Heart rate deflection point as a strategy to defend stroke volume during incremental exercise

Pierre-Marie Lepretre,1 Carl Foster,2 Jean-Pierre Koralsztein,3 and Veronique L. Billat1,3

1Laboratoire d’Etude de la Physiologie de l’Exercice, Department of Sciences and Technology in Sports and Physical Activities, University of Evry Val d’Essonne, Evry, France; 2University of Wisconsin-La Crosse, La Crosse, Wisconsin; and 3Sport Medicine Center of the Caisse Centrale d’Activités Sociales, Paris, France

Submitted 4 August 2004; accepted in final form 29 November 2004

Lepretre, Pierre-Marie, Carl Foster, Jean-Pierre Koralsztein, and Veronique L. Billat. Heart rate deflection point as a strategy to defend stroke volume during incremental exercise. J Appl Physiol 98: 1660–1665, 2005. First published December 23, 2004; doi:10.1152/japplphysiol.00837.2004.—The purpose of this study was to examine whether the heart rate (HR) deflection point (HRDP) in the HR-power relationship is concomitant with the maximal stroke volume (SVmax) value achievement in endurance-trained subjects. Twenty-two international male cyclists (30.3 ± 7.3 yr, 179.7 ± 7.2 cm, 71.3 ± 5.5 kg) undertook a graded cycling exercise (50 W every 3 min) in the upright position. Thoracic impedance was used to measure continuously the HR and stroke volume (SV) values. The HRDP was estimated by the third-order curvilinear regression method. As a result, 72.7% of the subjects (HRDP group, n = 16) presented a break point in their HR-work rate curve at 89.9 ± 2.8% of their maximal HR value. The SV value increased until 78.0 ± 9.3% of the power associated with maximal O2 uptake (V̇O2 max) in six other subjects (no-HRDP group; P = 0.004). Neither SVmax (ml/beat or ml·beat−1·m−2) nor VO2max (ml/min or ml·kg−1·min−1) were different between both groups. However, SV significantly decreased before exhaustion in the HRDP group (153 ± 44 vs. 144 ± 40 ml/beat; P = 0.005). In the HRDP group, 62% of the variance in the power associated with the SVmax could also be predicted by the power output at which HRDP appeared. In conclusion, in well-trained subjects, the power associated with the SVmax-HRDP relationship supposed that the HR deflection coincided with the optimal cardiac work for which SVmax was attained. Additionally, the application of the HR break point phenomenon is limited because a HR deflection cannot be found even in young subjects in some cases (16). Elsewhere, in 227 healthy young subjects (age: 23 ± 4 yr), Hofmann et al. (14) reported a significant relationship of the HR threshold to the anaerobic lactate threshold (LT) (2) in 85.9% of the subjects who showed a HR deflection. Furthermore, the expression of the linear deviation of HR value, which represented the start of the plateau at maximal HR (HRmax) in the conventional incremental tests, was dependent on the specifics of the Conconi test protocol (17, 27). Although Lucia et al. (22) reported an occurrence of HRDP of 88% in top-level cyclists with thicker heart walls, Jones et al. (16) showed a lack of the HRDP reproducibility in 9 out of 15 well-trained male distance runners who performed a treadmill simulation of the Conconi test protocol. Therefore, HRDP was not demonstrated in a considerable number of highly trained endurance athletes, whereas their HR response was curvilinear during the incremental exercise (22).

Beyond this methodological criticism, the cause of the HRDP phenomena can also be questioned. Although left ventricular ejection fraction (LVEF) increased from rest to the LT in healthy male subjects (12, 25, 26), the break point in the HR-work performance curve became less pronounced or was absent altogether when the decrease in LVEF toward the end of the incremental exercise became more distinct (24). Expressed as higher ejection fraction both at rest and during exercise, it has been established that the endurance-trained athletes had a better systolic function than untrained subjects (10, 13). The regulatory mechanisms, however, appeared similar for athletes and healthy sedentary men (15). However, the ability of athletes to decrease their end-systolic volumes (3) may be due to a better systolic function reflecting an enhanced myocardial contractility, contributing to the maintenance of a large SV during an incremental exercise compared with sedentary subjects (15, 30). The parallel increase in SV and left ventricular end-diastolic volume (EDV) could at least, in part, be contributed to the Frank-Starling mechanism, in either trained or untrained subjects. Considering the increase in the SV value almost until the power associated with the maximal oxygen uptake value (pV̇O2 max) in well-trained subjects, we can hypothesize a cardiovascular origin of the HRDP appearance. Such endurance-trained subjects have also been reported to increase their SV until 90–100% of maximal oxygen uptake (VO2 max) in contrast to nontrained subjects (13, 35). Indeed, if
this increase in SV could be explained by a greater left ventricular filling in well-trained men (13), only some cases of subjects presented a plateau of SV before the end of exercise, whereas others increased their SV until exhaustion. It would also be interesting to elucidate whether the occurrence of HRDP could be influenced by the SV-power output relationship.

