

Original Article

Clinical Significance of Impaired Heart Rate Recovery after Treadmill Exercise Test in Hypertensive Patients

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ABSTRACT

Objectives: To evaluate the recovery time after a treadmill exercise ECG test in hypertensive patients and to study the role of β -blockers on the recovery of the heart rate after exercise.

Design: Prospective controlled study conducted between March 1998 and January 2002 with a follow-up after six months.

Setting: A non-invasive cardiac laboratory, medical department, Farwania Hospital.

Methods: We studied 250 patients and divided them into two groups. The first group included 170 untreated hypertensive patients (140 men and 30 women) and the control group included 80 normotensive patients (70 men and 10 women). Echocardiography was done to assess left ventricular mass index and function. Exercise ECG test was done for all patients at baseline. Hypertensive patients were classified into two subgroups. The first subgroup included 70 patients with normal heart rate recovery (> 12 beat/minute) and the second subgroup included 100 patients with impaired heart rate recovery (< 12 beat/minute). A β -blocker was prescribed for 50 patients and other antihypertensive drugs was prescribed for another 50 patients for six months. The exercise ECG test was repeated again for all 100 patients.

Results: There was a significant impaired diastolic function of the left ventricle in hypertensive patients with impaired heart rate recovery than those with normal heart rate recovery ($p < 0.05$). However there was no significant difference regarding left ventricular mass index and systolic function. There was a significant decrease in resting and peak heart rate during exercise in patients with β -blockers than those without β -blockers. There was a significant decline in heart rate after exercise in patients with β -blockers at a follow-up check up ($p < 0.05$). The decline in heart rate during the first minute of recovery after exercise at follow-up was more increased as compared with the results of baseline exercise ECG test ($p < 0.01$). Stepwise logistic analysis revealed a significant relation between the age of the patients and left ventricular diastolic dysfunction and impaired heart rate recovery after exercise in hypertensive patients ($p < 0.05$).

Conclusion: Impaired heart rate recovery after exercise in hypertensive patients is related to the age of the patients and the presence of left ventricle diastolic dysfunction and that beta-blockers affect heart rate in recovery, most likely due to an indirect effect by the reduction of chronotropism.

KEYWORDS: exercise test, heart rate recovery, heart rate reserve, ischemic heart disease

INTRODUCTION

Recently, there has been increasing interest in the role of the autonomic nervous system regulation in heart disease. Heart rate or RR cycle variability, one marker of autonomic activity, has been found to be an important marker of risk both among survivors of myocardial infarction^[1] and among healthy adults in the Framingham Heart Study^[2]. Inability to mount a good chronotropic response is defined as an ability to achieve 85% of target heart rate (HR) during exercise^[3,4]. Increased age has been associated with increased mortality, partly due to its close association with chronotropic incompetence, secondary to decreased physical fitness^[5]. The rise in heart rate during exercise is considered to be due to a combination of parasympathetic withdrawal and sympathetic activation. Decline in HR after exercise has only

recently been studied. It has been proposed to be an indicator of reactivation of the parasympathetic nervous system and thus, an abnormally slow rate of heart rate recovery after exercise would indicate an abnormality of the parasympathetic nervous system^[6]. Recently, abnormal decline in HR after exercise has gained attention as an independent predictor of all-cause and cardiac mortality^[7,8].

The aim of the study is to evaluate the recovery time after treadmill exercise ECG test in hypertensive patients and to study the role of β -blockers on the recovery of heart rate after exercise.

PATIENTS AND METHODS

Two hundred and fifty patients were included in the study. All patients were referred by their physicians to the Non-Invasive Cardiac Laboratory,

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Medicine Department, Farwania Hospital between March 1998 and January 2002, for assessment of chest pain. All patients were evaluated clinically by looking at the history, physical examination, 12-lead ECG, routine laboratory investigations and echocardiography and Doppler study.

