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Review article

Effects of All Out-Cycling Bouts on Left Ventricular Function in Master Cyclists

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Abstract

The effects of different strenuous cycling bout's time of training regimes on left ventricular function were examined in male healthy cyclists at the national level (57.3±5.2 yrs.).

Methods: 43 Subjects were randomly assigned to perform four different strenuous cycling bouts in an increasing or decreasing order: 30, 60, 90, 120 sec or 120, 90, 60, and 30 sec on an electrical bicycle at a resistance corresponding to 80% of subjects' maximal VO2 test. All subjects performed both the increasing and decreasing orders in two separate visits. Recovery times between exercises bouts in the increasing schedule were 3, 4 and 5 minutes compared to 5, 4 and 3 minutes for the decreasing schedule respectively.

Results: There were significant interactions between bouts and schedule in heart rate, lactic acid accumulation, systolic and diastolic volumes. Cardiac responses indicate different response mechanisms of cardiac function during the two schedules. The decreasing schedule was characterized by higher mean heart rates ($p<0.05$), lower mean stroke volume ($p<0.05$) and lower mean end diastolic volumes ($p<0.05$). In both schedules mean end systolic volume and mean end diastolic volume decrease significantly ($p<0.05$) with increased time of performance.

Conclusions: These data suggest that master cyclists, had a higher lactic acid, during the decreasing schedule with venous return is impeded. The lower venous return might have been caused by inadequate vasodilatation during early phase of decreasing order. Thus, in master cyclists, the decreasing schedule triggers better cardiac training responses than the increasing schedule.

Keywords: Training intensity; cardiac output; left ventricular contractility; left ventricular volumes

Introduction

Echocardiography studies have been utilized in a variety of settings to evaluate left ventricular function and contractility. Left ventricular function indices measured previously have been shown to increase with aerobic exercise [1], primarily due to decreased afterload [2]. Anaerobic exercise brings about oxygen debt, which increases blood lactate concentration, in direct associated with increase in maximum performance [3] and exposes the subjects to a very high degree of sudden strenuous all-out exercise relying primarily on the glycolysis pathway [4, 5]. Accordingly, performance time can be limited by lactic acid levels in the blood and active muscles [6, 7]. Since competitive biking at high degree the training regimes include speed endurance performances to volitional exhaustion and anaerobic glycolysis during repeated exercise [8, 9], this may alter left ventricular global performance [10].

Two main factors influence the physical performance with aging: a) the decrease in cardiopulmonary function and b) a progressive decline in muscle mass and strength that occurs with aging [11]. Consequently, the maximal oxygen uptake and power ability decrease, irrespective of lifestyle because of genetic factors, thereby severely limiting body function, quality of life, and longevity [12, 13]. However, structured endurance training after 40 years of age is powerful enough to induce beneficial cardiovascular adaptations in later life [14].

To understand the cardiac limitations in master athletes, it is important to evaluate the master's ability to perform a highly intense effort at different lactic acid levels, brought about by different schedules of identical intensities and time performance. Therefore, the purpose of the present study was to evaluate the influence of two strenuous training regimes which differ in the sequencing of the efforts on left ventricular function in well-trained master cyclists at the national level.

Methods

Subjects: 43 well-trained master healthy male cyclists, at the national level (57.3±5.2 years) volunteered to participate in this study. A written consent form, approved by the Clinical Science Center Committee on human subjects was obtained from all subjects, after being fully informed of the details and possible discomforts associated with the experimental protocol.

Procedures and measurements: Each subject reported to the laboratory three times. Sessions were spaced by at least 48 hours and on the average by intervals of one week. The first session was devoted to introducing the subjects to the general scope of the study, risk stratification via health history questionnaire and to define maximal work capacity by means of graded stress test up to VO2 peak and accustoming the subjects to strenuous efforts on an electrical cycle ergometer.

Following warm up, subjects underwent a graded maximal bicycle exercise test on an electrical cycle ergometer (Lode Groningen, Holland). Maximal tests were terminated according to the following criteria: a) leveling off or no further increase in VO2 with increasing work rate, b) attainment of the age predicted maximum heart rate, c) respiratory exchange ratio > 1.1, and d) when the subject could not keep up with the load, according to the guidelines of the American College of Sports Medicine [10]. Following 10 minutes of sitting rest, in order to accustom the subjects to strenuous exercises each subject performed two strenuous bouts on the electrical cycle ergometer (Lode Groningen, Holland) for 30 and 120 sec. at a predetermined load which correspond to 80% of its maximal VO2peak test, with ten resting minutes separating the two bouts. During the 2nd and 3rd sessions at rest and during exercise all measured variables were taken in the sitting position. During one of the following sessions, subjects performed either schedule of increasing time of work in the order of: 30, 60, 90 and 120 sec or of decreasing order: 120, 90, 60, and 30 min randomly assigned to. Resting times between cycling bouts in the increasing schedule were 3, 4, and 5 minutes, and 5, 4 and 3 minutes for the decreasing schedule respectively.