Therefore, the aim of this study was to determine whether the break point [i.e., inflection or deflection according to the different studies (8, 9, 14, 22)] in the HR-power curve (HRDP) was concomitant with the achievement of a SV plateau in well-trained subjects who showed a SV increase almost until \( V_{\text{O}2_{\text{max}}} \).

METHODS

Twenty-two healthy male well-trained subjects (11 triathletes and 11 cyclists) participated in this study. These subjects were national short-distance triathletes and international elite cyclists. They were free of cardiac and pulmonary disease. Their physical characteristics (means \( \pm SD \)) are reported in Table 1. Each subject was habituated to the experimental procedures before the study. Before participation, they were informed of the risks and stresses associated with the protocol and gave written, informed consent in accordance with the guidelines of the University of Evry-Val d’Essonne.

Protocol. Two to three hours after a light breakfast, all subjects performed an incremental exercise in the upright position on an electronically braked ergometer (ERGOLINE 900, Hellige, Markett) in an air-conditioned room \((22.0 \pm 0.5^\circ \text{C})\). After a 3-min warm-up at 100 W, each subject performed a 3-min stage incremental exercise test to exhaustion with 50-W work increment for total exercise duration not exceeding 25 min. Seat and handlebar heights were set for each subject and kept constant for all of the tests. The pedaling frequency was freely selected by each subject between 70 and 120 rpm.

In this incremental protocol, \( V_{\text{O}2_{\text{max}}} \) was defined as the highest 30-s oxygen uptake \((V_{\text{O}2})\) value reached during exercise with a respiratory exchange ratio \((\text{RER}) > 1.1 [\text{RER} = \text{CO}_2 \text{ production} / \text{O}_2 \text{ production} (\text{VCO}_2 / \text{VO}_2)]\), blood lactate > 8 mM (19), and a peak HR at least equal to 90% of the age-predicted maximum. A plateau of VO2 was identified if the VO2 of the least stage was not greater than the previous stage by 2.1 ml·kg\(^{-1}\)·min\(^{-1}\) (32). The \( pV_{\text{O}2_{\text{max}}} \) was defined as the lowest associated power that elicited \( VO_{\text{2max}} \) (4). If, during the last stage, a subject achieved \( VO_{\text{2max}} \) without completing the 3-min stage, \( pVO_{\text{2max}} \) was calculated as follows:

\[
pVO_{2_{\text{max}}} = pF + \left[\frac{t}{180}\right] \times 50 \text{ W}
\]

where \( pF \) is the power of the last complete stage (W), \( t \) is the time the last workload was maintained (s), and 50 W is the power output increment between the last of two stages.

Measurement of gas exchanges. The minute ventilation and gas-exchange parameters \((V_{\text{O}2}, V_{\text{CO}2})\) were measured breath by breath by using a telemetric system (K4b\(^2\), COSMED, Rome, Italy) (23). The response times of the oxygen and carbon dioxide analyzers were <120 ms to reach 90% of the flow sample. The ventilation range of the flowmeter was from 0 to 300 l/min. The time delay of the gas analyzer (time necessary for the gas to transit through the sampling line before being analyzed) was ~500 ms. This time delay was automatically assessed and taken into account in the calculations when a delay calibration procedure was performed, according to the manufacturer’s specifications. Before each test, the \( O_2 \) analysis system was calibrated using ambient air (20.9% \( O_2 \) and 0.03% \( CO_2 \)) and calibration gas (16.0% \( O_2 \) and 5.0% \( CO_2 \)), according to K4b\(^2\) instruction manual. The calibration of the turbine flowmeter of the analyzer was performed with a 3-liter syringe (Quinton Instruments, Seattle, WA). In addition, HR was recorded beat to beat and averaged over each breath. During all tests, the breath-by-breath data were smoothed and averaged every 5 s.