Exclusion criteria included patients with diabetes mellitus, cerebrovascular disease, significant valvular disease, irritability, anxiety, chronic obstructive lung disease and history of heart failure. Exclusion was based on: medical history, physical examination, routine biological chemical tests, and echocardiography to avoid confounding factors. There were two groups:

Group I: included 170 pharmacologically untreated hypertensive patients (140 male and 30 female).

Group II: included 80 normotensive patients (70 male and 10 female).

Echocardiographic study:

Two-dimensional and M-mode echocardiography were performed for all patients included in the study on a Toshiba Power Vision using a 3.5 MHz phased array transducer. All echocardiographic studies were performed by the same cardiologist. Measurements were performed according to the recommendations of the American Society of Cardiology^[9]. The leading edge to leading edge convention was used. Left ventricular dimensions were measured, at or immediately below, the tips of the mitral leaflets and were averaged over five heart cycles. Left ventricular mass and left ventricular mass index were calculated.

Pulsed Doppler echocardiography was obtained from the standard apical four chamber view. Mitral inflow velocity was recorded with the sample volume at mitral annulus level. The transducer was then manipulated to obtain the maximal flow velocity as assessed by the auditory and spectral outputs. The Doppler measurements were made during at least three cardiac cycles using the darkest part of the spectral recording and were then averaged^[10]. The following measurements were obtained: peak velocity of early left ventricular filling (E), peak velocity of late left ventricular filling (A) and the ratio between early and late flow velocity (E/A).

Treadmill exercise ECG test protocol:

All patients of the study underwent an exercise ECG test using standard or modified Bruce models at the baseline of the study. Resting blood pressure (measured manually by arm-cuff sphygmomanometer) was measured in supine and standing positions before the test. Patients with orthostatic hypotension (defined as a decrease of > 20 mm Hg of systolic blood pressure after standing) were excluded. Resting ECG was done for all patients to exclude patients with significant ST-

segment changes, left bundle branch block or tachyarrhythmias. Patients with typical chest pain on the test day were excluded and were referred back to the physician and to come back after four weeks. Blood pressure was automatically recorded midway through each stage and at peak exercise. The stress ECG test was terminated, if there was a decrease in blood pressure (> 20 mm Hg), significant arrhythmias (non-sustained or sustained ventricular tachycardia), typical chest pain (test limiting angina) or > 2 mm ST-segment depression from baseline was noted^[7]. Peak heart rate (HR): achieved percentage of age-related peak heart rate = $(\text{peak HR} / 220 - \text{age}) \times 100$ and the heart rate reserve percent were computed for each patient. The heart rate reserve was calculated using the formula described by Lauer *et al*^[11]: $[\text{peak HR} - \text{HR at rest} (220 - \text{age}) - \text{HR at rest}] \times 100$. Chronotropic incompetence was considered present, if less than 80% of the patient's heart rate reserve was used at peak exercise^[12].

Recovery after exercise:

After achieving peak work-load, the treadmill was stopped and blood pressure, heart rate, rhythm and symptoms were recorded immediately with the patient in the standing position (no cool down period). The same data were also recorded at 1, 2, 3, 4, 5 and 6 minutes into recovery in the supine position. Heart rate recovery was calculated as reduction in heart rate from the peak to one minute of the recovery time and a cutoff value of 12 beats/minute or less was considered abnormal^[13,14]. Monitoring was terminated at six minutes into the recovery unless warranted by symptoms or electrocardiographic changes. There were two subgroups:

Subgroup (N): 70 hypertensive patients with normal heart rate recovery (more than 12 beats reduction in the first minute of the recovery time).

Subgroup (I): 100 hypertensive patients with impaired heart rate recovery (less than 12 beats reduction in the first minute of the recovery time).

Quintile analysis was applied to the age of the patients with impaired heart rate recovery after exercise in which the patients were subdivided into three groups:

The first quintile included the patients with the age less than 45 years, the second quintile included the patients with the age between 45 and 55 years, and the third quintile included the patients with the age more than 55 years.

