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Journal of Applied Physiology 98:1660-1665, 2005. First published Dec 23, 2004;
doi:10.1152/jappphysiol.00837.2004

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

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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/jappphysiol.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 (SV_{max}) 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 $89.9 \pm 2.8\%$ of their maximal HR value. The SV value increased until $78.0 \pm 9.3\%$ of the power associated with maximal O₂ uptake ($\dot{V}O_{2\max}$) in the HRDP group, whereas it increased until $94.4 \pm 8.6\%$ of the power associated with $\dot{V}O_{2\max}$ in six other subjects (no-HRDP group, $P = 0.004$). Neither SV_{max} (ml/beat or ml·beat⁻¹·m⁻²) nor $\dot{V}O_{2\max}$ (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 SV_{max} could also be predicted by the power output at which HRDP appeared. In conclusion, in well-trained subjects, the power associated with the SV_{max}-HRDP relationship supposed that the HR deflection coincided with the optimal cardiac work for which SV_{max} was attained.

physical work curve break point; left ventricular ejection fraction; cycling graded test

THE RESPONSE OF HEART RATE (HR) to incremental exercise is not always linear (5, 6) but shows a break point in the HR-physical work curve, the so-called HR deflection point (HRDP). Conconi et al. (8) reported that the intensity of the passage from the linear to the curvilinear phase in the HR-physical work curve coincided with the beginning of a sharp accumulation of blood lactate. The identification of the HRDP could be associated with a noninvasive determination of the anaerobic threshold (34). However, Conconi et al. supported a metabolic interpretation of the appearance of the break point in the HR-intensity relationship while lacking the interaction between the increase in the stroke volume (SV) and the HR values during the incremental exercise tests.

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 (HR_{max}) 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 ($\dot{V}O_{2\max}$) 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 ($\dot{V}O_{2\max}$) in contrast to nontrained subjects (13, 35). Indeed, if

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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 $\dot{V}O_{2\text{max}}$.

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, $\dot{V}O_{2\text{max}}$ was defined as the highest 30-s oxygen uptake ($\dot{V}O_2$) value reached during exercise with a respiratory exchange ratio (RER) > 1.1 [$\text{RER} = \text{CO}_2$ production ($\dot{V}\text{CO}_2$)/ $\dot{V}O_2$], blood lactate > 8 mM (19), and a peak HR at least equal to 90% of the age-predicted maximum. A plateau of $\dot{V}O_2$ was identified if the $\dot{V}O_2$ of the least stage was not greater than the previous stage by $2.1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (32). The $p\dot{V}O_{2\text{max}}$ was defined as the lowest associated power that elicited $\dot{V}O_{2\text{max}}$ (4). If, during the last stage, a subject achieved $\dot{V}O_{2\text{max}}$ without completing the 3-min stage, $p\dot{V}O_{2\text{max}}$ was calculated as follows:

$$p\dot{V}O_{2\text{max}} = pF + [(t/180) \times 50 \text{ W}]$$

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 ($\dot{V}O_2$, $\dot{V}\text{CO}_2$) were measured breath by breath by

using a telemetric system (K4b², 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 K4b² 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 $\dot{V}O_2$ corresponding to the starting point of an accelerated lactate accumulation of ~ 4 mM and expressed in percentage of $\dot{V}O_{2\text{max}}$ (2).

Measurements of HR and SV values. We used a bioimpedance determination for SV and HR (Physioflow, Manatec type PF05L1). The theoretical basis for this technique and its application and validity for exercise testing have been previously described (7) and used in previous studies from our laboratory (21). The physioflow device measures impedance changes (dZ) in response to a small administered electrical current. Two sets of electrodes (Ag-AgCl, Hewlett Packard 40493 E), one electrode transmitting and the other sensing, were applied above the supraclavicular fossa at the left base of the neck and along the xiphoid. A further set of two electrodes was used to monitor a single ECG (CM5 position). With this impedance device, a first evaluation of SV index (SVI) is calculated during a calibration procedure based on 30 consecutive heartbeats, recorded in the resting condition. This evaluation keeps the largest impedance variation during the systole (maximum impedance minus minimum impedance) and the largest rate of variation of the impedance signal (dZ/dt_{max} , called the contractility index). The SVI calculation also depends on the thoracic flow inversion time (TFIT; in m/s) measured on the first mathematical derivative of the impedance signal. TFIT is the time interval between the first zero value following the beginning of the cardiac cycle (beginning of the ECG's QRS complex) and the first nadir after the peak of the ejection velocity (dZ/dt_{max}). Afterward, TFIT is weighted using a specific algorithm. During the data-acquisition phase, the variation of parameters was analyzed and compared with those obtained during the calibration procedure. Therefore, the SV was the product between the electrical physical volume of thorax, the ventricular ejection time (VET), and the maximum rate of the dZ

