Heavy cycling exercise at fixed heart rate prevent the decline of stroke volume and delay time to exhaustion in trained adolescents

Implication de la réponse du volume d’éjection systolique à l’effort dans l’atteinte des valeurs maximales de consommation d’oxygène lors d’exercice épuisant réalisé à état stable de fréquence cardiaque

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Summary
Previous studies showed an interaction between the response over time of oxygen uptake (VO₂), the drift of heart rate (HR) and the drop of stroke volume (SV) during constant workload exercise.

Objective. — To compare the responses of oxygen uptake (VO₂) and stroke volume (SV) in exhaustive exercises performed at a constant workload or at heart rate steady state.

Methods. — Nine well-trained adolescents (14.6 ± 1.1 years, 1.7 ± 0.1 m and 59.7 ± 14.8 kg) performed an incremental exercise test on a cycle ergometer to determine the maximal power associated with VO₂ max (pVO₂ max) and the power inducing SVmax (pSV-max). Two days later, they performed two continuous exercises at pSVmax (tlimpSVmax):

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Résumé
Des interactions entre les réponses de consommation d’oxygène (VO₂) à la dérive de fréquence cardiaque (FC) et la chute du volume d’éjection systolique (VES) ont été observées lors d’exercices épuisants (tlim) réalisés à puissance mécanique constante.

Objectif. — Comparer les réponses de VO₂ et de VES au cours de tlim induisant une dérive ou non de FC.

Méthode. — Neuf adolescents bien-entraînés (14,6 ± 1,1 ans, 1,7 ± 0,1 m, 59,7 ± 14,8 kg) ont réalisé, sur ergomètre : un test incrémenté de détermination des valeurs maximales de VO₂ et des valeurs de FC (FC@VESmax) et de puissance mécanique (pVESmax) associées à la mesure maximale de VES ; deux tlim : à pVESmax (tlimpVESmax : 10 min 10 s ± 6 min 41 s) et à FC@VESmax (tlimpFC@VESmax : 21 min 04 s ± 13 min 20 s ; p < 0,01). Les valeurs de VO₂, de FC et de VES ont été mesurées en continu.

Résultats. — Les valeurs maximales de lactate sanguin et de VO₂ ne sont pas différentes entre les trois exercices. Les valeurs maximales de débit cardiaque et de VES sont significativement plus élevées lors du tlimpFC@VESmax (21,3 ± 8,6 L·min⁻¹ et 112 ± 46 mL·bat⁻¹) que lors du tlimpVESmax (19,8 ± 7,6 L·min⁻¹ et 102 ± 40 mL·bat⁻¹ ; p < 0,05). À l’inverse, les valeurs maximales de FC mesurées lors du tlimpFC@VESmax (192 ± 9 bat·min⁻¹) sont significativement plus faibles que celles enregistrées lors du tlimpVESmax (196 ± 7 bat·min⁻¹ ; p < 0,05) ou du test incrémenté (197 ± 6 bat·min⁻¹ ; p < 0,05). La durée de temps de maintien était plus corrélée aux temps d’atteinte des valeurs maximales de VES lors du tlimpFC@VESmax (R = 0,70 ; p = 0,03) que lors du tlimpVESmax (R = 0,61 ; p = 0,08). Les valeurs de FC, VES et VO₂ chutaient significativement avant l’arrêt des deux tlim (p < 0,05).

Conclusion. — L’atteinte des valeurs maximales de VO₂ et de débit cardiaque peut être induite par des adaptations cardiaques dépendantes de la modalité d’exercice imposée : puissance mécanique ou fréquence cardiaque constante.

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1. Introduction

After a rapid increase at the onset of incremental or constant exercise, arterial–venous oxygen difference ([a – \(
\dot{V}O_2\) diff] and stroke volume (SV) values remained relatively constant until exhaustion in non-active subjects [1,2]. The maximal heart rate value (HR) may also reach values three times higher than at rest, while SV may only double at the maximal aerobic exercise [3]. Therefore, the increase in the maximal HR values explained the increase in cardiac output (\(Q\)) until to exhaustion in sedentary people [4]. In contrast, recent studies performed with well-trained aerobic subjects showed that SV value did not plateau at 40–60% of maximal oxygen value (\(\dot{V}O_2\) max) but increased almost until \(\dot{V}O_2\) max (93 ± 6% of \(\dot{V}O_2\) max) [5]. Furthermore, the elevation of SV could only play a significant role in producing the increase in \(Q\) at the onset of exercise. In fact, dynamic muscular contractions facilitated the increase in SV by the rise of venous blood return during the transition from rest to exercise [6]. Else, both increases in HR and SV until the attainment of p\(\dot{V}O_2\) max were responsible of the attainment maximal \(Q\) (Q max) at the end of the incremental exercise.