Design for follow up:

Patients with impaired heart rate recovery after exercise were classified into two subgroups: the first; (-blocker +ve), which included 50 patients on beta blocker (atenolol tablets) for six

months and, the second; (-blocker -ve) which included 50 patients on other antihypertensive drugs. Exercise ECG test was done again after six months for the patients of both subgroups.

Statistical analysis:

Continuous variables are summarized as a mean \pm standard deviation (SD). Comparison between the three groups of quintiles of the age of the patients with impaired heart rate recovery after exercise was performed by using the ANOVA test. Comparison between two groups was performed with t-test for continuous variables and chi-square test for categorical variables. A P-value < 0.05 was considered statistically significant and a P-value < 0.01 was considered statistically highly significant. A stepwise multivariate regression model was used to identify possible independent variables associated with impaired heart rate recovery after exercise in hypertensive patients. The strength of the association with impaired heart rate recovery is presented as 95% confidence intervals. Potential confounding clinical variables were entered as independent variables.

RESULTS

Clinical characteristics:

When considering age and gender, there was no significant difference between hypertensive and normotensive patients (53.31 ± 8.24 versus 48.6 ± 3.43 years, $P = \text{NS}$, 140 (82.4%) versus 70 (87.5%) males, $P = \text{NS}$ and 30 (17.6%) versus 10 (12.5%) females, $P = \text{NS}$, respectively). There was no significant difference between both groups with respect to the percentage of patients with history of smoking, hypercholesterolaemia and lipid lowering drug's use [100 (58.8%) versus 50 (62%) patients, $P = \text{NS}$, 90 (52.9%) versus 40 (50%) patients, $P = \text{NS}$, and 50 (29.4%) versus 30 (37.5%) patients, $P = \text{NS}$ respectively]. The hypertensive patients had a significant increased percentage of history of prior myocardial infarction than normotensives [20 (11.7%) versus six (7.5%) patients, $P < 0.05$]. There was no significant difference in the resting heart rate between both groups (89.25 ± 5.93 versus 78.5 ± 8.72 beat/minute, $P = \text{NS}$), but there was a significant increase in systolic and diastolic blood pressure in the hypertensive patients than in normotensives (179.5 ± 12.41 versus 122.35 ± 8.11 mm Hg, and 104.8 ± 5.32 versus 76.84 ± 6.18 mm Hg, respectively, $P < 0.05$). Among the quintiles of the age of the patients with impaired heart rate recovery, there was an insignificant difference in the number of the patients, gender, history of old myocardial infarction, ECG criteria of old myocardial infarction and smoking ($P = \text{NS}$) (Table 1).

Echocardiographic findings:

There were no significant difference between hypertensives and normotensives when evaluating left ventricular mass index (LVMI) and left ventricular systolic function (EF %) (127.3 ± 5.32 versus 119.4 ± 4.68 gm/m², $P = \text{NS}$ and 62.53 ± 4.26 versus 63.41 ± 2.18 %, $P = \text{NS}$ respectively), but there was a significant impaired diastolic function of the left ventricle in the hypertensives than normotensives as there was a significant decreased E/Aratio (0.88 ± 0.15 versus 1.51 ± 0.16 $P < 0.05$).

Among the quintiles of the age of the patients with impaired heart rate recovery after exercise, there was an insignificant difference regarding LVMI between the three quintiles and E/A ratio between the first and second quintiles ($P = \text{NS}$). There was a significant decrease in E/Aratio in the third quintile as compared with the first and the second quintiles ($P < 0.5$) (Table 1).

Among the hypertensive patients group, there was no significant difference between patients with normal heart rate recovery and those with impaired heart rate recovery after exercise when evaluating LVMI and EF [124.3 ± 5.37 versus 127.63 ± 4.28 gm/m², ($P = \text{NS}$) and 60.91 ± 3.28 versus 61.82 ± 4.22 %, ($P = \text{NS}$) respectively]. There was a significant decreased E/Aratio in the hypertensives with impaired heart rate recovery than those with normal heart rate recovery (0.81 ± 0.12 versus 1.08 ± 0.19 , respectively, $P < 0.05$) (Table 2).