Table 1. Physical characteristics of the subjects

	Age, yr	Height, cm	Weight, kg	Body Fat, %	$\dot{V}O_{2\text{max}}$, ml/min	$\dot{V}O_{2\text{max}}$, $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	\dot{Q}_{max} , l/min	$\dot{I}\dot{Q}_{\text{max}}$, $\text{l}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$	HR _{max} , beats/min	SV _{max} , ml/beat	ISV _{max} , $\text{ml}\cdot\text{beat}^{-1}\cdot\text{m}^{-2}$	SV _{end-of-exercise} , ml/beat
HRDP group* (n = 16)	30.7 \pm 7.1	177.9 \pm 6.9	69.8 \pm 5.5	11.9 \pm 3.6	5,116 \pm 554	72.4 \pm 5.5	30.9 \pm 9.5	14.6 \pm 4.3	183 \pm 10	153 \pm 44	81 \pm 23	144 \pm 40†
No-HRDP group (n = 6)	29.8 \pm 7.5	181.4 \pm 7.5	72.8 \pm 5.4	11.6 \pm 2.9	5,091 \pm 562	72.1 \pm 6.4	32.2 \pm 4.9	14.8 \pm 2.4	191 \pm 6	158 \pm 31	78 \pm 13	158 \pm 31

Values are means \pm SD; n, no. of subjects. HRDP, heart rate deflection point; $\dot{V}O_{2\text{max}}$, maximal oxygen uptake; \dot{Q}_{max} , maximal cardiac output; $\dot{I}\dot{Q}_{\text{max}}$, maximal cardiac output related to body surface area; HR_{max}, maximal heart rate; SV_{max}, maximal stroke volume; ISV_{max}, maximal stroke volume related to body surface area; SV_{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: $P < 0.05$.

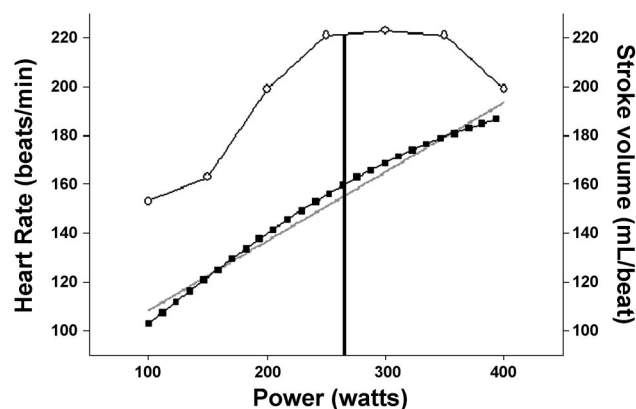


Fig. 1. Time course of heart rate (HR) and stroke volume (SV) during the incremental test. This is a typical example of a subject who presented a break point in the HR-power curve. ○, SV responses; ■ and solid line, HR responses; gray line, HR responses if the relation between HR and power has been linear.

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 (CO_{PF}) and the direct Fick method (CO_{Fick}) 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 between the CO_{Fick} and the CO_{PF} method was not significant at rest (0.07 l/min, i.e., 1%) or during steady-state exercise (0.26 l/min, i.e., 2%). Furthermore, Charloux et al. (7) reported a high relation between CO_{PF} and CO_{Fick} during the moderate steady-state exercise ($r^2 = 0.79$, $P < 0.05$, $n = 40$). They concluded that the physioflow provides a clinically acceptable and noninvasive evaluation of cardiac output under these conditions. In addition, Richard et al. (29) also reported that the direct Fick method was highly correlated with the impedance method during incremental testing ($r = 0.94$, $P < 0.01$, $n = 50$). Previously, highly significant correlations were obtained in the SV ($r = 0.84$, $P < 0.001$) and the cardiac output values ($r = 0.98$, $P < 0.001$) between the direct Fick and impedance cardiography methods in six healthy male subjects during maximal cycling exercise tests (33). Therefore, it seems that the impedance cardiography provides accurate cardiac output measurements during exercise.

Anthropometry. Height and weight were measured before and after each test. Five skinfold measurements were made (triceps, biceps, suprailiac, subscapular, and midhigh), and percent body fat was calculated using the formula of Durnin and Womersley (11).

