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ABSTRACT

The purpose of this study was to characterize the hemodynamic responses during recovery from moderate intensity exercise in young Black normotensive males. Nineteen normotensive men (age 24-26 years) walked continuously on a treadmill for 40 minutes at 50-60 percent heart rate reserve. Following exercise, blood pressure (by auscultation) and hemodynamic variables (impedance cardiography) were monitored for 2 hours in a quiet room with subjects in a seated posture. During the initial 40 minutes of recovery, cardiac output was significantly greater than the pre-exercise baseline. This was mediated by heart rate, which was elevated above the pre-exercise baseline for 75 minutes. Mean systolic blood pressure (SBP) was reduced from 30 minutes to 2 hours post-exercise, compared to the pre-exercise baseline. Total peripheral resistance was depressed for 60 minutes compared to the pre-exercise baseline, but was fully recovered by 2 hours. It was concluded that in this group of young Black normotensive males, moderate intensity aerobic exercise led to a prolonged reduction in SBP through relative changes in cardiac output and total peripheral resistance. These observations have not been consistently observed by other investigators in normotensive white males under similar experimental conditions. (Contains 26 references.) (Author/ND)

Title: Hemodynamic Responses Associated with Post-exercise Hypotension in Normotensive Black Males.

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Abstract

The purpose of this study was to characterize the hemodynamic responses during recovery from moderate intensity exercise in young black normotensive males. Nineteen normotensive men (age 25.0 ± 1.0 yrs, $\dot{V}O_{2peak}$ 48.8 ± 1.5 $ml \cdot kg^{-1} \cdot min^{-1}$) walked continuously on a treadmill for 40 minutes at 50-60% heart rate reserve. Following exercise, blood pressure (by auscultation) and hemodynamic variables (impedance cardiography) were monitored for 2 hours in a quiet room with subjects in a seated posture. During the initial 40 minutes of recovery, cardiac output, (Q) was significantly greater than the pre-exercise baseline ($p < 0.05$). This was mediated by heart rate, which was elevated above the pre-exercise baseline for 75 minutes. Mean systolic blood pressure (SBP) was reduced by at least 7 mm Hg from 30 minutes to 2 hours post-exercise, compared to the pre-exercise baseline, ($\bar{X}$ = 112 ± 1.6) ($p < 0.05$). Total peripheral resistance (TPR, $dyne \cdot s \cdot cm^{-5}$) was depressed for 60 minutes compared to the pre-exercise baseline (BL, 959.8 ± 40.6), $p < 0.05$, but was fully recovered by 2 hours. It was concluded that in this group of young black normotensive males, moderate intensity aerobic exercise led to a prolonged reduction in SBP through relative changes in Q and TPR. These observations have not been consistently observed by other investigators in normotensive white males under similar experimental conditions.

Introduction

Epidemiological evidence reveals higher prevalence rates for hypertension in the black population compared with other racial groups. The evidence is also clear that this disease is more severe in blacks, resulting in greater target organ damage and mortality[1]. Yet, relatively few experimental studies have focussed on the responses of black subjects to aerobic exercise.

Previous research has demonstrated that during dynamic exercise there is an increase in systolic blood pressure (SBP) but in the recovery period, SBP is temporarily reduced[2-15]. Of these published studies, only three have reported on the hemodynamic changes that occur during the recovery period[4,8,9]. There is no indication in any of these studies that black individuals were specifically investigated. Furthermore, there is conflicting evidence about the underlying hemodynamic mechanisms associated with post-exercise hypotension.

Hagberg et al.[9] studied 18 hypertensive subjects (12 men and 6 women) between the ages of 60 to 69. One group of subjects worked at 50% of their maximal oxygen uptake for 45 minutes while the other group worked at 70% for the same period of time. At rest prior to the exercise, the mean SBP was 155 ± 19 mm Hg for the low intensity group and 159 ± 21 for the group that exercised at the higher intensity. The results from this study indicated that following a bout of intermittent aerobic exercise, SBP was

reduced by 8-13 mmHg for 1 to 3 hours depending upon the exercise intensity. Furthermore, the lower SBP in recovery was attributed to a reduction in the cardiac output (Q) which was mediated by a reduced stroke volume (SV). The authors speculated that the lower SV was due to a reduction in myocardial contractility following exercise. However, no measurements were made of this parameter. Contrary to previous assumptions[5,10,12,15], Hagberg et al.[9] reported that total peripheral resistance (TPR) was elevated for the duration of the hypotensive recovery period.

An elevation in TPR during the recovery period following 45 minutes of treadmill exercise at 70% heart rate reserve was also observed by Floras and Wesche [8] in their group of 5 hypertensives (4 men, 1 woman). These subjects had a mean age of 26.6 years and were characterized by resting heart rates that exceeded 90 beats per minute. In this study, the reduction in blood pressure was also associated with a reduction in Q.

In contrast, to the two other hemodynamic studies[8,9], Cléroux et al.[4] found that the reduction in blood pressure after 30 minutes of moderate intensity exercise was associated with an elevation in Q and a reduction in TPR. The subjects in this study were 13 hypertensives (11 men, 2 women) and 9 (7 men, 2 women) normotensive individuals. Prior to exercise, mean resting SBP (mm Hg) was 138 ± 2 and 105 ± 3 for the hypertensive and normotensive groups respectively. In addition to the previously

mentioned hemodynamic responses these investigators also reported a reduction in plasma norepinephrine levels, suggesting that a reduction in sympathetic nervous activity is related to this phenomenon.

The responses of a group of black subjects have not been specifically examined in any of the three published studies designed to examine the hemodynamic responses following acute aerobic exercise[4,8,9]. In young black normotensive subjects, there is some evidence that peripheral resistance is elevated compared to whites[16]. This could potentially alter the hemodynamic responses in black individuals.

To date, the self-report by Fitzgerald[5] is the only published study that has focused upon the effect of exercise on recovery blood pressures specifically in a black individual. By attempting to elucidate the mechanisms underlying the acute hemodynamic responses to aerobic exercise during the recovery period following exercise, some insights may be gained to help understand those changes that follow a period of chronic training.