Exercise ECG test:

There was an insignificant difference between hypertensives and normotensives in peak heart rate during exercise (155.8 ± 7.66 versus 165.4 ± 9.21 beats/minute, respectively, $P = \text{NS}$), but there was a significant increase in peak blood pressure and a significant decrease in the duration of the exercise

Table 1

Clinical characteristics and echocardiographic data among quintiles of the age of the hypertensive patients with impaired heart rate recovery after exercise

Variables	Age of the patients (years)			P-Value
	< 45 n (%)	45-55 n (%)	> 55 n (%)	
Number of patients)	30 (30)	30 (30)	40 (40)	NS
History of MI	5 (16)	7 (23)	8 (20)	NS
ECG criteria of MI	5 (16)	6 (20)	7 (17.5)	NS
Smoking	20 (66.6)	22 (73.3)	30 (75)	NS
No. and Gender (M/F)	27 M/3 F	26 M/4 F	37 M/3 F	NS
LVMI (gm/m ²)	120.2 ± 3.21	117.5 ± 5.23	129.7 ± 7.7	NS
E/Aratio	0.99 ± 0.03	0.94 ± 0.07	0.76 ± 0.11	*

*E/Aratio of the third quintile was significantly decreased ($P < 0.05$) than the other quintiles and no significant difference ($P = \text{NS}$) between the first and second quintiles

BMI = body mass index, ECG = electrocardiogram, F = female, M = male
LVMI = left ventricular mass index, n = number, MI = myocardial infarction.

test in hypertensives than normotensives (210.6 ± 8.31 versus 177.6 ± 10.45 mm Hg, $P < 0.05$ and 5.88 ± 1.62 versus 8.35 ± 2.27 minutes, $P < 0.05$) respectively. There was an insignificant difference in heart rate reserve between hypertensives and normotensives (85.87 ± 4.25 versus 95.43 ± 2.76 %, $P = \text{NS}$). There was also an insignificant increase in the number of hypertensive patients who had chronotropic incompetence during exercise than normotensives {57 (33.5%) versus 20 (25%) patients, respectively, $P = \text{NS}$ } table 3. There was a significant decrease in the decline of the heart rate among the hypertensive patients than normotensive subjects during recovery time ($p < 0.05$), but no significant difference after six minutes (Fig. 1). There was an insignificant difference when considering the heart rate reserve among patients with normal and impaired heart rate recovery (Fig. 4).

At a follow-up exercise, there was a significant decreased resting heart rate, peak heart rate and peak blood pressure in patients with beta-blockers than those without beta-blockers (65.9 ± 4.16 versus 79.7 ± 6.12 beats/minute, $P < 0.05$, 149.5 ± 8.29 versus 165.3 ± 6.76 beats/minute, $P = \text{NS}$, and 188.4 ± 10.52 versus 216.7 ± 9.45 mm Hg, $P = \text{NS}$ respectively). However, there was no significant difference between both subgroups when evaluated of exercise duration and heart rate reserve (5.88 ± 1.54 versus 6.28 ± 1.02 minutes, $P = \text{NS}$ and 81.51 ± 5.38 versus 89.41 ± 2.06 %, $P = \text{NS}$ respectively). There was an insignificant increase in the number of patients who had chronotropic incompetence during the exercise test among the patients with beta-blockers than those without beta-blockers {35 (70%) versus 30 (60%), respectively, $P = \text{NS}$ } and there was an insignificant difference between both subgroups in regards to positive or negative test results [six (12%) versus four (8%) respectively, $P = \text{NS}$], Table 4. There was a significant decrease in the decline of the heart rate during recovery in the hypertensive patients than the normotensive patients ($P < 0.05$), but there was an insignificant difference after six minutes of the recovery time ($P = \text{NS}$), Fig. 1. There was a significant increase in decline of the heart rate during recovery in patients with beta-blockers than those without beta-blockers ($P < 0.01$), but there was an insignificant difference after six minutes of the recovery time ($P = \text{NS}$), (Fig. 2). There was a significant increase in the heart rate during the first minute of recovery after exercise at follow up as compared with the results of baseline exercise ECG test ($P < 0.01$) while there was no significant difference between the baseline and the follow-up exercise test results in patients without beta-blockers ($P = \text{NS}$), Fig. 3.

