

## Velocity of Heart Rate Recovery in Post-Exercise Under Different Protocols of Active Recovery

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### ABSTRACT

Abnormalities in the modulation of parasympathetic activity have been identified as a possible pathophysiological link with the association between decreases in heart rate recovery after exercise test. Hypothesis: To investigate if the application of different protocols of active recovery promotes difference in reducing Heart Rate (HR) in Post-Exercise (PE). This was a prospective, randomized trial of patients undergoing Exercise Testing (ET) and applied to two different protocols of active recovery. They were divided into G1 with recovery of 1.5 mph/2.5% and G2 40% of the speed and slope of the peak in the PE. Variables were evaluated pre-test and intrinsic to the TE. We compared the recovery of FC 1 and 2 min in the PE. Statistical analysis used the chi-square test, Student's t test and considered  $p < 0.05$ . We analyzed 939 patients and 852 selected for randomization. Were allocated to G1 403 patients with mean age  $47.86 \pm 14.31$  years, 53.35% female and G2 with 449 patients with mean age  $48.56 \pm 14.23$  years and 51.7% were female. In the analysis of HR recovery of first (22.00 Vs. 23.57;  $p = 0.315$ ) and second (36.17 Vs. 37.70,  $p = 0.06$ ) minutes, G1 and G2 did not obtain differences. The use of different models of active recovery, applying fixed workload of 1.5 mph/2.5% slope or individually in 40% of the workload of the peak stress does not alter the recovery of HR in first or second minutes in the PE.

**Keywords:** Exercise, Autonomic System, Heart Rate

### 1. INTRODUCTION

The HR recovery after exercise represents the reactivation of parasympathetic activity and a reduction in sympathetic activity that typically occurs during the 30 sec after exercise. Abnormalities in the modulation of parasympathetic activity have been identified as a possible pathophysiological link with the association between decrease in Heart Rate (HR) recovery after exercise test on a treadmill and increased mortality in the follow-up (Imai *et al.*, 1994; Nishime *et al.*, 2000).

Currently, the behavior of HR has been widely used in diagnostic analysis and prognosis of asymptomatic patients or with cardiovascular disease and prescription

of exercise and control training (Lahiri *et al.*, 2008). Researches in this field have studied the behavior of HR at rest, during exercise and recovery.

The rate of HR recovery proved to be prognostic in the first and second minute after the effort in populations referred for exercise testing than conventional, independent of exercise protocol employed (Cole *et al.*, 1999; 2000). Since the first studies, Lauer *et al.* (2002) demonstrates conventionally that in using the active recovery protocols with treadmill speed of 1.5 mph and 5% incline, the speed reduction would be considered satisfactory HR of 12 bpm for the first minute and 22 bpm in the second minute. Several studies have proven the prognostic value of HR recovery on

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cardiovascular morbidity and mortality. Shetler *et al.* (2001) Nevertheless, all papers have considered the application of the methodology of active recovery (1.5 mph/5% slope) for study validation (Ellestad, 2008). However, in our laboratory routinely employ active recovery in proportion to the workload peak (40% of the speed and incline at peak exercise).

The aim of our study was to evaluate if different protocols of speed and incline of the treadmill the period of active recovery could influence the rate of HR reduction in post-exercise, promoting differences in the duration of inhibition of the sympathetic and parasympathetic activation.

## 2. MATERIALS AND METHODS

We evaluated 939 consecutive patients referred by their physician for TE and selected 852. Considering an alpha error of 5% and a beta error of 20%, the sample size needed to identify the lowest correlation coefficient with statistical significance would be 402 patients in each group. We included patients over the age of eighteen and able to perform treadmill stress test. Exclusion criteria were: Contraindication to perform stress testing according to II Exercise Testing Guideline of the Brazilian Society of Cardiology (Andrade *et al.*, 2002), did not have completed 2 min of active recovery phase, effort lasting less than 6 min arrhythmias and preventing the correct draft assessment of the HR. All patients underwent ET on treadmill ramp protocol with active recovery period post-exercise lasting at least 2 minutes and 30 sec and kept under observation in passive recovery for at least 5 min. Patients were randomized before the start of the test, with Group one (G1) underwent active recovery at 40% speed and slope of peak exercise and in Group two (G2) was kept fixed speed of 1.5 mph and absence of inclination.

### 2.1. Exercise Test

The maximum ET (symptom limited), was performed by a ramp protocol, according to the estimated functional capacity. Individuals under drug therapy have been tested on their medication regularly. Blood pressure was measured in an indirect way in mercury sphygmomanometer brand Wanros, fixed on a support within 1 meter of soil. During exercise blood pressure was measured and accomplished record of short electrocardiographic register with 13 derivations lead every 2 min at maximum effort in the immediate

post-exercise (10 sec) and 1, 2, 3 and 5 min of recovery and each event or change HR. The HR was determined automatically by the program for ergometry at rest, every 2 min of exercise, peak exercise and every minute during recovery. The measurements taken by the program at peak exercise and the first and second minute of recovery were checked manually at the end of the examination and corrected when necessary. The interruption of the exercise was done in accordance with the II Guidelines for Exercise Testing of the Brazilian Society of Cardiology (Andrade *et al.*, 2002).