Measurement of blood lactate values. During both exercise tests, a capillary blood sample was obtained from the ear tip and analyzed for blood lactate concentration (Lactate Pro LT, ARKAY, Kyoto, Japan) (28). The samples were taken at rest, at the end of cycling, and every 3 min during the all-out exercise and at 2 and 4 min during recovery after the exercise bout. For the incremental test, the LT was defined as the \( VO_2 \) corresponding to the starting point of an accelerated lactate accumulation of ~4 mM and expressed in percentage of \( VO_{2_{\text{max}}} \) (2).

Table 1. Physical characteristics of the subjects

<table>
<thead>
<tr>
<th>HRDP group*</th>
<th>( n = 16 )</th>
<th>( n = 6 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>30.7±7.1</td>
<td>29.8±7.5</td>
</tr>
<tr>
<td>Height, cm</td>
<td>177.9±6.9</td>
<td>181.4±7.5</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>69.8±5.5</td>
<td>72.8±5.4</td>
</tr>
<tr>
<td>Body Fat, %</td>
<td>11.9±3.6</td>
<td>11.6±2.9</td>
</tr>
<tr>
<td>( V_{2_{\text{O}2_{\text{max}}}} ), ml/min</td>
<td>5,116±554</td>
<td>5,091±562</td>
</tr>
<tr>
<td>( V_{2_{\text{O}2_{\text{max}}}} ), ml/kg·min(^{-1})</td>
<td>72.4±5.5</td>
<td>72.1±6.4</td>
</tr>
<tr>
<td>( Q_{\text{max}} ), l/min</td>
<td>14.6±4.3</td>
<td>14.8±2.4</td>
</tr>
<tr>
<td>HRmax, beats/min</td>
<td>183±20</td>
<td>191±6</td>
</tr>
<tr>
<td>SVmax, ml/min</td>
<td>153±34</td>
<td>158±31</td>
</tr>
<tr>
<td>ISVmax, ml·beat(^{-1})</td>
<td>81±23</td>
<td>78±13</td>
</tr>
<tr>
<td>( ISV_{\text{end-of-exercise}} ), ml·beat(^{-1})</td>
<td>144±40†</td>
<td>158±31</td>
</tr>
</tbody>
</table>

Values are means ± SD; \( n \), no. of subjects. HRDP, heart rate deflection point; \( V_{2_{\text{O}2_{\text{max}}}} \), maximal oxygen uptake; \( Q_{\text{max}} \), maximal cardiac output; \( ISV_{\text{max}} \), maximal stroke volume related to body surface area; \( SV_{\text{end-of-exercise}} \), stroke volume at end of exercise. Level of significance between *HRDP and No-HRDP groups and †maximal and the end-exercise values: \( P < 0.05 \).
During the systolic upstroke divided by the basal thoracic impedance. Each displayed SV value represents the mean of five successive artifact-free beats. For this experiment, SV and HR were measured continuously during each test with beat-to-beat data smoothed by a 5-s moving averaging algorithm. Recently, Charloux et al. (7) compared cardiac output determined simultaneously by the physioflow impedance (COPF) and the direct Fick method (COFick) in 40 patients who performed a steady-state exercise below the LT (10–50 W, according to the patient’s fitness). Their results showed that the mean difference could also be predicted by the power associated with the SVmax (pSVmax) could also be predicted by the power associated with HRDP (pHRDP). Hence, a strong relationship was also found between the work rate values at HRDP and at SVmax in the HRDP group (r = 0.96, P < 0.01, n = 16, Fig. 3). In the HRDP group, the power associated with the LT (pLT) was lower than the pHRDP (pLT = 75.1 ± 6.7 vs. pHRDP = 78.3 ± 7.0% of the pV̇O₂ max, P = 0.04). The pSVmax value was also higher than the pLT (pSVmax = 78.0 ± 9.3 vs. pLT = 75.1 ± 6.7% of the pV̇O₂ max, P = 0.038) in this group.

Conversely, neither a deflection point in the HR-intensity curve nor a SV plateau was detected in 23.1% of the subjects (no-HRDP group, n = 6). Whatever the SV responses in the incremental test, the SVmax and HRmax values were not differ-