Determination of the HRDP. The HRDP was estimated by the third-order curvilinear regression method (D_{max}) (18). In the D_{max} method, the third-order curvilinear regression curve was calculated from the original HR values vs. time, and two end points of the curve were connected by a straight line. Then the distance from point to line

was used to calculate the distance from each point on the curve to the line. The point yielding the maximal distance derived from the computation was taken as the HRDP (Fig. 1). It is recommended that the minimal HR points for the accurate determination of the HRDP should be obtained between 140 and 150 points, which were taken from HR values vs. time (20).

Statistical methods. Descriptive statistics are expressed as means and standard deviation (SD). According to the data, the normality distribution of the HRDP group population was analyzed by variance comparison by means of the Fisher Snedecor test. A Student's *t*-test for paired data was used to compare the intensity at which the HR deflexion point appeared and the power associated with the achievement of the maximal value of SV. Then to compare the values of subjects who did or did not demonstrate a HRDP in their HR-power curve (HRDP group vs. no-HRDP group), we used the nonparametric Wald Wolfowitz test. The slope between the power associated with the HRDP and that with the maximal SV (SV_{max}) value was assessed using a linear regression obtained from the least squares method for each individual. All significant differences are at the $P < 0.05$ level, unless stated otherwise.

RESULTS

The maximal values measured during the incremental test are in Table 1. All subjects showed a linear relationship between HR and SV values during the progressive exercise ($r = 0.82 \pm 0.20$, $P < 0.01$). Additionally, the HR reached its maximal value at $\dot{V}O_{2max}$. Using the D_{max} method showed a break point in the HR-workload exercise curve to $78.3 \pm 7.0\%$ of the $p\dot{V}O_{2max}$ in 72.7% of the subjects (i.e., $89.9 \pm 2.8\%$ of the HR_{max} , HRDP group, $n = 16$). The SV value also increased to $78.3 \pm 9.3\%$ of the $p\dot{V}O_{2max}$ in the HRDP group (Table 2). It then significantly decreased from 153 ± 44 to 144 ± 40 ml/beat ($P < 0.005$) until the attainment of $\dot{V}O_{2max}$ (Fig. 2). Furthermore, the power at which HRDP appeared was not significantly different from the power associated with the SV plateau in the HRDP group ($P = 0.462$). Sixty-two percent of the variance in the power associated with the SV_{max} (pSV_{max}) 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 SV_{max} 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 $pHRDP$ ($pLT = 75.1 \pm 6.7$ vs. $pHRDP = 78.3 \pm 7.0\%$ of the $p\dot{V}O_{2max}$, $P = 0.04$). The pSV_{max} value was also higher than the pLT ($pSV_{max} = 78.0 \pm 9.3$ vs. $pLT = 75.1 \pm 6.7\%$ of the $p\dot{V}O_{2max}$, $P = 0.038$) 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 SV_{max} and HR_{max} values were not differ-

Table 2. Maximal values determined during incremental test

Subjects	$p\dot{V}O_{2max}$		pLT		pSV_{max}		$pHR_{deflection}$	
	W	W/kg	W	% $p\dot{V}O_{2max}$	W	% $p\dot{V}O_{2max}$	W	% $p\dot{V}O_{2max}$
HRDP group ($n = 16$)	383 ± 66	5.5 ± 0.8	289 ± 57	75.1 ± 6.7	$300 \pm 67^{\dagger\ddagger}$	$78.0 \pm 9.3^{\dagger\ddagger}$	$300 \pm 52^{\dagger\ddagger}$	78.3 ± 7.0
No-HRDP group ($n = 6$)	402 ± 59	5.5 ± 0.8	313 ± 42	78.2 ± 3.4	381 ± 75	94.4 ± 8.6		

Values are means \pm SD; *n*, no. of subjects. $p\dot{V}O_{2max}$, power at $\dot{V}O_{2max}$; pLT, power associated with lactate threshold; pSV_{max} , power associated with SV_{max} ; $pHR_{deflection}$, power associated with the appearance of HRDP. Level of significance between *HRDP and no-HRDP groups; $^{\dagger}p\dot{V}O_{2max}$ and the other intensities; $^{\ddagger}pLT$ and pSV_{max} - $pHR_{deflection}$: $P < 0.05$.