In healthy trained subjects, Billat et al. [7] also showed that the SV response was the main factor of the increase in \(\dot{V}O_2\) at the onset of constant exhaustive exercise performed above the lactate threshold and, then, induced the attainment of \(\dot{V}O_2\) max [8]. However, in most well-trained athletes, maximal SV tend to level off during maximal efforts [9]. Fritzsche et al. [10] showed that the decline in SV is related to the increase in HR to maintain \(\dot{V}O_2\) value during prolonged exercise performed in a neutral environment. Nonetheless, a significant correlation between the magnitude of the SV
decrease and the end-exercise decrease in $\dot{V}O_2$ was recently observed [11]. Previously, Nobrega et al. [12] showed that the increase in SV compensated for the lack of HR response in the patients to produce the normal increase in blood pressure during mild static exercise (50% of maximal voluntary contraction). CO was also increased by an elevation in SV of 16 mL when HR was fixed at a rate found at peak exercise [12]. Foster et al. [13] moreover reported a continuous rise in left ventricular ejection fraction (LVEF) during steady state exercise intensity (inducing maximal value of LVEF during an incremental test). The hemodynamic data obtained in previous studies suggested that the modulation of cycling mechanical power for maintaining the HR value associated with the maximal SV value initially determined during an incremental test, could prevent the decrease in SV and the end-exercise $\dot{V}O_2$ drop and then, delay the exercise exhaustion. Therefore, the purpose of this study was to compare the $\dot{V}O_2$ and SV responses during exhaustive cycling exercise performed under the control of mechanical power to maintain a fixed heart rate value ($t_{\text{lim}}_{HR@SV_{\max}}$) and a constant mechanical power output ($t_{\text{lim}}_{pSV_{\max}}$) associated with the intensity for which the maximal stroke volume was reached during the initial incremental test. We hypothesized that exercise can be sustained for a longer duration when heart rate is controlled to prevent a stroke volume decline.

2. Methods

2.1. Subjects

Nine healthy well-trained pubertal subjects (7 males and 2 females) participated in this study. These subjects were regional elite triathletes. All subjects were free of cardiac and pulmonary disease. Individual morphological features of the subjects (mean ± SD) are 14.6 ± 1.1 years, 1.7 ± 0.1 m and 59.7 ± 14.8 kg. Each subject was accustomed to the experimental procedure prior to the study. Before participation, they were informed of the risks and stresses associated with the protocol, and gave a written voluntary informed consent in accordance with the local guidelines.

2.2. Protocol

All subjects performed exercise bouts in the upright position on an electronically braked cycle ergometer (ERGOLINE 900, Hellige, Markett, USA), two to three hours after a light breakfast, in an air-conditioned room (STP: 20.0 ± 0.5°C, 745 ± 4 mmHg and relative humidity: 50%). Seat and handlebar heights were set for each subject and kept constant during all tests. Each subject freely chose its pedaling rate frequency.

2.3. Incremental test

The initial power was set 40 watts and was increased by 20 watts every 3 min and 20 watts every 2 min after lactate threshold for the total exercise duration not exceeding 20 min. VO$_2$ max was defined as the highest oxygen uptake obtained in two successive 15 seconds-interval periods during this incremental test with a respiratory exchange ratio greater than 1.1 ($RER = VCO_2/\dot{V}O_2$) and a HR peak at least equal to 90% of the age-predicted maximum. A plateau of VO$_2$ was identified if the VO$_2$ of the latest stage was not greater than the previous one by 2.0 mL·min$^{-1}$·kg$^{-1}$ [14]. pVO$_2$ max was defined as the lowest power associated that elicited VO$_2$ max [15]. The power and the HR value associated with maximal SV value and the power associated with the lactate threshold were estimating during the incremental test by two independent experimenters.