### Table 2. Maximal values determined during incremental test

<table>
<thead>
<tr>
<th></th>
<th>pV̇O₂ max</th>
<th>pLT</th>
<th>pSV max</th>
<th>pHr deflection</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRDP group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 16)</td>
<td>W</td>
<td>W/kg</td>
<td>%pV̇O₂ max</td>
<td>W</td>
</tr>
<tr>
<td></td>
<td>383 ± 66</td>
<td>5.5 ± 0.8</td>
<td>289 ± 57</td>
<td>75.1 ± 6.7</td>
</tr>
<tr>
<td></td>
<td>300 ± 67‡</td>
<td>78.0 ± 9.3‡</td>
<td>381 ± 75</td>
<td>94.4 ± 8.6</td>
</tr>
<tr>
<td></td>
<td>W</td>
<td>%pV̇O₂ max</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRDP group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n = 16)</td>
<td>W</td>
<td>W/kg</td>
<td>%pV̇O₂ max</td>
<td>W</td>
</tr>
<tr>
<td></td>
<td>402 ± 59</td>
<td>5.5 ± 0.8</td>
<td>313 ± 42</td>
<td>78.2 ± 3.4</td>
</tr>
<tr>
<td></td>
<td>381 ± 75</td>
<td>94.4 ± 8.6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. pV̇O₂ max, power at V̇O₂ max; pLT, power associated with lactate threshold; pSVmax, power associated with SVmax; pHr deflection, power associated with the appearance of HRDP. Level of significance between HRDP group and no-HRDP group; ‡pV̇O₂ max and the other intensities; ‡pLT and pSVmax-pHr deflection: P < 0.05.
ent between HRDP and no-HRDP groups (153 ± 44 vs. 158 ± 31 ml/beat, \( P = 0.1218 \), and 183 ± 10 vs. 191 ± 6 beats/min, \( P < 0.05 \)). Therefore, \( \dot{V}O_2 \) reached its maximal value without any significant difference between HRDP and no-HRDP groups (5,116 ± 554 vs. 5,091 ± 562 ml/min, \( P = 0.899 \)).

**DISCUSSION**

The focus of this study was to determine, in endurance-trained male cyclists, whether the break point of HR-power curve (HRDP) was concomitant to the achievement of SV plateau during the incremental test. The main finding from this study showed that the power at which the SV reached its maximal value was not significantly different from the pHRD in most of the subjects (72.7%). Moreover, the power at which HRDP appeared is highly correlated with the pSV\(_{\text{max}}\) but not associated with a higher plateau of SV values.

In the present study, the HR increase was not linear until the end of the incremental exercise but presented a break at 78.3 ± 7.0% of the p\( \dot{V}O_2\)\(_{\text{max}}\) (i.e., 89.9 ± 2.8% of the HR\(_{\text{max}}\) value) in 16 of our 22 subjects. In a previous investigation, Conconi et al. (9) showed a break point in the HR-exercise intensity relationship during the incremental exercise. According to this study, the change from the linear to the curvilinear phase in HR-exercise intensity relationship coincided with the beginning of a sharp accumulation of blood lactate (9). However, in the present study, the pLT was lower than the pHRDP, according to previous studies performed in highly trained endurance athletes (14, 22). Jones et al. (17) still reported that, in the conventional incremental tests, the expression of the linear deviation of HR value represented the start of the plateau at HR\(_{\text{max}}\) but not the lactate turning point.

In agreement with other studies performed with trained subjects (21, 35), SV reached its maximal value almost until \( \dot{V}O_2\)\(_{\text{max}}\) (from 82.4 ± 12.2 to 100% of the p\( \dot{V}O_2\)\(_{\text{max}}\)) in the present study. Indeed, SV did not plateau at 40–60% of \( \dot{V}O_2\)\(_{\text{max}}\) as reported in nonactive subjects (1). Thus, if the SV increased in a similar magnitude from rest to light exercise in all individuals, regardless of training state (1, 31), the SV of elite distance runners continued to increase without a plateau during a graded exercise test (35). Currently, the mechanisms
of this continuous increase in SV with increasing intensity in trained subjects are unclear. Previously, in 14 adult men, Gledhill et al. (13) showed a progressive increase in the SV until the end of the incremental exercise associated with a longer left VET and a shorter diastolic filling time in the endurance-trained athletes compared with the untrained subjects. Moreover, at the HRmax (near to 190 beats/min), the rate of filling for trained subjects was 86% greater than their rate of ventricular emptying (1,880 vs. 1,010 ml/s). Therefore, although trained athletes rely on enhancements in both ventricular filling and ventricular emptying, they concluded that, during the incremental work rates, the continuous increase in SV until exhaustion could be explained by a greater left ventricular filling in well-trained men compared with the untrained male subjects (13).