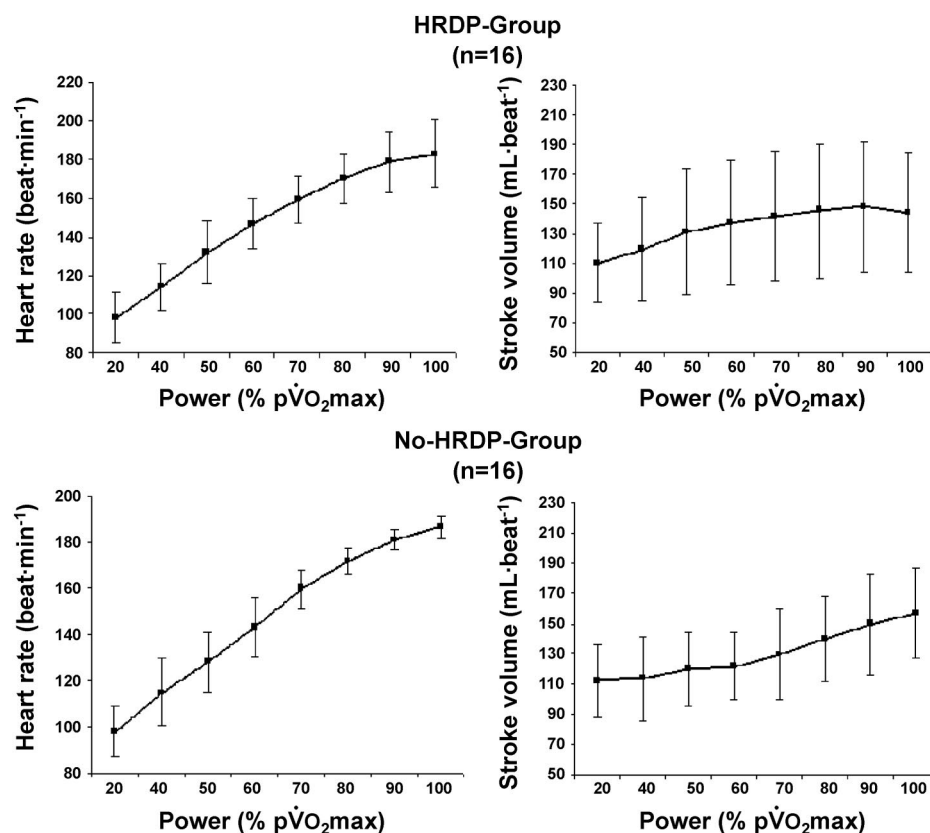


Fig. 2. SV and HR responses during the incremental test in HR deflection point (HRDP) ($n = 16$) and no-HRDP ($n = 6$) groups. $\dot{V}O_{2\max}$, power associated with the maximal oxygen uptake.

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 ($5,116 \pm 554$ vs. $5,091 \pm 562$ 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 pHRDP in most of the subjects (72.7%). Moreover, the power at which HRDP appeared is highly correlated with the pSV_{\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 $78.3 \pm 7.0\%$ of the $\dot{V}O_{2\max}$ (i.e., $89.9 \pm 2.8\%$ of the HR_{\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_{\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 $\dot{V}O_{2\max}$ (from 82.4 ± 12.2 to 100% of the $\dot{V}O_{2\max}$) in the present study. Indeed, SV did not plateau at 40–60% of $\dot{V}O_{2\max}$ 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

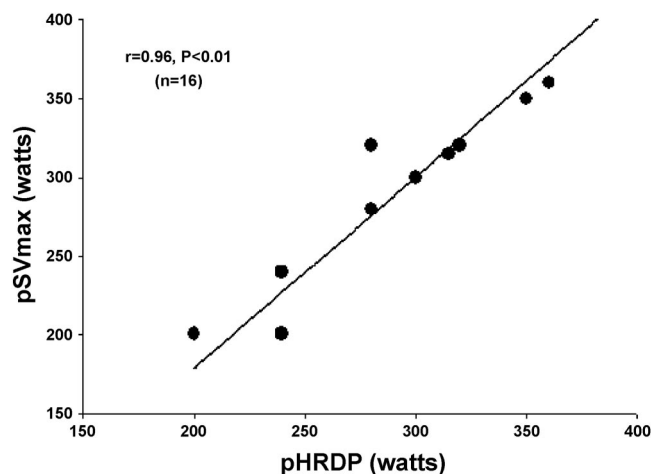


Fig. 3. Relationship between the power at which the HRDP appeared (pHRDP) and the power at which the SV reached its maximal value (pSV_{\max}) in the HRDP group ($n = 16$) during the incremental test.

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 HR_{\max} (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 pSV_{\max} was not significantly different than the $pHRDP$ in the group in which the HRDP appeared (HRDP group). Thus a high correlation coefficient was found between the pSV_{\max} 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 $\dot{V}O_{2\max}$ 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 $pHRDP$ 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 $pHRDP$ to $p\dot{V}O_{2\max}$ may reflect the decrease in the LVEF to prevent myocardial overloading. This may have a conserving effect on myocardial $\dot{V}O_2$ 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 $\dot{V}O_{2\max}$, most of the variance in the power output at the SV_{\max} 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 Fondation La Française des Jeux, and Evry Genopole.

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