an exercise intensity (82.0±4.2% of \( \dot{V}O_{2\text{max}} \)) which was significantly higher (\( p<0.05 \)) than that corresponding to \( V_{T1} \) (62.8±1.6% of \( \dot{V}O_{2\text{max}} \)) but similar to that of \( V_{T2} \) (83.3±3.3% of \( \dot{V}O_{2\text{max}} \)). In contrast, in ~69% of the subjects (NoD group; \( n=9 \)), the HR/S relationship was either linear (\( n=6 \)) or showed an upward deflection (\( n=3 \)). Figure 1 shows the HR kinetics (downward or upward deflection, linear response) for each of the subjects.

On the other hand, no significant differences (\( p>0.05 \)) were found between D and NoD for physiological parameters recorded during the tests or in the echocardiographic data (Table 1).

To our knowledge, this study represents the first attempt to analyze the kinetics of the HR/workload intensity with reference to the Conconi hypothesis in a group of aged (>55–60 yr), well-trained athletes. HR showed a downward deflection in only ~31% of the
subjects. Following the most recent recommendations made by Conconi et al. [2] for HRd determination, a ramp protocol was selected for this investigation. According to Conconi’s team, the fact that some authors have previously failed to detect HRd may be explained by the protocol used (i.e., step-like workload increases rather than the more gradual ramp method). On the other hand, in those cases in which the initial data points (i.e., below 100–110 beats · min$^{-1}$) did not fall on the straight line (such as in Fig. 1), the aforementioned data were ignored according to the methodology of Conconi et al. [2]. This phenomenon might be attributed to the fact that at the lowest workload heart rate often adapts to the increasing intensity by increasing stroke volume more than rate [2].

To date, little attention has been paid to the physiological mechanisms involved in the occurrence of HRd. Conconi et al. [2] propose that the 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 indeed facilitate the release of oxygen from hemoglobin (the so-called “Bohr effect”), and thus improve cardiocirculatory efficiency and attenuate the HR increase. The present findings suggest that, when existing, the occurrence of HRd might be partly attributed to the aforementioned mechanism since both HRd and $V_{T2}$ occurred at similar intensities. The great variability of HR response (linear, downward or upward deflection) could reflect, on the other hand, a great inter-individual variability in sympathetic/parasympathetic activity. However, Pokan et al. [10] suggest that the HR response to

Table 1. Comparisons between groups.

<table>
<thead>
<tr>
<th></th>
<th>D ($n=4$)</th>
<th>NoD ($n=9$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>62±1</td>
<td>61±1</td>
</tr>
<tr>
<td>$V_{O2\text{max}}$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>50.1±4.0</td>
<td>49.4±2.9</td>
</tr>
<tr>
<td>$VT_1$ (%$V_{O2\text{max}}$)</td>
<td>62.8±1.6</td>
<td>64.9±2.4</td>
</tr>
<tr>
<td>$VT_2$ (%$V_{O2\text{max}}$)</td>
<td>83.3±3.3</td>
<td>83.1±2.9</td>
</tr>
<tr>
<td>HRd (beats · min$^{-1}$)</td>
<td>160±4</td>
<td>164±3</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td>55.9±1.0</td>
<td>53.4±0.7</td>
</tr>
<tr>
<td>LVPWTd (mm)</td>
<td>9.1±1.1</td>
<td>9.6±0.3</td>
</tr>
<tr>
<td>IVSTD (mm)</td>
<td>10.0±1.0</td>
<td>10.3±0.4</td>
</tr>
<tr>
<td>LVMi (g/min$^2$)</td>
<td>111.7±10.5</td>
<td>114.7±6.0</td>
</tr>
<tr>
<td>LAD (mm)</td>
<td>33.2±1.2</td>
<td>36.0±1.3</td>
</tr>
<tr>
<td>LLAD (mm)</td>
<td>55.9±3.5</td>
<td>54.0±1.7</td>
</tr>
<tr>
<td>LRAD (mm)</td>
<td>58.2±2.6</td>
<td>57.2±2.0</td>
</tr>
<tr>
<td>E/A</td>
<td>1.0±0.1</td>
<td>1.1±0.1</td>
</tr>
</tbody>
</table>

All values are expressed as means±SEM. No significant differences were found between means ($p>0.05$). $V_{O2\text{max}}$, maximal oxygen consumption; HRd, maximal heart rate; 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 (LVMi); LAD, left atrial dimension; LLAD, longitudinal left atrial dimension; LRAD, longitudinal right atrial dimension; E/A, diastolic flow velocity.

Fig. 1. HR kinetics (downward or upward deflection, linear response in each of thirteen subjects. HR data points are average values for each 5 s-interval. Upper: Arrows indicate the point of upward deflection in the HR/S relationship; bottom: they indicate the point of downward deflection (HRd).
gradual exercise may be dependent on heart dimension, and HRd could indeed represent an effort to favor diastolic filling during high-intensity exercise in those with greater heart dimensions. Our results are not in agreement with such hypothesis since no differences in the measured echocardiographic parameters were found between both groups. Finally, cardiovascular performance (i.e., diastolic function and especially rapid diastolic filling, maximal attainable HR, etc.) during exercise is known to decline with aging [14, 15], and endurance training does not necessarily prevent such decline [14]. In this perspective, the lack of downward deflection (linear or even upward response) found in 69% of the subjects might reflect the need to rely on HR in order to maintain cardiac output at high intensities. Thus, those master runners showing an upward deflection in HR response may also be those with a greater age-associated decrease in cardiac pump performance.

In conclusion, HR does not seem to show a curvilinear response (downward deflection) in most aged athletes. Our results suggest that Conconi’s test should not be applied for routine functional evaluation (i.e., training prescription) in this population group. Further research is needed to analyze the mechanisms involved in the kinetics of HR response during incremental exercise in aged athletes.

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