2.4. Constant work rate and heart rate steady state exercise

After a 10-min warm-up at 30% of pVO$_2$ max and three minutes at rest, the work rate was increased within 45 s to pSVmax and each subject performed in both conditions: an all-out constant load exercise at pSVmax (interpolated from the incremental test), and an all-out steady state HR exercise at a HR value associated with pSVmax during the incremental test. All subjects were given verbal encouragement throughout each trial. Time to fatigue was the time at which the subject was no longer able to pedal at a given power output ($t_{\text{lim}}_{pSV_{\max}}$ and $t_{\text{lim}}_{HR@SV_{\max}}$ respectively).

2.5. Steady state heart rate control

After the increase within 30 s and 3 min bout at constant pSVmax intensity, experimenter was enabled Physio-Trainer function (COSMED systems, Roma, Italy). Physio-Trainer software contained the handshake protocols to communicate with Ergoline 900 ERG, and to allow a manual or automatic ergometer control. The software controlled dynamically the ergometer workload in order to maintain the desired HR value, according to a feedback algorithm based on the target use, the actual HR, and the dynamics of its changes. The theoretical basis for this technique and its application for exercise testing have been previously described [16]. The maintenance of constant pSVmax workload during the first 3 min of exercise allowed to avoid large variations in power during the early moments of exercise before VO$_2$ and SV had stabilized.

2.6. Gas and cardiac parameters measurements

$\dot{V}O_2$, $\dot{V}CO_2$ and minute ventilation ($\dot{V}E$) were performed throughout each test using a telemetric system ($K_{b}$, COSMED, Roma, Italy) [17]. Expired gases were measured, breath-by-breath, and averaged every 5 seconds. In addition, we used a bio-impedance method to determine stroke volume (SV), heart rate (HR) and cardiac output (Q) (Physioflow, Manatec Type PF05L1, Strasbourg, France). The theoretical basis for this technique and its application and validity for exercise testing have been previously described [18,19] and used in previous studies [16,18,19]. For this experiment, SV, HR, and Q were recorded beat-by-beat and averaged over each breath during each test.
2.7. Measurement of blood lactate concentration

A capillary blood sample was obtained from the ear tip and analyzed for blood lactate concentration (Lactate Pro LT, ARKAY INC., Kyoto, Japan) [20]. The samples were taken at rest, every three minutes during the all-out exercise, at the end of the cycling exercise test and at two and four minutes during recovery. For the incremental test, the lactate threshold (LT) was defined as the VO₂ corresponding to the starting point of an accelerated lactate accumulation and expressed in % of VO₂max. The theoretical basis for this method and its validity for exercise testing have been previously described and used in previous studies and training intensity prescriptions [15].

2.8. Statistical methods

Descriptive statistics are expressed as mean and standard deviation (±SD). According to the data, the normality of the data was analyzed by variance comparison by means of the Fisher Snedecor test. Statistical comparisons over time of the physiological variables (VO₂, Q, HR and SV) were made using a one-way repeated measure analysis of variance (ANOVA). A non-parametric Wilcoxon test for paired data was used to compare the performance of subjects (i.e. the mean power output and time to fatigue) and the physiological responses in constant workload versus HR steady state exercises at 3 min and at exhaustion. Scheffe Post hoc tests were carried out when justified by ANOVA. The slope between the magnitude of heart rate drift (HR) in constant pSVmax and the variability of power (power) to exhaustion at HR@SVmax exercise was assessed using a linear regression obtained from the least square method for each individual. All significant differences were set at P < 0.05.

3. Results

The maximal values measured during all exercise tests are given in Table 1. During constant exercise, the control of the power output with HR induced an oscillation of the power output throughout the tlimHR@SVmax test (Fig. 1). Hence, the mean power was lower in tlimHR@SVmax compared to the tlimpSVmax (213 ± 70 vs. 239 ± 62 W, P < 0.05) and tlimHR@SVmax was longer compared to tlimpSVmax (21 min 04 s ± 13 min 20 s vs. 10 min 10 s ± 6 min 41 s, P < 0.01). Maximal values of blood lactate and VO₂ were not different between the three exercises, but maximal cardiac output and SV values were higher in tlimHR@SVmax (21.3 ± 8.6 L min⁻¹ and 112 ± 46 mL beat⁻¹) compared to tlimpSVmax (19.8 ± 7.6 L min⁻¹ and 102 ± 40 mL beat⁻¹, P < 0.05). Conversely, tlimHR@SVmax induced a lower maximal HR value (192 ± 9 beat·min⁻¹ vs. tlimpSVmax: 196 ± 7 beat·min⁻¹, P < 0.05 and vs. incremental test: 197 ± 6 beat·min⁻¹, P < 0.05). The time to reach SVmax was significantly correlated with tlimHR@SVmax (R = 0.70, P < 0.05) compared to tlimpSVmax (R = 0.61, P = NS). Finally, HR, SV and VO₂ values significantly fell before both exercise exhaustion (P < 0.05).