Therefore, the present study was designed to (a), characterize the blood pressure responses during recovery from acute aerobic exercise of moderate intensity in young black normotensive males and (b), to determine the hemodynamic mechanisms underlying these post-exercise blood pressure changes.

Methods

Subjects. Nineteen moderately active normotensive black males (25.0 ± 1.0 yr; mean ± SEM) were recruited and paid to participate in the study. All were free of drugs and medications (one subject was a smoker) and met the criteria of "apparently healthy" as defined by the American College of Sports Medicine. After the study was approved by the Gettysburg College Human Subjects' Committee, subjects received medical examinations from a physician and were cleared for participation.

Session 1. Testing consisted of three sessions separated by no fewer than 48 hours. All sessions were scheduled at approximately the same time of day in order to control for diurnal variation. Subjects were instructed to abstain from alcohol, smoking, nicotine, food, and exercise for at least 6 hours prior to testing. During the first session subjects reported to the Human Performance Laboratory to complete medical history forms and to sign informed consent documents. In addition, they were instructed to respond to Forms X-1 and X-2 of the Spielberger Self-Evaluation Questionnaire[17]. These inventories were administered to evaluate anxiety levels prior to testing. After 15 minutes of seated rest in a quiet room, baseline blood pressures were determined. Three readings were made per arm with 1-2 minutes between trials. Height, weight, and waist-to-hip ratio were measured. Skinfolds were taken at the

chest, abdomen, and thigh[18]. Triplicate readings were made and recorded to the nearest millimeter. Body fat percentage was estimated by the Siri equation[19] (see Table 1. for descriptive data). At the end of the testing session, subjects were introduced to the treadmill and given a practice trial simulating the stages of the test to be given during the next session.

Session 2. During the second session, subjects were again asked to fill out Form X-1 of the Self Evaluation Questionnaire. They were then prepared for a 12-lead EKG (Quinton Q4000) and bioelectrical impedance (BoMed, NCCOM3-R7) measurements of heart rate (HR), Q, SV, end diastolic volume (EDV), and the index of contractility (IC)[20]. Bioelectrical impedance values were displayed at 5-minute intervals. IC is a measure of the contractile state of the myocardium. It was determined by equation 1:

$$IC = (\Delta Z/\Delta t)_{max}/Z_0 \qquad (1)$$

where Z and Z_0 are for impedance and base thoracic impedance respectively. $(\Delta Z/\Delta t)_{max}$ represents the first derivative of ΔZ with respect to time[20]. Impedance cardiography was used in this study since it measures resting hemodynamic variables non-invasively. While it does have limitations with respect to some

subject populations and some conditions (e.g. exercise), its greatest strength is its ability to track directional changes in Q and SV[21].

After a 15-minute seated rest period, baseline blood pressures were measured following the same procedures as in the previous session. Impedance measures were also made coinciding with the systolic and diastolic pressure readings for the calculation of total peripheral resistance during the second and third sessions. Peak oxygen uptake ($\dot{V}O_{2peak}$) was estimated by a graded treadmill test (Quinton Model Q55) using the Modified Bruce Protocol. The American College of Sports Medicine treadmill metabolic equation was used to estimate the $\dot{V}O_{2peak}$[22]. Heart rate was recorded during every minute of exercise. Ratings of perceived exertion (RPE), and blood pressures were measured during the third minute of each stage. The arm with the higher pressure readings from the baseline measures was used during testing. The 5th phase Karotkoff sound was the criterion for diastolic pressure. Subjects were instructed to continue to exercise until volitional exhaustion. A 5- minute active recovery followed the test, during which time cardiovascular measures were recorded every other minute.

Session 3. During the third testing session, subjects again completed the Self-Evaluation Questionnaire and were prepped for a 40-minute exercise session at a workload that would elicit a

heart rate within a range of 50-60% of heart rate reserve (maximum heart rate - resting heart rate). After a 15-minute rest period, baseline BP and cardiovascular measures (impedance cardiography) were taken. Following exercise, subjects were monitored for a 2-hour recovery period during which time blood pressure and cardiovascular data were collected every 10 minutes during the first hour and every 15 minutes thereafter.

Statistical analysis. All data are presented as mean ± SEM. Resting baseline (BL) (session 3) hemodynamic variables were compared to recovery values using a one-way repeated measures ANOVA. The Newman-Keuls multiple comparison procedure was used to determine which recovery values differed from baseline. Mean arterial pressure (MAP) and TPR were calculated by using equations 2 and 3 respectively:

$$MAP = DBP + 1/3\ (\ SBP - DBP) \qquad (2)$$

$$TPR = MAP \div Q \qquad (3)$$

The alpha level was set at the 0.05 level of significance.

Results

Subject characteristics & exercise task. Table 1 contains the

subjects' descriptive data. Mean estimated $\dot{V}O_{2peak}$ was 48.8 ± 1.5 $ml \cdot kg^{-1} \cdot min^{-1}$. Peak heart rates were directly measured during the graded exercise test using 12 lead electrocardiography. With the exception of one subject, all subjects achieved at least 90% their age predicted maximum heart rates and they were within one standard deviation of this value[22] (Table 2). During the 40 minutes on the treadmill (in session three), subjects exercised at an intensity that represented 57.3 ±1.2% of heart rate reserve with a mean RPE between 11 and 12.

Insert Table 1 & 2 about here

State anxiety. There was no significant difference in state anxiety between sessions 1-3, ($p > 0.05$). Likewise, there were no significant differences between baseline SBP and DBP readings taken between sessions 1, 2, and 3, ($p > 0.05$) (Table 3).

Insert Table 3 about here

Blood pressures. The mean resting baseline SBP (session 3) was

112.3 ± 1.6; during exercise it rose normally averaging 156.2 ± 1.1 mm Hg during the 40 minute period. In the recovery period, it was significantly reduced by at least 7 mm Hg from 30 minutes to 2 hrs post-exercise compared to the resting pre-exercise baseline value, ($p < 0.05$) (Figure 1). Neither the DBP nor the MAP showed any significant difference from resting baseline values during the recovery period.