A 12-lead electrocardiogram and heart rate were continuously monitored throughout the experiment (V5). A 25 µl fingertip blood sample was taken at rest and at the end of the 2nd minute post any sprint for the determination of lactic acid response to strenuous exercise. The sample was immediately transferred to a micro-tube containing 100 µl of 7% perchloric acid. The tubes were centrifuged after standing at least for an hour. Twenty- microliter aliquots of the supernatant were subsequently used for lactic acid analysis on the Analox LM3 analyzer (Analox Instruments, England; Reagent Kit # GMRD-071). Echocardiographic data processing: Although reliable measurements have been performed in multiple studies using conventional techniques, this study differs by its unique echocardiograph approach for measurements which were taken at peak exercise from 5-chamber view with the subjects seated and strapped to a wall. This helped to minimize movement of the upper body thus, enabling clear and reliable imaging and blood pressure measurement even at peak effort [15]. 2-D, echocardiographic and M-mode images were performed at rest and at peak exercise utilizing Vingmed 725 Sonotron and Sony recorder equipped with 2 and 3 MHz transducers. The diameters of the aorta were determined by two-dimensionally directed M-mode. The left atrium was measured from the parasternal long-axis view. At rest, left ventricular end-diastolic and end-systolic diameters and intraventricular septum and left ventricular posterior wall thicknesses were measured from the parasternal long and short-axis views as well as from 4 and 5 chamber views, just below the mitral valve level, according to the recommendations of the American Society of Echocardiography [16]. At peak exercise, due to the short time available for measurements left ventricular volumes and ejection fraction were determined using Simpson's rule from apical 5 chamber view [17].

All echocardiographic studies were performed with the subjects in the sitting position at rest, and at peak efforts. The probe was hand held and directed to a marked point from which the resting data were obtained. The beam was directed to the aortic valve outflow tract in the 5-chamber view, or from the suprasternal approach for those subjects in whom adequate imaging of 5-chamber or parasternal long axis views was not obtained. To assess the objectivity of the echocardiographic readings, all recordings were evaluated by two independent experts. A high correlation (r = 0.91) was found for inter-observer reliability.

Calculations: At rest and exercise cardiovascular variables were computed as follows:

Stroke volume was computed as the difference between left ventricular end diastolic volume and end systolic volume.

Cardiac output was determined as the product of heart rate and stroke volume.

Ejection fraction = [(end diastolic volume - end systolic volume)/ end diastolic volume] x 100%.

Contractility (ratio) was defined following the division of systolic blood pressure by end systolic volume.

Statistical Methods

Each variable was analyzed by a two way ANOVA (increased/decreased schedules by exercise time) with repeated measures on both factors. Tukey HSD procedure was employed for post hoc evaluation of overall significant main effects and interactions. In addition, individual patterns of response were investigated in order to assess the generalization characteristics of the group curves.

Results

Subjects' physical characteristics are presented in table 1 Means ± SD, while, in

Variable	Mean ± SD
Number of subjects	43
Age (years)	57.3±5.2
Weight (kg)	77.4 ± 2.2
Height (cm)	175.1 ± 5.2
Fat (%)	11.0 ± 1.0
Aortic dimension (cm)	2.4±0.4
Inter-ventricular septum thickness(cm)	1.0+0.1.0
End diastolic dimension (cm)	5.1±0.2
End systolic dimension (cm)	3.5±0.2
Posterior wall thickness (cm)	1.0±0.1

Table 1. Subjects physical characteristics (Mean ± SD)

Table 2 values for left ventricular function and hemodynamic variables at rest are presented. Left ventricular function means and hemodynamic variables during increased and decreased schedules are presented in table 3. Comparisons of mean end diastolic volume and end systolic volume between the increasing and the decreasing schedules are presented in figures 1 and 2 respectively.