4. Discussion

The focus of this study was to compare the responses of VO₂ and SV in exhaustive exercises performed at HR steady state compared to a constant workload exercise. The main findings of this study are that:

- the control of HR induced a decrease in the power output and a longer time to fatigue;
- though the tlimpSVmax test elicited maximal values of HR while tlimHR@SVmax did not, all subjects reached their maximal Q and VO₂ with a higher stroke volume value in tlimHR@SVmax compared to tlimpSVmax test;
- the time to reach the maximal stroke volume value was correlated with the time to fatigue in both conditions.

Cardiorespiratory response to exercise and the metabolic profile of the muscle appear to change with growth and development. So, Fawkner et al. [21] suggested that the significantly shorter time constant of oxygen uptake kinetics in children than adults may reflect an enhanced potential for
oxidative metabolism in children. However, in 14 children and 16 young adults with a similar aerobic aptitude, who performed a progressive cycle exercise, Vinet et al. [22] reported no significant difference in mean sub-maximal and VO₂ max between children and adults. It has been reported that SV was the main determinant of the VO₂ response during exhaustive supra lactate threshold exercise [7]. The SV response to exercise also depends on changes in the cardiac filling, the intrinsic myocardial contractility and the left ventricular after-load. So, the lower cardiovascular responses to exercise in children compared to adults may be attributed to the children’s smaller heart size. Hence, if adult demonstrated larger absolute values of SV and Q at all levels of intensity compared to children, the differences disappeared when SV and Q values were related to body surface area [22]. Whatever the exercise intensity or at rest, Nottin et al. [23] did not also show a significant difference in SV, Q and left ventricular dimensions when they were scaled to body surface area, between 17 boys (11.7 ± 0.6 yr) and 23 young adult men (21.2 ± 2.7 yr) with a similar aerobic potential. Moreover, Proctor et al. [24] did not show a significant difference between the absolute Q value with a given VO₂ between young and older endurance trained subjects for a range of intensities between 40 to 90% of VO₂ max. The slopes of Q – VO₂ relationship across submaximal levels of cycling exercises were also similar among all groups. Hence, these findings suggested that age has not a significant impact on the Q – VO₂ relationships during cycling exercise in endurance trained subjects [22,24]. The SV patterns, as well as its underlying mechanisms, were also not age-related during an upright maximal cycling exercise [23]. However, it has been reported that older trained subjects presented a reduced ability to maintain SV value at exercise intensities above 70% of VO₂ max compared to younger athletes [23]. Because of the similar patterns of SV and the greater ability to maintain a high SV value in the young compared to old subjects, we choose 9 well-trained children to examine the effect of a prolonged exercise performed at the steady state of HR value associated at the maximal SV value during a progressive cycling exercise. During dynamic exercise, it has been also reported that heart rate response at the onset of exercise is faster in children than adults. Although a higher HR and total peripheral resistance values, SV and Q values were lower in children compared to adults. In fact, the SV at a given work rate was closely related to left ventricular mass. So the smaller heart mass was responsible of the
lower SV and $\dot{Q}$ values in children compared to adults [25].

However, the lower $\dot{Q}$ in children was compensated by a significant higher $(\alpha - \gamma)O_2$ diff to achieve the similar oxygen uptake than adults with a similar aerobic potential [23,25]. In the same way, trained endurance child and adult athletes demonstrated a greater maximal SV and $\dot{Q}$ indexes compared to untrained children and adults. Rowland et al. [26] reported a progressive rise in SV in 8 pre-pubertal distance runners while SV remained stable below low intensities in 14 untrained boys. Previously Oyen et al. [27] have showed that trained children increased their $\dot{Q}$ in the first minutes of exercise by a simultaneous increase in HR and SV values whereas the $\dot{Q}$ increase was mainly due by elevating heart rate in untrained subjects. Thus, it has been suggested that physical training causes quick adaptations of the left ventricular function to exercise in adults as well as children [27].