### 2.2. Statistical Analysis

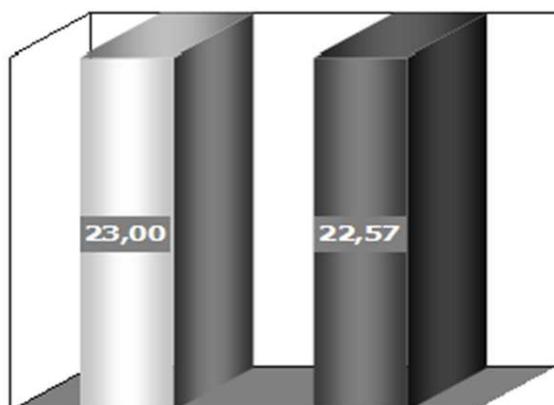
The results were presented as frequencies (%), mean and standard deviation. The chi-square and Student t test for independent samples were used for statistical evaluation of differences between proportions and means, respectively, after considering if the data were in line with the assumptions for these tests. The level of significance was  $p < 0.05$ .

## 3. RESULTS

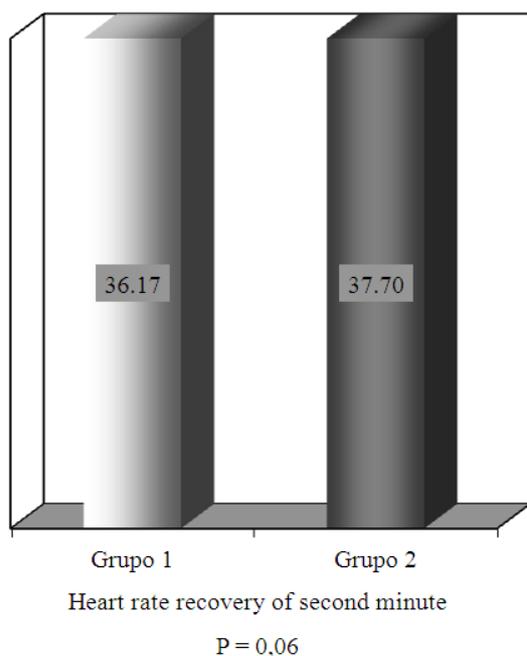
The sample was evaluated for 852 patients with a mean age of  $48.23 \pm 4.26$  years and 52.5% female. The G1 compound 403 patients with a mean age of  $47.86 \pm 14.31$  years, 53.35% female and 4.5% were diabetic while the G2 consists of 449 patients with mean age  $48.56 \pm 14.23$  years, 51.7% female and 5.1% had diabetes, both with similar percentages of female patients and diabetes mellitus. The characteristics of the groups are in **Table 1**.

Regarding the data evaluated in ET, there was no difference between HR pre-test between both groups ( $80.03 \pm 13.98$  Vs  $83.22 \pm 40.40$ ,  $p = 0.132$ ) and number of Metabolic Equivalent (METs) obtained at the end of the examination ( $9.91 \pm 2.36$  Vs  $9.69 \pm 2.19$ ,  $p = 0.164$ ). (**Table 2**) There were no differences in the data intrinsic to the TE as the FC in the first minute of recovery post-exercise between the G1 and G2 ( $23.0$  Vs  $22.57$ ,  $p = 0.315$ ) in the second minute of recovery (Vs  $36.17$ .  $37.70$ ,  $p = 0.06$ ) and  $VO_2$  max ( $39.19 \pm 5.7$  Vs  $40.00 \pm 7.3$ ,  $p = 0.38$ ) (**Fig. 1 and 2**).

In the other variables in patients  $< 35$  years there was no difference between groups ( $23.00 \pm 9.8$  Vs  $24.05 \pm 8.2$ ,  $p = 0.45$ ) and those  $> 65$  years as HR first minute ( $17.78 \pm 7.3$  Vs  $17.87 \pm 8.2$ ,  $p = 0.47$ ) and second minute ( $28.00 \pm 9.7$  Vs  $26.77 \pm 10.01$ ,  $p = 0.24$ ) recovery effort in the post and an average age between the groups ( $71.34 \pm 4.0$  Vs  $70.85 \pm 4.3$ ,  $p = 0.24$ ).