In the present study, each subject presented a linear relationship between the HR and the SV values. Additionally, the pSVmax was not significantly different than the pHHRDP in the group in which the HRDP appeared (HRDP group). Thus a high correlation coefficient was found between the pSVmax and HRDP in the HRDP group. In contrast, the SV of 23.1% of the subjects who did not present a HRDP (no-HRDP group) increased until exhaustion without reaching a plateau before the VO2max attainment. It has been established that training status explained the SV response during the incremental exercise (13, 31, 35). Therefore, in healthy male volunteers, 1) the LVEF (i.e., the ratio between the SV and the EDV) increased up to ventilatory threshold during an incremental cycling test (12), and 2) the identification of the HR deflection was useful to determine LT during a progressive cycling exercise (18). Previously, Pokan et al. (24) have shown, in 15 college students, a lack or a less pronounced deflection in the HR-power output relationship when the decrease in LVEF toward the end of the exercise became more distinct. Furthermore, a significant negative correlation has been found between the existence and extent of the HR-performance curve break point and the stress-dependent myocardial function, expressed as the deflection of LVEF-performance curve (r = 0.673, P < 0.01). Thus these authors have concluded that the absence of a HR-power curve variation was related to a diminished stress-dependent myocardial function. In contrast, in endurance-trained subjects, the LVEF rose until exhaustion (12), and a HR deflection was not found in all subjects during the incremental cycling exercise (14). It is not clear whether the detection or lack of detection of a SV plateau and a HRDP with increasing exercise intensity was an adaptation to training, a genetic consequence, or a combination of both. Nevertheless, Pokan et al. (25) reported the increase in the LVEF from rest to LT in 21 sports students. Thus it remained relatively constant until the maximal aerobic power attainment. Furthermore, all of these subjects showed an increase in SV below the LT, reaching its maximum value at the LT. They concluded, by the evidence, that there was a relationship between the higher SV value and an improvement of the systolic function with a constant left ventricular EDV in sports students compared with sedentary subjects. In the past, Bar-Shlomo et al. (3) did not show a significant change in the mean EDV but a less decrease in the mean end-systolic volume (to 64% of rest, P < 0.05) in nine endurance-trained athletes engaged in an incremental cycle exercise. In contrast, in 18 sedentary men, there was no significant difference between HR, blood pressure, or ejection fraction values between the healthy sedentary subjects and the endurance-trained athletes (3). More recently, Schairer et al. (30) observed that the greater increase in SV value was due to an increase of left ventricular EDV from 119 ± 23 to 152 ± 28 ml (P < 0.001) and a reduction in left ventricular end-systolic volume from 46 ± 14 to 31 ± 9 ml (P < 0.001) in athletes. On the other hand, these authors showed that left ventricular EDV did not change (96 ± 20 vs. 97 ± 28 ml) (P = not significant) and left ventricular end-systolic volume decreased (33 ± 11 vs. 20 ± 9 ml) (P < 0.001) in sedentary men. In both studies, the pooling between left ventricular EDV and the end-systolic volume responses was responsible for the higher SV value in endurance-trained athletes compared with sedentary subjects. The endurance-trained athletes increased cardiac output through a more prominent augmentation of SV than sedentary subjects during exercise. Hence, for the relationship between the power associated with the SV plateau and the power at which the HRDP appeared it is supposed that the HRDP coincided with the optimal cardiac work (left VET/diastolic filling time) for which SV attained its maximal value. Our results showed that, above the HRDP, the SV decreased. In a previous investigation, Pokan et al. (26) have shown a relationship between the power associated with the beginning of the decrease in the left ventricular function and the pHHRDP in 44 of 49 patients during an incremental cycling test (r = 0.93, P < 0.001). In our study, the drop of SV value from the pHHRDP to pVO2max may reflect the decrease in the LVEF to prevent myocardial overloading. This may have a conserving effect on myocardial VO2 at the supra-LT levels of exercise in subjects to present a deflection point in their HR-power output relationship (30). This could explain the finding that the HRDP application is limited because a break in the HR-exercise intensity relationship was not found in some endurance-trained subjects who presented a greater systolic filling compared with untrained subjects for whom SV reached a plateau near the LT intensity (12, 13).

Conclusion. In the present study, except for the no-HRDP group (n = 6) in which neither a HR point deflection nor a SV plateau was observed before reaching VO2max, most of the variance in the power output at the SVmax value can be predicted by the power output at which HRDP appears. The mechanisms underlying the HRDP could be mediated by cardiodynamic means in well-trained endurance cycling subjects to maintain diastolic filling time to uphold SV value.

GRANTS

This study was supported by grants from Caisse Centrale des Activités Sociales d’Electricité et de Gaz de France, the Foundation La Française des Jeux, and Evry Genopole.

REFERENCES