Table 2

Echocardiographic data in hypertensive patients with normal and impaired heart rate recovery

Variable	Normal HRR N = 70	Impaired HRR N = 100	P Value
LVMI (gm/m ²)	124.3 ± 5.37	127.63 ± 4.28	NS
EF (%)	60.91 ± 3.28	61.82 ± 4.22	NS
E/Aratio	1.08 ± 0.19	0.81 ± .12	< 0.05

HRR=heart rate recovery, LVMI=left ventricular mass index

Table 3

Exercise ECG test data in hypertensive patients versus normotensive patients

Variable	Hypertensives N = 170	Normotensives N = 80	P Value
Resting HR (bpm)	78.4 ± 9.16	85.5 ± 6.12	NS
Peak HR (bpm)	155.8 ± 7.66	165.4 ± 9.21	NS
Peak BP(mmHg)	210.6 ± 10.41	176.7 ± 12.54	< 0.05
Exercise duration (min)	5.88 ± 1.62	8.31 ± 2.25	< 0.05
Heart rate reserve (%)	85.87 ± 4.25	95.43 ± 2.76	NS
Chronotropic incompetence	57 (33.5%)	20 (25%)	NS

BP=blood pressure, HR= heart rate, min=minute.

Table 4

Exercise ECG test data in hypertensive patients with and those without beta blocker at follow up after 6 months

Variable	B-blocker + ve N = 50	B-blocker-ve N = 50	P Value
Resting HR (bpm)	65.9 ± 4.16	79.7 ± 6.12	< 0.05
Peak HR (bpm)	149.5 ± 8.29	165.3 ± 6.76	< 0.05
Peak BP(mmHg)	188.4 ± 10.52	216.7 ± 9.45	< 0.05
Exercise duration (min)	5.88 ± 1.54	6.28 ± 1.02	NS
Heart rate reserve (%)	81.51 ± 5.38	89.41 ± 2.06	NS
Chronotropic Incompetence	40 (80%)	30 (60%)	< 0.05
Positive exercise test	6(12%)	4(8%)	NS

BP=blood pressure, HR= heart rate, min=minute.

There was an insignificant difference regarding decline in the heart rate in patients with normal heart rate recovery, and patients with impaired heart rate recovery between the patients who reached the target heart rate and those failed to reach the target heart rate ($P = \text{NS}$) (Fig. 4).

Among the quintiles of the patients with impaired heart rate recovery, there was an insignificant difference between the first and second quintiles and between the second and third quintiles ($P = \text{NS}$), but there was a significant decrease in the decline of the heart rate during the recovery after exercise in the third quintile, as compared with the first and the second quintiles ($P < 0.05$) (Fig. 5).

Stepwise logistic multivariate analysis revealed a significant relation between the age of the patients and left ventricular diastolic dysfunction and impaired heart rate recovery after exercise in hypertensive

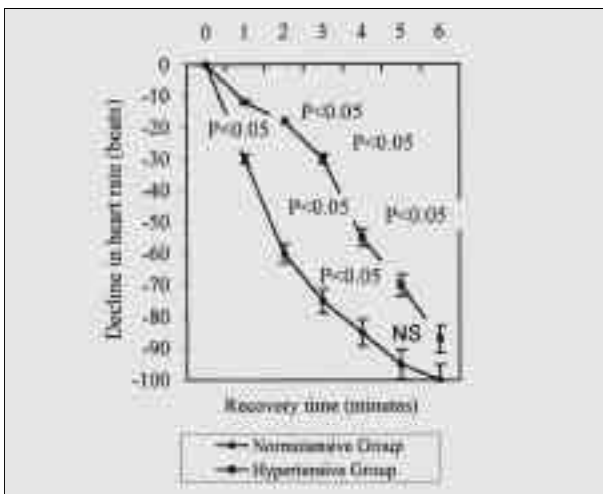


Fig. 1: Decline in heart rate after exercise in normotensive and hypertensive patients.