Systolic blood pressure (mmHg)	108.0±5.3
Diastolic blood pressure (mmHg)	74.1±4.7
Total peripheral resistance (dynes▪s^{-1}▪cm-^{5})	989±168
Heart rate (beats▪min^{-1})	66.8±5.8
Stroke volume (mL)	72.9±6.0
Cardiac output (L▪min^{-1})	4.9±0.5
End diastolic volume (mL)	123.8±9.7
End systolic volume (mL)	50.9±3.1
Ejection fraction (%)	58.9±6.7
Systolic pressure/volume (ratio)	2.2±0.3
Lactic acid (mmol▪L^{-1}) actic	1.4±0.3

Table 2. Left Ventricular Function And Hemodynamics Responses At Rest (Mean±Sd)

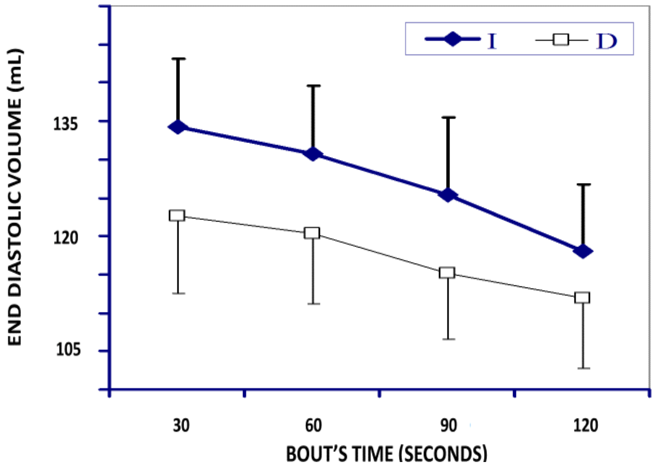

Figure 1. End Diastolic Volume (EDV) during increasing (I) and decreasing (D) training.

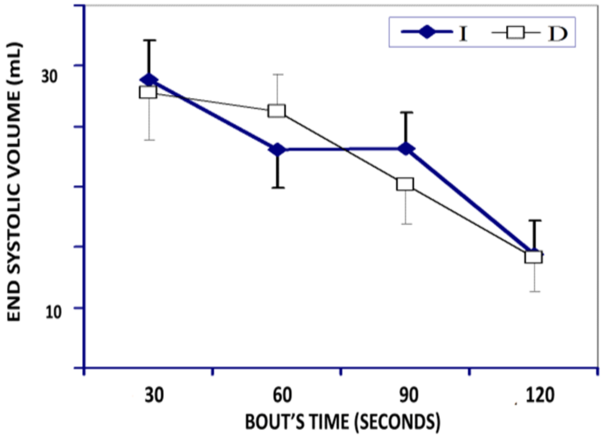

Figure 2. End Systolic Volume (ESV) during increasing (I) and decreasing (D) training.

Variable		**30 sec**	**60 sec**	**90 sec**	**120 sec**	**Sig.**
SBP (mmHg)	I	166±4	179±4.3	187±6.1	200±4.0	b
	D	176±5	184±4	190±5	199±4	
DBP (mmHg)	I	80±4	80±3	83±4	84±3	
	D	78±4	79±4	82±4	82±5	
TPR (dynes▪s^{-1}▪cm-^{5})	I	476±65	452±77	450±69	492±61	
	D	467±70	480±66	472±74	484±58	
HR (beats▪min^{-1})	I	169±7	180±6	196±4	199±5	a
	D	191±7	198±5	197±5	193±4	
SV (mL)	I	104±4	108±4	102±5	102±5	b
	D	98±4	95±4	98±5	97±5	
Q (L▪min^{-1})	I	18±2	20±3	21±3	20±3	
	D	19±4	19±4	20±4	20±5	
EF (%)	I	76±3	81±3	79±3	82±4	
	D	74.±4	75±3	80±2	82±3	
P/V (ratio)	I	6±.1	8±1	8±1	14±2	a
	D	6±1	7±1	10±1	14±2	
LA (mmol▪L^{-1})	I	4.8±1	6.8±2	9.6±2	11±2	a
	D	10.±3	10.5±3	10.6±2	9.7±2	

[a] - Significant interaction effect, $\alpha < 0.05$; b - Significant difference between schedules, $\alpha < 0.05$; c- significant differences between bouts 0.05; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; Total peripheral resistance = TPR; HR = heart Rate; SV = Stroke Volume; Q = Cardiac output; EF = Ejection Fraction; LA = Lactic Acid. P/V = Systolic/pressure ratio; I = increased schedule; D = decreased schedule

Table 3. Left Ventricular Function During Increased And Decreased Schedules (Mean±Sd)

There were significant interactions ($p < 0.05$) between bouts and schedule in heart rate and lactic acid accumulation, systolic and diastolic volumes. This indicate that during the increasing schedule means of heart rate, and lactic acid were gradually increased while in the decreasing schedule mean levels were maintained at a high level throughout all the bouts. Mean stroke volume is significantly higher in the increasing schedule than in the decreasing schedule. ANOVA does not reveal any significant main effects or interaction effect in mean cardiac output, diastolic Contractility and total peripheral resistance with means being similar for the two schedules and stable across conditions.