**Fig. 1.** Comparison between G1 and G2 in HR recovery at first minute post-exercise recovery



**Fig. 2.** Comparison between G1 and G2 in HR recovery at 2 minutes post-exercise recovery

**Table 1.** Characteristics of sample

	Group 1	Group 2	p. value
n	403	449	
Age (years)	47,86±14,31	48,56±14,23	0.48
Male Sex (%)	53,35	51.67	0.63
BMI (kg/m <sup>2</sup> )	26,08±4,28	26,18±4,04	0.72
Diabetes Melittus (%)	0.4.46	0.5.12	0.51
Regular physical activity (%)	21.34	20.21	0.32
Cardiac disease (%)	0.2.32	0.1.78	0.37

**Table 2.** Comparative analysis between groups about intrinsic variables in ET

	Group 1	Group 2	p. value
HR pre test (bpm)	80,03±13,98	83,22±40,40	0.13
HR peak exercise (bpm)	155,74±22,47	154,65±22,74	0.48
METs	9,91±2,36	9,69±2,19	0.16
Exercise time (s)	519,84±83,03	506,10±85,32	0.41
Cronothropic deficit (%)	0.9.67	0.10.29	0.33
FAI (%)	-15,41±25,60	-13,65±25,23	0.31
Maximum doubled-product	27807±5796	27943±5968	0.73
Ischemic (%)	0.5.21	0.5.34	0.45

HR: Heart Rate; METs: Metabolic Equivalents; FAI: Functional Aerobic Impairment

#### 4. DISCUSSION

In our study, no differences were observed between the recoveries of HR in post-exercise, regardless of active recovery protocol applied. As the HR recovery at first minute exercise depends basically on the return of parasympathetic autonomic activity, our results suggest no differences in the intensity of vagal activity, when applying speeds and different workloads in the period of active recovery.

During exercise, HR increase is due to increased sympathetic activity and decreased vagal activity (Cay, 2009). After cessation of exercise, the rapid decrease in HR is initially determined by vagal reactivation and subsequently by inhibition of sympathetic doing HR recovery a marker of cardiovascular parasympathetic activity (Schwartz *et al.*, 1992). Therefore, the slow decline of HR after exercise is an indicator of malfunction of the ANS, specifically, the parasympathetic activity (Jouven *et al.*, 2005). In our sample, the autonomic balance and cardiovascular system's ability to respond to withdrawal of sympathetic activity and beginning of the parasympathetic activity appear to have been affected when subjected to different methods of active recovery. When analyzed as extreme age <35 years old and >65 years old, both groups showed no differences when subjected to different workloads in active recovery.

Some studies (Gaibazzi *et al.*, 2004; Kizilbash *et al.*, 2006) have shown that the rate of HR recovery post-exercise may be influenced by the type of ergometer. Ergometer in a reduction less than 22 bpm has been associated with increased mortality. In the wake of these values can be influenced by the type of recovery applied,

if passive, with abrupt cessation of movement, or active, with the belt still in motion for at least 2 min. Abrupt stopping of the treadmill determines a more rapid decrease in HR. According Halliwill *et al.* (2001), we can speculate that the presence of hypotension in the post stress can stimulate the arterial baroreflex vagal, bringing restoration and reducing the sympathetic activity (Halliwill *et al.*, 1996). With the patient in the supine position immediately after exercise and during stress echocardiography, a reduction less than 18 bpm at 1 min has been associated with higher morbimortality. Halliwill *et al.* (1996) with the abrupt interruption and the patient kept in orthostatic position, we can expect reductions of around 30 bpm probably due to increased blood pressure by stimulating the parasympathetic activity (Perini and Veicsteinas, 2003). The type of exercise protocol employed, ramp or multistage during the exercise test on a treadmill, does not seem to influence the rate of HR recovery, since the protocol employed was appropriate to the functional condition of the individual. In our study, we excluded patients who arrived exhaustion less than 6 min of exercise, whereas the protocol applied in these cases was inappropriate and can result in different hemodynamic responses. However, for submaximal tests, Parekh and Lee (2005) suggest that exercise loads of 80%  $VO_2$  reserve, result in major changes in parasympathetic activity, when compared to exercise at 50%  $VO_2$  reserve. Patients who have not reached the level of 10 fatigues by Borg scale of 1-10 were excluded, existing therefore a mismatch between exercise intensity in both groups.

Concerning the type of active recovery protocol on a treadmill, since the early work comes up routinely using the recovery speed of 1.5 mph with slope of 2.5% (Schwartz *et al.*, 1992; Jouven *et al.*, 2005; Lauer *et al.*, 1996); in our laboratory we apply proportional reduction in the speed and incline at 40% maximum  $VO_2$  by considering that in populations with very high or very low functional capacity, recovery fixed at 1.5 mph/2.5% is not adequate and can be very slow for an athlete race or too fast for the elderly or cardiac patients, which often reach a final speed of only two mph at peak effort, as in the Naughton protocol.

Our results show that in maximum treadmill exercise tests, with active recovery at a speed of 1.5 mph/2.5% or with speed and incline to 40% of  $VO_2$  peak; we can use the same cutoffs as markers of risk: 12 bpm for 1 min and 22 bpm for 2 min of recovery.

## 5. CONCLUSION

The use of different models of active recovery, applying fixed workload of 1.5 mph/2.5% slope or individually in 40% of the workload of the peak stress does not alter the recovery of heart rate in the first or in the second minute after exertion.

Although the hypothesis has been tested negative, this result is very important, since the demonstration that two different modes of active recovery did not influence the presence or absence of autonomic dysfunction, or even natural recovery of heart rate. More studies are necessary to assess the autonomic function in post-exercise, seeking to identify the mechanisms that determine the return of parasympathetic activity during recovery and its relation to cardiovascular disease and mortality.

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