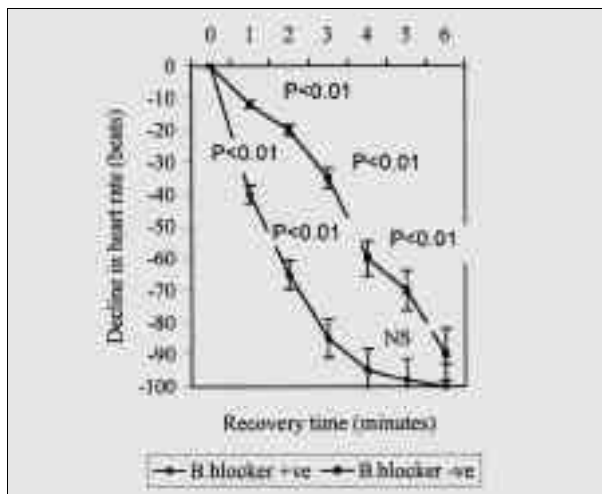


Fig. 2: Decline in heart rate after exercise in patients with and without beta blockers.

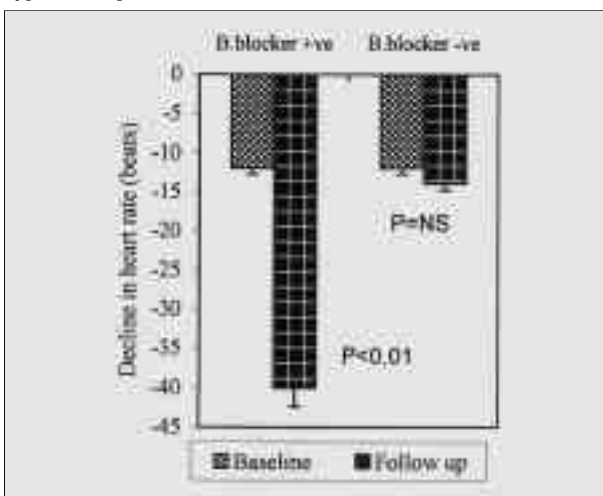


Fig. 3: Decline in heart rate after first minute recovery at baseline and follow-up in patients with versus without beta blockers.

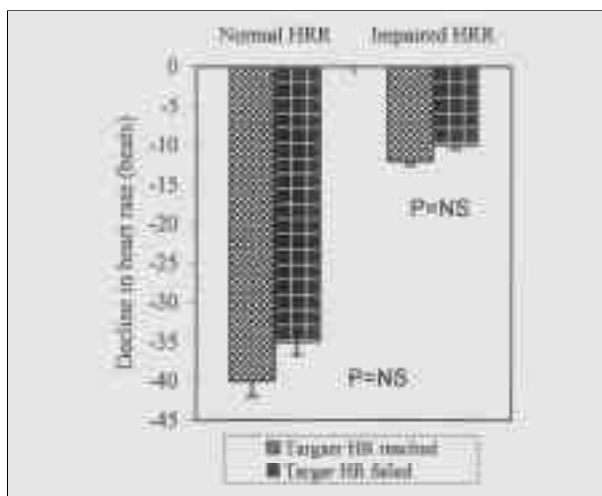


Fig. 4: Decline in heart rate in patients with normal and impaired heart rate recovery (HRR) among those who reached and those who failed to reach the target heart rate.