A negative relationship was evident between means of end diastolic volume and end systolic volume with exercise time, without interaction effects. However, there is a significant ($p < 0.05$) schedule effect for end diastolic volume with consistent lower mean values in the decreasing schedule than in the increasing schedule. The two schedules are not significantly different with respect to end systolic volume. There is a significant difference between exercising time in mean ejection fraction with higher mean values in the 120 sec. bouts than in the 30 sec. exercise bouts.

Discussion

Reliable measurements have been performed in multiple studies using conventional techniques, this study differs by its unique echocardiography approach for measurements which were taken at peak exercise from 5-chamber view with the subject seated on the bicycle and strapped to a wall [1, 18]. This helped to minimize movement of the upper body, thus enabling clear and reliable imaging and blood pressure measurement even at peak effort.

Although total time work performed and power output were equal for both schedules (increased and decreased), this study indicated that these two training regime schedules differ in left ventricular function responses. In addition, the present study suggests that there is a strong link between the training regime schedule and the significant differences in physiological responses.

Strenuous efforts in both schedules did not produce the expected increases in the left ventricular volumes, ejection fraction, stroke volume, cardiac output and the decrease in total peripheral resistance from resting values compared to responses observed during aerobic-type effort [19]. While all-out anaerobic exercise is a dynamic type of exercise, the cardiovascular responses resembled those seen during isometric exercise [19].
The mechanics of left ventricular contractions include the concept of the interrelationship of force, length, velocity, and time. The pressure volume loop reflects a smaller end-systolic volume and a higher end-systolic pressure, so that the slope of the pressure volume relationship moves upward and to the left. The pressure volume relationship is the most reliable index for assessing myocardial contractility in the intact circulation and is almost insensitive to changes in preload and afterload [20]. The extent of myocardial fiber shortening is a reflection of the interaction between the initial fiber stretch (preload) load-opposing shortening (afterload), and the intrinsic contractile state [21, 22, 23].

Although left ventricular contractility was increased in the master athletes, during both anaerobic schedules, it produced lower values of ejection fraction, stroke volume cardiac output and total resistance, compared to those seen during aerobic exercise [1, 18]. Such responses could be the results of known inverse relationship between left ventricular performance to the greater afterload [24]. We speculate that compared to aerobic bouts, the diminished left ventricular volumes and function in the present study during strenuous anaerobic exercise may be due to the significant increase in catecholamine levels and consequently, it reduces the myocardial beta receptors responsiveness which in turn attenuates left ventricular function. This response may be the result of the sympathetic nervous system response to strenuous exercise [25, 26]. Second possible mechanism involved during strenuous anaerobic exercise, is the interplay between the capability of the heart to distend, left ventricular contractility state of the myocardium and the arterial load [27].

The early establishment of high lactic acid levels in the decreasing schedule elicits different responses in left ventricular function end diastolic and end systolic volumes compared to that observed during the increasing schedule. Both schedules did not differ in cardiac output, however; the production of similar cardiac output levels was achieved through different interplay between heart rate and stroke volume. The decreasing schedule had a lower stroke volume than that of the increasing schedule and a higher heart rate maintained throughout the schedule. This response can be explained by the influence of the higher levels of lactic acid during the decreasing schedule on the chemo-receptors which increased heart rate through the autonomic nervous system, which in turn reduces left ventricular filling time and hence, lowering end diastolic volumes [28].

The assumed lack of autoregulation mechanism during the strenuous bouts may explicate the moderate increases in ejection fraction and stroke volume, compared to the maximal ejection fraction and stroke volume that subjects can reach during dynamic exercise when achieving VO2max [1]. In addition, the combined short duration of the anaerobic bouts performed was insufficient in facilitating an appropriate adjustment of the circulatory system, resulting in low stroke volume, ejection fraction and maximal heart rate thus the relatively lower cardiac output achieved compare to the higher cardiac output at peak aerobic exercise.

Conclusions

The present study indicates that master athletes performing an all-out anaerobic exercise with similar distance and output, under different schedules training regimes, a different interplay exists between stroke volume and heart rate, due to the impeded venous return. This response may be attributed to the sympathetic system i.e. chemo-receptors' sensitivity to lactic acid accumulation. Thus, the decreasing schedule triggers better cardiac training responses than the increasing schedule. Consequently the strategy to utilize the decreasing schedule training may induce a higher training stimulus for the same power and total work output than during the increasing schedule.

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