Insert Figure 1 about here

Hemodynamics. In recovery, Q was significantly elevated above mean baseline resting values (BL, 6.9 ± 0.25 $L \cdot min^{-1}$) for the first 40 minutes, ($p < 0.05$) (Figure 2). This elevation was mediated by the heart rate (HR) since this was significantly

elevated above baseline (BL, 63 ± 2.6) for 75 minutes ($p < 0.05$) (Figure 3).

Insert Figure 2 about here

Insert Figure 3 about here

In contrast, during the recovery period, the SV was not significantly different from the baseline value at any time

(Figure 4). This was also true of the EDV. IC was significantly lower than the pre-exercise baseline at minutes 75 and 120 during the recovery period ($p < 0.05$), (Figure 5).

Insert Figures 4 & 5 about here

The TPR during the resting baseline conditions was 959.8 ± 40.6 $dyn \cdot s \cdot cm^{-5}$. This was significantly reduced during the first 60 minutes of the recovery period compared with the pre-exercise baseline ($p < 0.05$). Beyond that point, the TPR rose steadily throughout the remainder of the recovery period reaching a mean peak of 988.4 ± 51.9 $dyn \cdot s \cdot cm^{-5}$ by the end of the 2 hour period (Figure 6).

Insert Figure 6 about here

Discussion

To the authors' knowledge, this is the first study that has been conducted to specifically investigate the hemodynamic pattern associated with post-exercise hypotension in a group of black subjects. There has been little research conducted to

examine the impact of aerobic exercise upon modifying blood pressure in this racial group even though the incidence of hypertension is higher in this group than any other racial group[1,23]. Our results have demonstrated that forty minutes of moderate intensity aerobic exercise led to a significant reduction in SBP even 2 hours after the cessation of exercise. The magnitude of this reduction, ranging from 7-10 mm Hg, was consistent with what others have reported following exercise of similar intensity and duration, predominantly in hypertensive groups .[4,6,8-11,13] Furthermore, as others have found, the DBP did not differ from baseline during the recovery period[9].

In the study conducted by Cléroux et al.[4] there was an 11 mm Hg reduction in SBP in the recovery period compared with the control session following 30 minutes of cycle ergometry. DBP was 4 mm Hg lower than in the control condition during this time. These differences were only observed in the hypertensive group. Hagberg et al.[9] found that SBP was depressed by ~10 mm Hg below baseline values during the recovery period following 45 minutes of treadmill exercise. In this study(part A), there was no change in DBP compared with the preexercise value. The workrate used in that study represented approximately 50% of maximal oxygen consumption for each subject. At the higher intensity (i.e.,70% maximal oxygen uptake, part B), these researchers found that the magnitude of the reduction in SBP increased to 12-

19 mm Hg[9]. As in part A of this study, there was no change in DBP. Floras & Wesche [8] reported a 10.8 mm Hg reduction in SBP one hour following 45 minutes of treadmill exercise(70% heart rate reserve). This was not observed in the normotensive group. Therefore, it is evident that the changes in blood pressure that are reported in this study are very similar to the three previously published studies that were designed to document the hemodynamic changes associated with these blood pressure changes.

Kaufman et al.[11] studied the effects of dynamic exercise on recovery blood pressures in 8 normotensives ages 19-29, 8 normotensives 35-62 and 8 hypertensives 44-57 years. Subjects were required to attend two familiarization sessions prior to the experimental session. Their results indicated that there was a significant reduction in SBP, in all groups, between the first and third session. As a result of these findings the research design for this study included two familiarization sessions prior to the actual experimental session. These procedures were followed to ensure that the pre-exercise baseline was not spuriously high because of subject anxiety. The low state anxiety scores prior to the experimental sessions suggest that this goal was achieved. Since one of the major objectives of the present study was to elucidate the mechanisms associated with post-exercise hypotension, it was critical that the magnitude of any measured changes be as accurate as possible; hence the

importance of a good baseline.

A variety of mechanisms have been proposed to explain the phenomenon of post-exercise hypotension. These have included thermoregulatory mediated peripheral vasodilation[6,11], a reduction in blood volume[6,10,11], a reduction in sympathetic nervous system activity[6], a reduction in venous return[15], baroreflex resetting,[2,14] facilitation of cardiopulmonary reflexes[6] and more recently, endorphin release[3]. It was beyond the scope of this study to attempt to address some of these mechanisms. The major focus was on the hemodynamic changes.

The results of the present study, like those reported by Cléroux et al.[4], showed that cardiac output was higher in recovery compared to the pre-exercise baseline and was associated with a reduction in TPR. Furthermore, these changes were observed in the hypertensive group but to a lesser extent in the normotensive.

The hemodynamic findings of this present study are in contrast with those reported by Hagberg et al.[9] and Floras et al.[8]. In these studies the post-exercise reduction in blood pressure was associated with a decrease in cardiac output and an elevation in TPR. The major differences between the present study and the one reported by Hagberg et al.[9] relates the subjects used. The subjects in their study were older (i.e., 60-69 vs 25 yrs), females were included (4) and they were

hypertensive. The individuals in this study were all normotensive. However, the exercise intensities (part A) and duration were similar. This was also true for the study conducted by Floras et al.[8] However, there were differences related to the subject population studied. Their study included 5 hypertensives between the ages of 18-35 and 5 normotensives between 23 to 39. In their study, a reduction in blood pressure was not observed in the normotensive group.

The elevated cardiac output in the Cléroux study, as well as this present study, was mediated by an elevation in HR. Such an elevation in HR has also been observed by Floras et al.[6] who examined neural and cardiovascular responses of two groups of individuals following 45 minutes of treadmill exercise at 70% of heart rate reserve.

Bennett et al.[2] took a total of 18 subjects (9 hypertensive men vs nine normotensive individuals) and showed that during the recovery period when the SBP was reduced, the baroreceptors were still functional. Somers et al.[14], found evidence to suggest enhanced baroreceptor sensitivity beyond 20 minutes into the recovery period following exercise in 12 borderline hypertensive subjects. It is therefore possible that because of the reduction in SBP, the baroreceptor reflex (involving the carotid sinus and aortic arch receptors) was elicited with the attendant reflex increase in heart rate and increase in vascular resistance.