On the other hand, Nóbrega et al. [28] showed, in 10 healthy men, a simultaneous increase in mean arterial pressure and $\dot{Q}$ with a decrease in total peripheral resistance during the first 10 heartbeats after the onset of 20 s trials of upright cycling exercise (30 W; 60 rpm). Initially, the $\dot{Q}$ response was accounted for by the rapid heart rate acceleration and, after 15 cardiac cycles, by an increase in stroke volume, which occurred with a decrease in left ventricular end-systolic volume and no change in end-diastolic volume. In the present study, all subjects reached their maximal $\dot{Q}$ and $VO_2$ in both conditions. Furthermore, SV remained stable or increase almost until the exhaustion. Previously, it has been observed that both adults and children exhibit a gradual heart rate increase and a decrease in SV during prolonged constant workload exercise [25]. Coyle and Gonzalez-Alonso [29] suggested that the cardiovascular drift, characterized by a progressive decline in stroke volume after 10–20 min of exercise, is primarily due to increased HR rather than a progressive increase in cutaneous blood flow as body temperature rises. In the past, Fritzsche et al. [10] have been questioned about the relation between the declines in SV, the increases HR and cutaneous blood flow in seven active men cycled for 60 min at approximately 57% of $VO_2$ max in a neutral environment (i.e., 27 degrees C, <40% relative humidity). These authors showed that an oral ingestion of beta-1-adrenoceptor blocker atenolol (i.e., 7 mg) at the onset of exercise prevented the HR increase and the decline of stroke volume during prolonged exercise [10]. Therefore, the decline in SV in a neutral environment is related to the increase in HR to maintain $\dot{Q}$ value during prolonged exercise [10]. Krip et al. [30] also suggested that the changes in SV and $\dot{Q}$ consequent to alterations in blood volume were attributable primarily to changes in diastolic function. Previously, Foster et al. [13] showed that the left ventricular ejection fraction (i.e. the ratio between the left ventricular end-systolic and end-diastolic volumes) increased until the end of exercise performed at an intensity soliciting the maximal value of left ventricular ejection fraction during the progressive exercise. Furthermore, our results showed that the control of the mechanical power by heart rate steady state induced the attainment of a higher stroke volume value in $t_{limHR@SVmax}$ than in $t_{limPSVmax}$ test. During a strenuous prolonged exercise with 5% of power variation, Liedl et al. [31] did not show a significant difference in mean HR and $VO_2$ values compared to a constant workload test. Furthermore, Fukuba et al. [32] did not report a significant difference in the heart rate responses between 7 pre-pubertal and 5 adult subjects engaged in a sinusoidal work rate exercise. Previously, Nóbrega et al. [12] have been interested about the effect of the heart rate steady state on the stroke volume response during static one-legged exercise at 20% of the maximal voluntary contraction for 5 min bouts. Their results showed a significant increase in cardiac output due to a rise of stroke volume during static exercise at fixed heart rate values. The decrease in end-systolic value from 55 to 38 mL during fixed HR at peak exercise suggested a greater left ventricular contractility, which mediated the increase in SV in this condition. Therefore, the mechanism eliciting this SV response was an increased left ventricular contractility with no change in end-diastolic volume during exercise under control of HR [12]. So, if peripheral vasodilatation plays an important role in the rise in SV at the onset of exercise, the increase in HR acts to maintain a stable SV during severe constant workload exercise. The mechanical power oscillation to maintain a steady state of HR could induce a increase in diastolic filling time and a shorter systolic ejec- tion time, both responsible to the higher SV value during the $t_{limHR@SVmax}$ compared to the $t_{limPSVmax}$ in the present study.

5. Practical applications

The current general prescription of training based on power output has been potentially inaccurate to optimize cardiac function in contrast with fixed heart rate exercise, which induced a higher SV value. Coaches also should consider that heart rate-based intervals performed under stable conditions may provide an additional advantage over power-based intervals on cardiac function.

6. Conclusion

In conclusion, the cardiac responses to exercise are influ- enced by a complex interplay of changes in heart rate and left ventricular function. Factors influencing left ventricular diastolic filling may determine the maximal values of cardiac output and oxygen uptake. The higher stroke volume value during the $t_{limHR@SVmax}$ suggested a greater diastolic filling when the mechanical power was control by the heart rate steady state value. Hence, the greater left ventricular contractility was responsible to the $\dot{Q}$ max and $VO_2$ max attainment during dynamic exercises performed until exhaustion.

Disclosure of interest

The authors declare that they have no competing interest.

References


\[ \dot{V}O_2 \text{ and stroke volume during constant power and heart rate steady state trials} \]