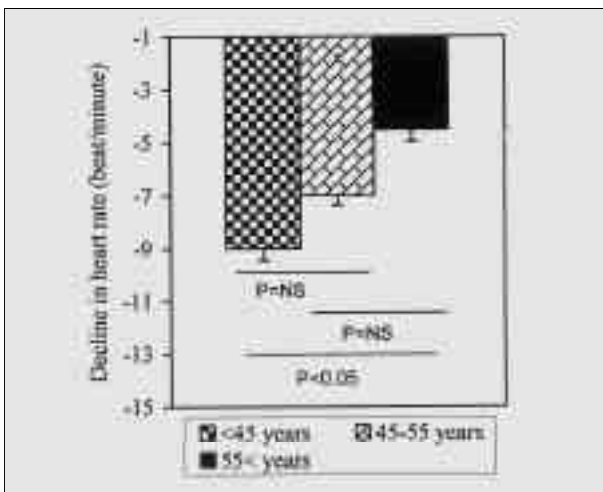


Fig. 5: Decline in heart rate among quintiles of the age of the patients with impaired heart rate recovery after exercise.

patients ($R = 0.1522$ and 0.1789), $95\% \text{ CI} = 1.054 - 1.731$ and $1.123 - 1.976$ respectively, $P < 0.05$). However, there was no significant relation when evaluating gender, smoking, left ventricle mass index and ST-segment depression during or after exercise (Table 5).

Table 5

Stepwise logistic analysis of hypertensive patients with-versus-without impaired heart rate recovery after exercise, with different variables

Variable	R	SE	P-value	95% CI
Age	0.1522	0.0978	< 0.05	1.054 - 1.731
Gender	0.0258	0.0789	NS	0.923 - 1.176
Smoking	0.0632	0.0649	NS	0.976 - 1.023
LVMI	0.0143	0.0174	NS	0.761 - 0.892
ST-depression	0.0678	0.0378	NS	0.654 - 1.531
LV Diast. Dys.	0.1789	0.0489	< 0.05	1.123 - 1.976

No. of observations = 170, CI = confidence interval, LV Diast Dys. = left ventricular diastolic dysfunction, LVMI = left ventricular mass index, R = regression coefficient, SE = standard error.

DISCUSSION

There was a strong correlation between the chronotropic variables (peak heart rate, percent peak heart rate and heart rate recovery percent) and the heart rate recovery after exercise in normal and coronary artery disease groups. Accordingly, heart

rate recovery depends greatly on the chronotropic response and chronotropic incompetence will lead to an abnormally slow heart rate recovery. To separate the heart rate response during the two phases of exercise (during exercise and in recovery), it is necessary to correct the effect of the chronotropic response on the heart rate recovery. This was performed using two chronotropic variables: percent peak and heart rate reserve percent. Based on these findings, it appears that much of the differences in the heart rate recovery between coronary artery disease and normal group can be explained by the heart rate recovery which is strongly dependent on the chronotropic response. These findings point toward the concept of accentuated antagonism (the higher the chronotropic response, the faster the recovery of heart rate after exercise and vice versa)^[14]. We found that the hypertensive patients have a significantly impaired heart rate recovery after exercise than normotensives, but there was no significant difference when evaluating chronotropic incompetence between both groups. Warner and Russell^[15] found that vagal stimulation alone resulted in a decrease of heart rate from 133 to 60 beats/minute in dogs. However, in the presence of sympathetic stimulation, the same vagal stimulation produced a greater decrease in heart rate (230 to 60 beats/minute), suggesting that the effect of vagal stimulation was enhanced when the sympathetic output was increased. It has also been reported that bilateral stellate ganglionectomy reduced the magnitude of the heart rate reduction by vagal stimulation in cats^[16]. Heart rate recovery in patients with orthotopic cardiac transplantation was significantly different from normal groups and patients with coronary artery disease. This seems to indicate the recovery pattern in the absence of an intact parasympathetic nervous system^[17]. In patients with cardiac transplantation, the heart rate recovery corrected for the heart rate reserve percent was significantly slower during the initial recovery phase until after about five minutes. This initial difference could very well represent the absence of an intact parasympathetic nervous system. Also, the correlation between the chronotropic variables and heart rate recovery in the normal subjects became stronger, later in the recovery period. These findings points to the possibility that heart rate recovery depends upon more than one mechanism involving the autonomic nervous system, a hypothesis that needs further study^[17,18,19].