However, this does not fully explain the findings in this study since as the TPR was rising between minutes 60 to 120 post-exercise, the HR was actually falling. Therefore, other mechanisms may be responsible for mediating the changes in TPR and HR that were observed. Such mechanisms may involve cardiopulmonary baroreceptors located in the atria, ventricles and pulmonary vessels in addition to centrally acting opioid and serotonergic systems[6]. This is speculative since we did not address these issues in this study but these mechanisms have been proposed by other investigators[6,7].

The reduction in SBP that was observed in this study could be attributed to a number of factors. It has been well established that SBP is determined by the SV, the initial velocity of blood as it leaves the left ventricle, and arterial compliance[24]. The SV and the IC (which is based on the initial velocity of blood flow from the left ventricle), tended to be lower during the recovery period. These small reductions in SV and IC, probably combined to contribute to the 7-10 mm Hg reduction in SBP that was observed in this study which did not include hypertensive individuals.

As was alluded to earlier, the changes in SBP and other hemodynamic variables that were observed in this study have not been consistently noted in normotensive subjects by other investigators. In the group of normotensives studied by Floras

et al.[8] there was no change in the TPR during recovery. Furthermore, the normotensives from the studies conducted by Floras and Senn[7], Floras and Wesche[8] and Cléroux et al.[4] did not demonstrate post-exercise hypotension. The reduction in SBP and TPR observed in the subjects in the present study may result from enhanced inhibitory cardiopulmonary reflex activity post-exercise. This could be the case since Floras et al.[6] showed that individuals who demonstrate post-exercise reductions in SBP have a reduction in sympathetic activity to peripheral blood vessels. In contrast, those individuals who fail to show post-exercise hypotension following submaximal exercise, do not have sympathoinhibition during this period. We are unable to state whether any of our subjects had sympathoinhibition following exercise. There was only one subject who did not show a drop in SBP below baseline of between 7-10 mm Hg for at least two measurement periods during the recovery period. In this subject, there was a drop in TPR below baseline values but this was compensated by a relatively greater increase in cardiac output.

The results from this study may indicate that normotensive black males may find regular periods of moderate intensity aerobic training beneficial in maintaining blood pressures within the normotensive range. This observation has already been made in predominantly white cohorts[27,28]. Paffenbarger et al.[27] demonstrated that Harvard alumni who performed vigorous physical

activity on a regular basis had a reduced incidence of hypertension compared with their less active peers. Blair et al.[28] performed a longitudinal study in which fitness levels and blood pressure status were assessed initially and after a 1 to 12 year period. They found that a reduced level of fitness was associated with an increased incidence in the development of hypertension in previously normotensive individuals. Similar studies are warranted in a cohort of black subjects.

In summary, the present study demonstrated that aerobic exercise of moderate intensity and duration led to a 7-10 mm Hg reduction in SBP for up to 2 hours post-exercise with no changes in DBP. The reduction in SBP was mediated by relative changes in TPR and Q. Results of the present study have given some initial indication of the mechanisms mediating changes in blood pressure after moderate intensity aerobic exercise in normotensive black males.

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TABLE 1. Descriptive and exercise data for 19 normotensive males.

Variable	Mean	SEM
Age (yrs)	25.4	1.3
HT (cms)	181.0	1.4
Wt (kg)	85.8	3.3
Waist/Hip	0.86	0.0
% Fat	14.1	1.5
*VO_{2peak}	48.8	1.5
THR (bpm)	135.4	2.6
% $HR_{reserve}$	57.3	1.2

* = $ml \cdot kg^{-1} \cdot min-1$

TABLE 2. Heart rates and systolic blood pressure (SBP) at peak exercise for the 19 subjects.

Sub #	Predicted HR_{max}	Measured HR_{max}	± beats	%predicted HR_{max}	Peak SBP (mm Hg)
1	192	180	-12	93.8%	230
2	196	200	+4	101.0%	210
3	200	191	-9	96.0%	168
4	202	188	-14	93.1%	184
5	200	198	-2	99.0%	198
6	193	200	+7	103.6%	154
7	184	185	+1	100.5%	188
8	187	188	+1	100.5%	183
9	191	176	-15	92.1%	188
10	196	205	+9	104.6%	170
11	202	190	-12	94.1%	194
12	199	188	-11	94.5%	210
13	187	175	-12	93.6%	164
14	202	188	-14	93.1%	170
15	201	198	-3	98.5%	180
16	191	198	+7	103.7%	182
17	198	188	-10	94.9%	200
18	190	188	-2	98.9%	190
19	187	155	-32	82.9%	198

TABLE 3. Baseline pre-exercise mean anxiety, systolic and diastolic pressure values. Values represent means ± SEM.

BASELINE	STATE ANXIETY	SYSTOLIC PRESSURE (SBP) (mm Hg)	DIASTOLIC PRESSURE (DBP) (mm Hg)
Session 1	30.1 ± 1.6	113.2 ± 2.1	67.7 ± 2.1
Session 2	32.4 ± 2.1	114.7 ± 1.9	70.0 ± 2.2
Session 3	31.1 ± 1.8	112.3 ± 1.6	64.6 ± 1.4

Figure legends

Figure 1. Systolic blood pressures during two hours of recovery after exercise. Pre-exercise baseline (BL), recovery (R), (* = $p < 0.05$ vs baseline). Values = mean ± SEM.

Figure 2. Cardiac output during two hours of recovery after exercise. Pre-exercise baseline (BL), recovery (R), (* = $p < 0.05$ vs baseline). Values = mean ± SEM.

Figure 3. Heart rate during two hours of recovery after exercise. Pre-exercise baseline (BL), recovery (R), (* = $p < 0.05$ vs baseline) Values = mean ± SEM.

Figure 4. Stroke volume during two hours of recovery after exercise. Pre-exercise baseline (BL), recovery (R), (* = $p < 0.05$ vs baseline). Values = mean ± SEM.

Figure 5. Index of contraction during two hours of recovery. Pre-exercise baseline (BL), recovery (R), (* = $p < 0.05$ vs baseline).Values = mean ± SEM.

Figure 6. Total Peripheral resistance ($dyn \cdot s \cdot cm^{-5}$) during two hours of recovery after exercise. Pre-exercise baseline (BL), recovery (R), (* = $p < 0.05$ vs baseline). Values = mean ± SEM.

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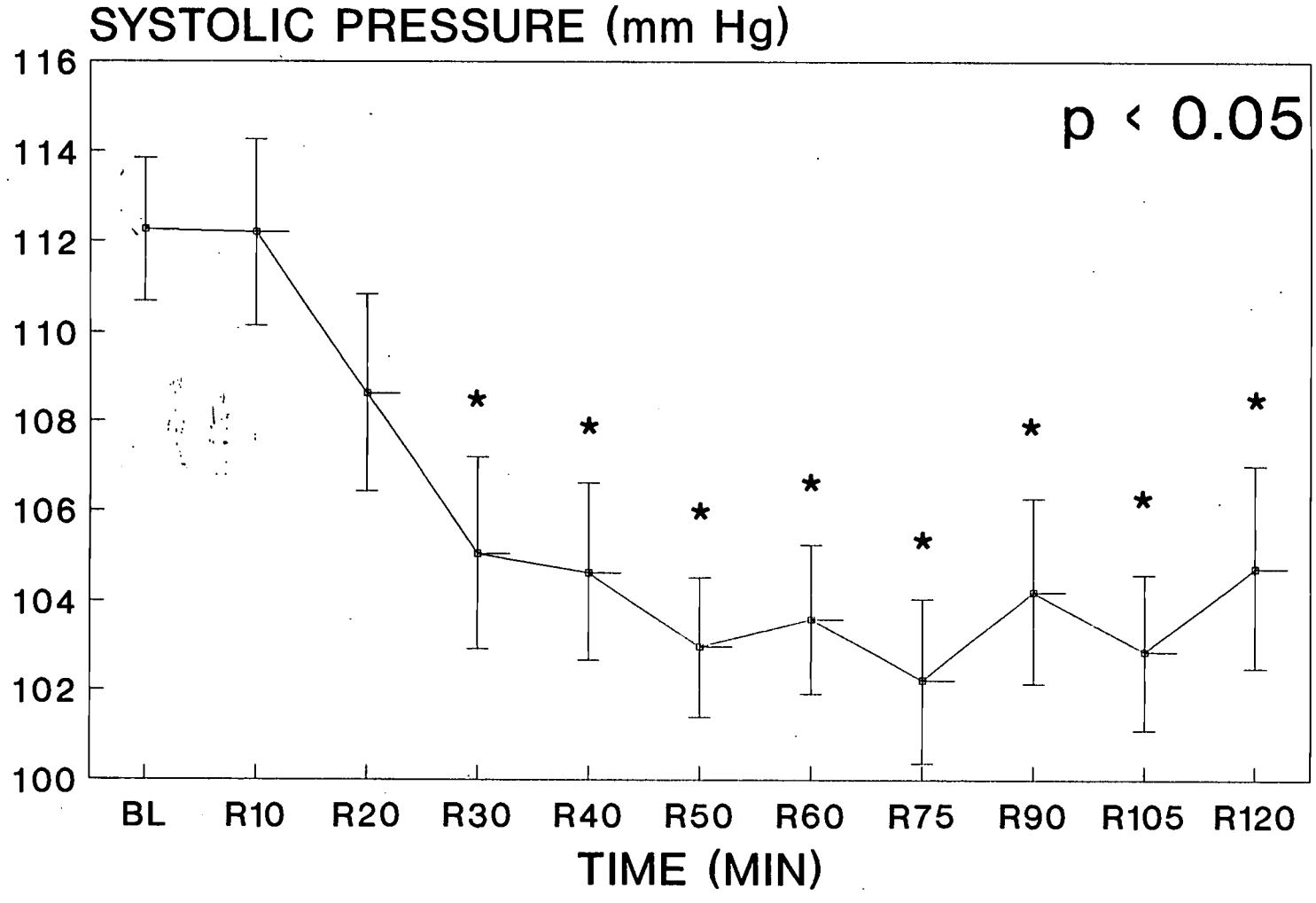