The mechanism by which impaired heart rate recovery confers an increased risk of death, even among patients without heart failure or myocardial

perfusion defects, are not clear. Imai *et al*^[6] examined the physiologic characteristics of heart rate recovery after exercise in healthy adults, athletes and patients with chronic heart failure. They demonstrated that, in all groups of study (normal control group, coronary artery disease patients and transplant patients group) vagal reactivation was the principal determination of the decrease in heart rate during the first 30 seconds of recovery and that this mechanism was independent of age and the intensity of exercise. Heart rate recovery was rapid in athletes but was blunted in patients with heart failure and was completely abolished by the administration of atropine. Cole *et al*^[7] found a marked inverse association between heart rate recovery and exercise capacity. The Autonomic Tone and Reflexes after Myocardial Infarction Study was a large prospective multicenter study, in which patients who had myocardial infarction were stratified according to markers of autonomic control. Both markers used – variability in heart rate and baroreflex sensitivity – proved to be strong predictors of outcome. Cole *et al*^[7] reported that the value for the recovery of heart rate is a simple marker that is easily calculated on the basis of data already contained in a standard exercise test and does not require 24-hour Holter monitoring or specialized baroreflex sensitivity testing.

A low value for heart rate recovery after exercise testing, which has been previously shown to be a marker of decreased vagal activity, is a powerful and independent predictor of the risk of death. This marker is simple to calculate from data that are already contained in the results of standard exercise tests and may be valuable for the assessment of risk in routine clinical practice^[7,20]. Nishime *et al*^[8] found that when heart rate recovery decreases to less than 10 beats/minute from 12 beats/minute, risk of death increases markedly and the patients with both low-risk treadmill exercise scores and normal heart rate recovery had low risk of death. They also found that the heart rate recovery was not predictive of mortality among patients taking β -blockers. They reported that heart rate recovery provides additional prognostic information to the established treadmill exercise score and should be considered for routine incorporation into exercise test interpretation. Our results revealed a significant improved heart rate recovery after exercise test in hypertensive patients on β -blocker for six months. Lauer *et al*^[11] reported that chronotropic response into the routine interpretation of stress thallium studies may improve the prognostic power of the test. Nishime *et al*^[8] reported that it is not known, if heart rate recovery is a modifiable risk function, but Desai *et al*^[14] reported a significant impact of β -blockers on heart rate recovery.

We found that hypertensive patients with impaired heart rate recovery had a significant left ventricle diastolic dysfunction than those with normal heart rate recovery. Stepwise logistic multivariate analysis revealed a significant relation between the age of the patients and diastolic dysfunction and impaired heart rate recovery after exercise. Pitzalis *et al*^[10] reported that hypertensive patients show impaired baroreflex heart rate response and that this is particularly evident in those with functional and anatomic involvement.

Limitations of the study:

1. It is a single center experience.
2. Coronary angiography was not done for all patients to define whether they had coronary artery disease or not.
3. Blood pressure recovery after exercise was not included in the study, so further prospective study is needed to evaluate clinical significance of blood pressure recovery.

CONCLUSION

In hypertensive patients, the age and the left ventricular diastolic dysfunction are independent associate variables of impaired heart rate recovery after treadmill exercise ECG test. Beta-blockers affect heart rate in recovery, most likely due to an indirect effect by reduction of chronotropism. Incorporation of chronotropic response and heart rate recovery into the routine evaluation of the hypertensive patients may help in pharmacological management.

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