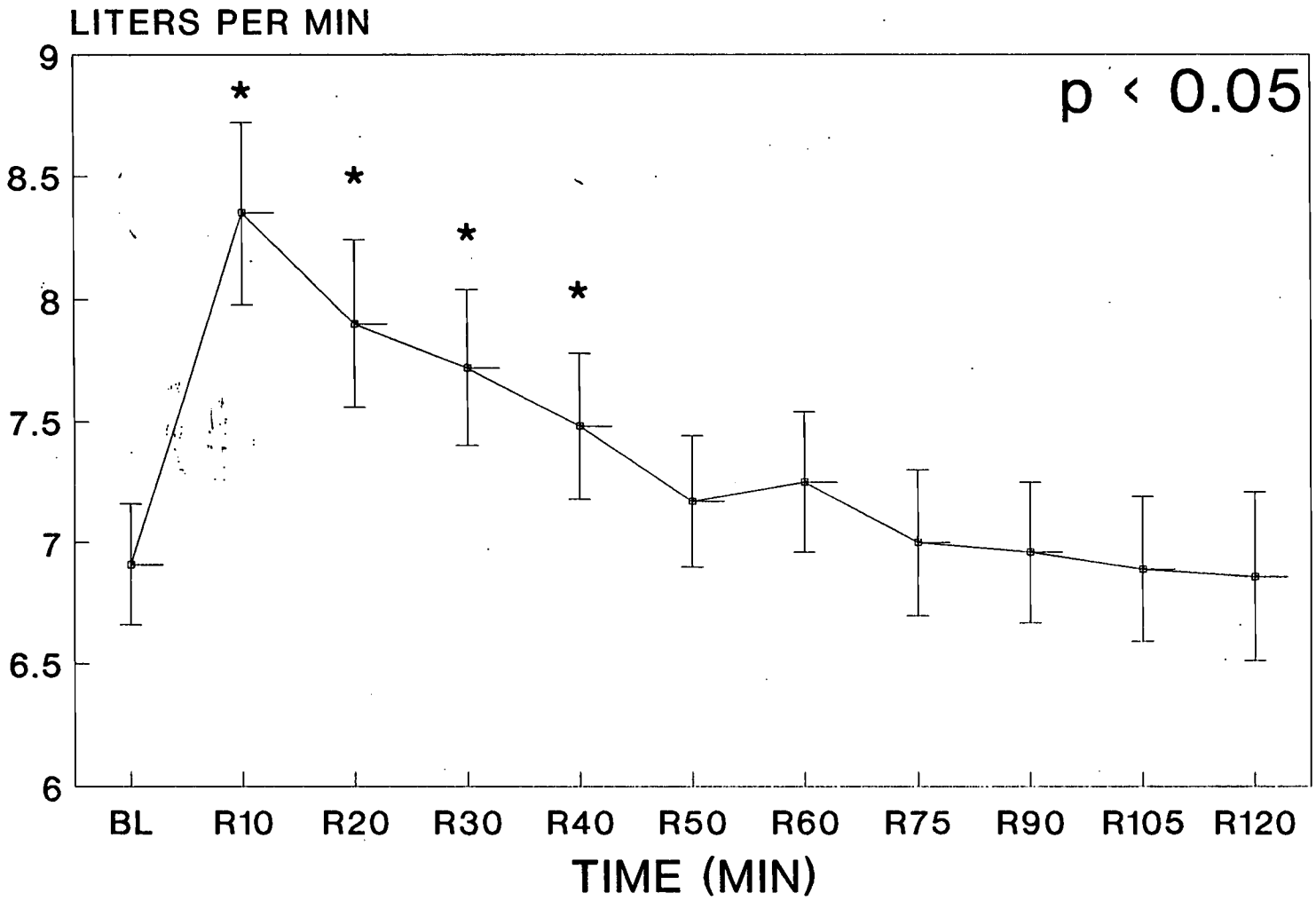
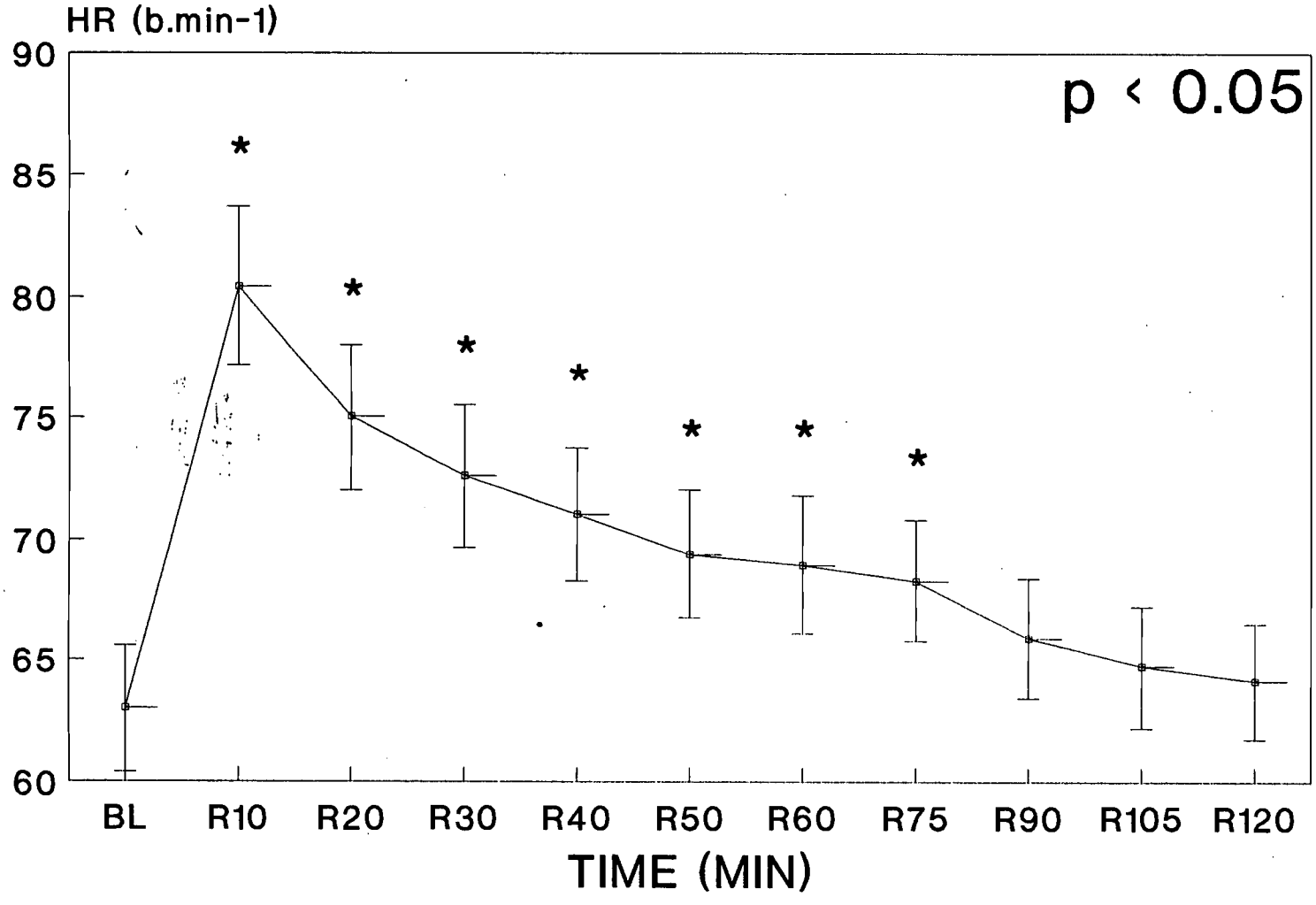
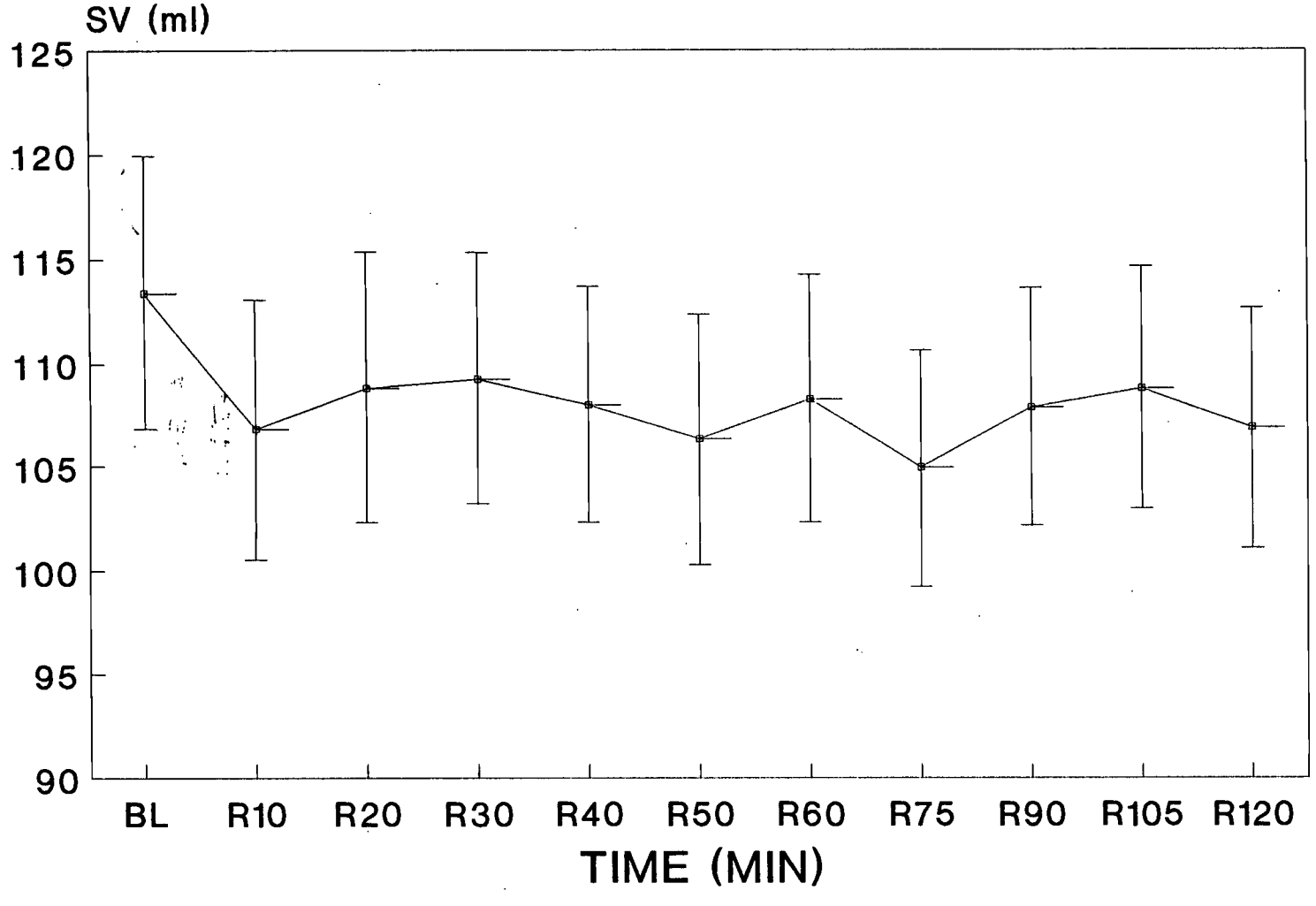
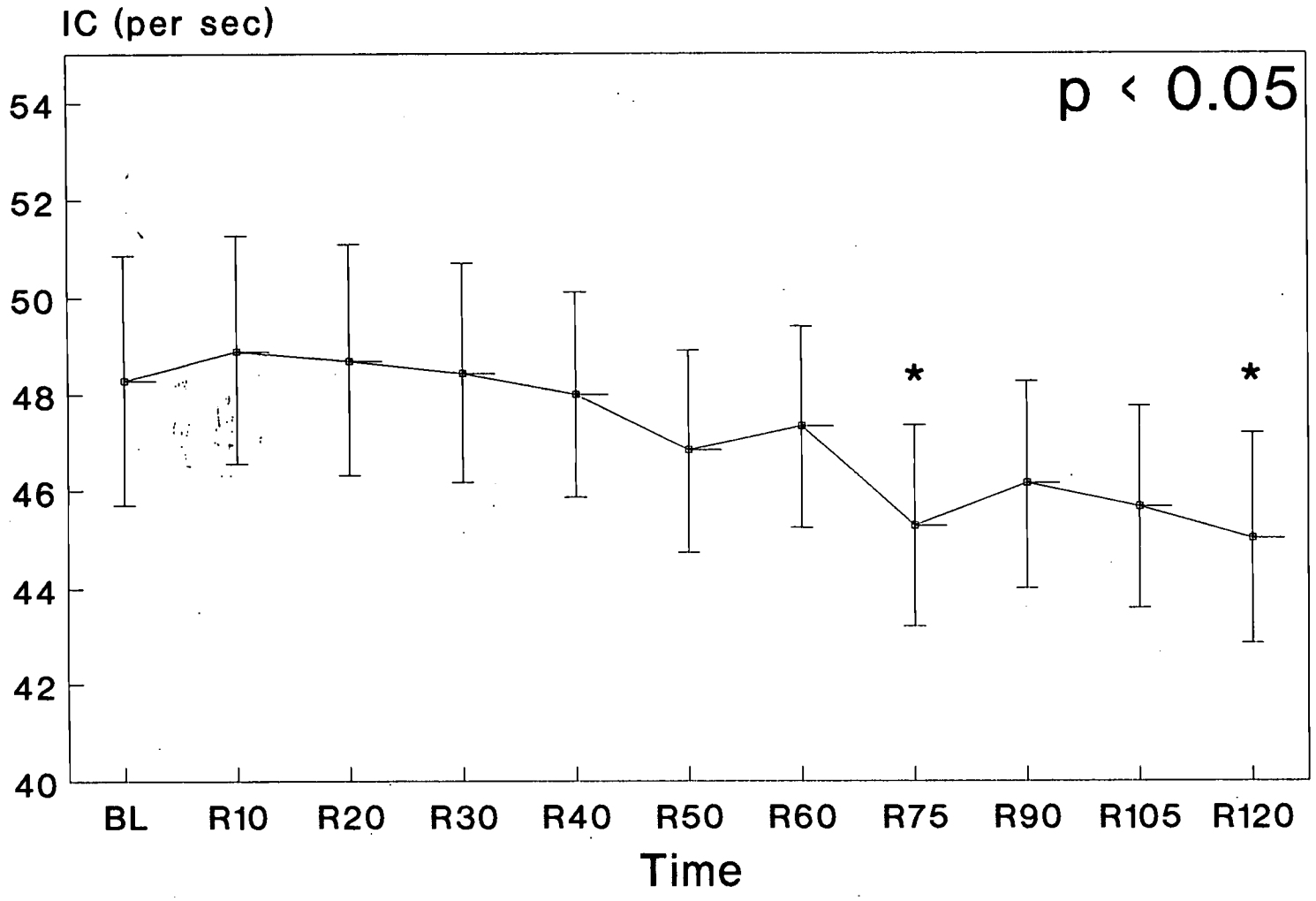
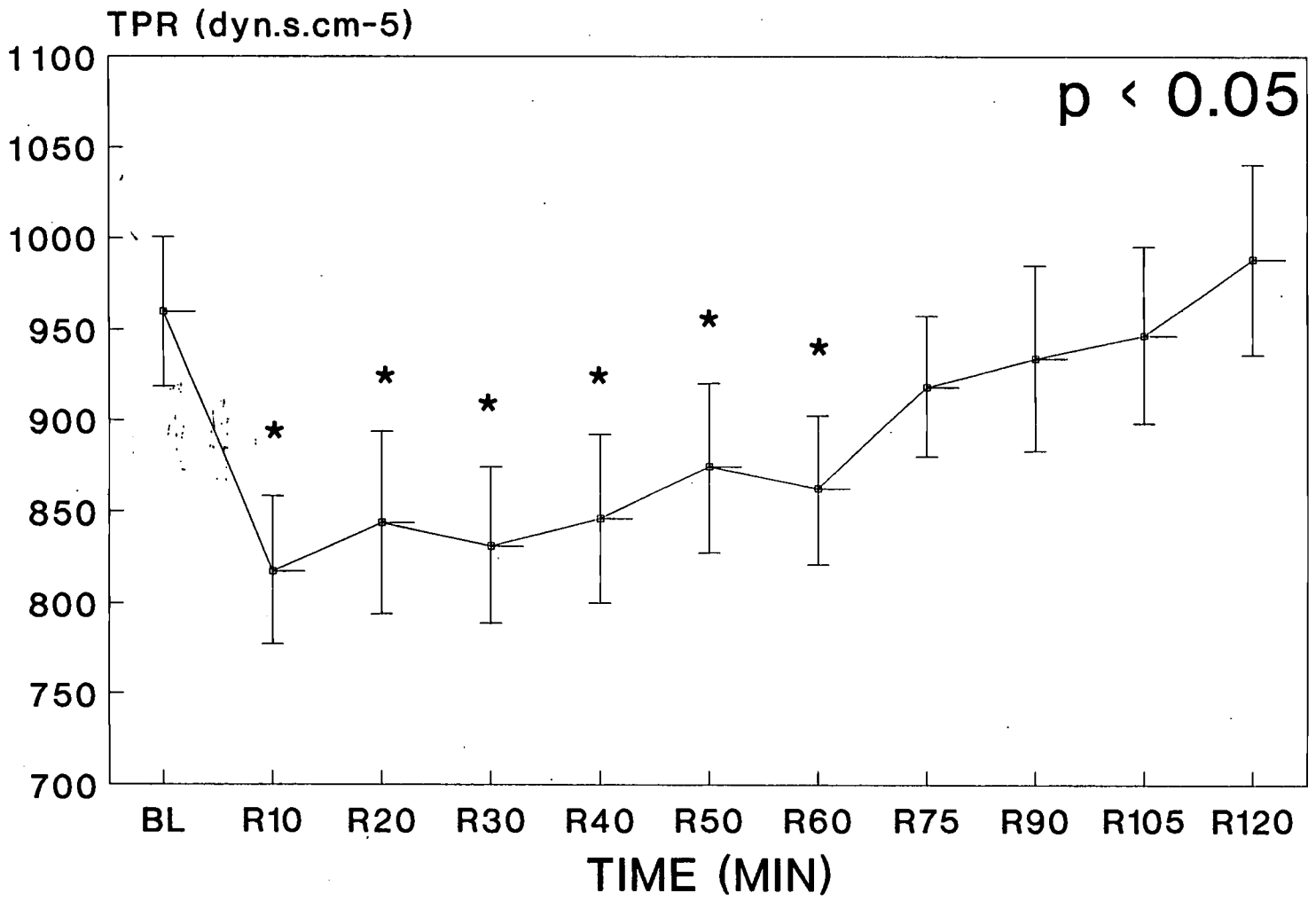

SYSTOLIC PRESSURE (mm Hg)
116
114
112
110
108
106
104
102
100
p < 0.05
*
BL
R10
R20
R30
R40
R50
R60
R75
R90
R105
R120
TIME (MIN)




HR (b.min-1)
90
85
80
75
70
65
60
p < 0.05
*
*
*
*
*
*
*
BL
R10
R20
R30
R40
R50
R60
R75
R90
R105
R120
TIME (MIN)


SV (ml)
125
120
115
110
105
100
95
90
BL
R10
R20
R30
R40
R50
R60
R75
R90
R105
R120
TIME (MIN)


IC (per sec)
p < 0.05
54
52
50
48
46
44
42
40
*
*
BL
R10
R20
R30
R40
R50
R60
R75
R90
R105
R120
Time


TPR (dyn.s.cm-5)
1100
1050
1000
950
900
850
800
750
700
p < 0.05
*
*
*
*
*
*
BL
R10
R20
R30
R40
R50
R60
R75
R90
R105
R120
TIME (MIN)

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