RESEARCH PAPER

Effects of progressive exercise during phase I cardiac rehabilitation on the heart rate variability of patients with acute myocardial infarction

Michele D. B. Santos-Hiss, Ruth C. Melo, Victor R. Neves, Flávio C. Hiss, Roberto M. M. Verzola, Ester Silva, Audrey Borghi-Silva, Alberto Porta, Nicola Montano, & Aparecida Maria Catai

1Department of Physiotherapy, Federal University of São Carlos, São Carlos, SP, Brazil, 2School of Arts, Science and Humanities, University of São Paulo, São Paulo, Brazil, 3Medical School of Ribeirão Preto, University of São Paulo, Ribeirão Preto, SP, Brazil, 4Department of Physiology, Federal University of São Carlos, São Carlos, SP, Brazil, 5Faculty of Health Science, Methodist University of Piracicaba, Piracicaba, SP, Brazil, 6Department of Technologies for Health, University of Milan, Galeazzi Orthopaedic Institute, Milan, Italy, and 7Department of Clinical Sciences, University of Milan, Internal Medicine II, L. Sacco Hospital, Milan, Italy

Accepted August 2010

Abstract

Purpose. Heart rate variability (HRV) decreases after an acute myocardial infarction (AMI) due to changes in cardiac autonomic balance. The purpose of the present study, therefore, was to evaluate the effects of a progressive exercise protocol used in phase I cardiac rehabilitation on the HRV of patients with post-AMI.

Material and methods. Thirty-seven patients who had been admitted to hospital with their first non-complicated AMI were studied. The treated group (TG, n = 21, age = 52 ± 12 years) performed a 5-day programme of progressive exercise during phase I cardiac rehabilitation, while the control group (CG, n = 16, age = 54 ± 11 years) performed only respiratory exercises. Instantaneous heart rate (HR) and RR interval were acquired by a HR monitor (Polar S810i). HRV was analysed by frequency domain methods. Power spectral density was expressed as normalised units (nu) at low (LF) and high (HF) frequencies, and as LF/HF.

Results. After 5 days of progressive exercise, the TG showed an increase in HFnu (35.9 ± 19.5 to 65.19 ± 25.4) and a decrease in LFnu and LF/HF (58.9 ± 21.4 to 32.5 ± 24.1; 3.12 ± 4.0 to 1.0 ± 1.5, respectively) in the resting position (p < 0.05). No changes were observed in the CG.

Conclusions. A progressive physiotherapeutic exercise programme carried out during phase I cardiac rehabilitation, as supplement to clinical treatment increased vagal and decreased sympathetic cardiac modulation in patients with post-AMI.

Keywords: Acute myocardial infarction, cardiac rehabilitation, heart rate variability

Introduction

Analysis of beat-to-beat heart rate variability (HRV) provides a simple, reproducible, and non-invasive method for quantifying the influence of the autonomic nervous system on the heart and, consequently, for identifying the presence of autonomic imbalance [1,2]. In general, HRV is analysed by time and frequency domain methods [2–5], which allow the characterisation of some conditions and/or diseases that affect cardiac autonomic control [3].

It is well established that autonomic imbalance plays an important role in the pathophysiology of cardiovascular diseases [6]. In patients with myocardial infarction, sympathetic hyperactivity has been shown to occur soon after the acute event [7]. Therefore, these patients have low HRV, which is characterised by decreased cardiac vagal modulation and consequent sympathetic predominance [8–10]. Because of its prognostic value, HRV has been used in several clinical trials with coronary artery disease (CAD) patients as an index for clinical outcome.
The literature has reported that reduced HRV is an important predictor for arrhythmic complications [1,2,8,9], as well as an independent mortality predictor associated with other post-acute myocardial infarction (AMI) risk factors (i.e., reduced ejection fraction and increased resting HR) [8–10,12]. On the other hand, a recent meta-analysis showed that HRV increases significantly in response to pharmacological interventions, biobehavioural treatments and exercise training in patients with CAD [11]. Although the exercise training in phase II outpatient cardiac rehabilitation improves the HRV of patients with CAD [13,14] and myocardial infarction [14,15], little is known about the effects of progressive exercise applied during phase I cardiac rehabilitation on the cardiac autonomic modulation of patients with post-AMI. Considering that cardiopulmonary rehabilitation provides significant improvement in health outcomes, reduced hospital admissions and length of hospitalisation, maintenance of the patient’s functional level, and improvement in quality of life and overall risk factor control through lifestyle change [16,17], it is possible that progressive exercise in the early phase of recovery could bring additional benefits to patients with post-AMI. Therefore, we hypothesised that progressive exercise during phase I cardiac rehabilitation in addition to clinical intervention might aid in the recuperation of cardiac autonomic balance in patients with post-AMI. Thus, the purpose of the present study was to evaluate the effects of phase I cardiac rehabilitation on the HRV of patients with post-AMI.

**Methods**

**Subjects**

One hundred and sixty-two patients (both genders) with AMI were admitted to the Coronary Care Unit (CCU) of a local hospital during the periods November 2004 through November 2005 and September 2006 through April 2007. Only fifty-nine (mean age: 53-years old) fulfilled the inclusion criteria, i.e., having suffered their first non-complicated AMI with ST-segment elevation. The exclusion criteria were as follows: a history of previous AMI, complicated AMI, AMI without ST-segment elevation, signs and/or symptoms of post-AMI chest pain or re-infarction, presence of diabetes mellitus associated with cardiac autonomic dysfunction, persistence of altered pressure response (refractory hypertension with levels greater than 180/100 mmHg), atrial fibrillation, malignant ventricular arrhythmias, complex ectopic ventricular beats, supraventricular or sinus tachycardia (greater than 120 beats per minute), 2° and 3° AV block; pacemaker implantation; signs of low output or ventricular failure, hypotension and heart failure; debility, fever, respiratory insufficiency, chronic obstructive pulmonary disease, illegal drug consumption, sequelae of stroke, lower limb amputation, severe aortic stenosis, severe left main coronary injury (>50%), prior coronary artery bypass graft surgery, inability to progress to next protocol step and hospital entry at least 48 h after the AMI event.

Initially, 59 patients were evaluated during the first day of hospitalisation, but only 40 were followed until hospital discharge. Because of noise in the electrocardiogram (ECG) signal, three patients were excluded before the data analysis. Thus, a total of 37 patients were studied, of which 16 belonged to control group (CG, 54 ± 11 years old) and 21 to the treatment group (TG, 52 ± 12 years old) (Figure 1). Among this sample, seven (19%) were submitted to thrombolyze with streptokinase (STK), 24 (65%) were submitted to primary percutaneous transluminal coronary angioplasty (PTCA) and six (16%) were treated with neither STK nor PTCA due to delayed admission to hospital (12 h after the beginning of chest pain). During hospitalisation, all patients were submitted to cardiac catheterisation, and 34 (92%) were successfully treated with PTCA (primary or elective).

**Procedures**

In accordance with the Helsinki Declaration, all patients were informed of the experimental procedures and signed an informed consent form approved by the Ethics Committee of the local institution (Process no. 023/2004 and no. 232/2006).

Clinical evaluations were based on daily clinical and physical examinations and laboratory tests, including: CK-MB enzyme concentration, total blood count, clinical biochemical screening (glucose), chest X-ray, and standard ECG, as well as cardiac catheterisation.

Experiments were always carried out in the afternoon. The patients were followed over 5 days, starting in the CCU (first two days) and concluding in the ward (remaining four days). The experimental protocol was initiated 22 ± 5 h after CCU admission for both groups, as recommended by Antman et al. [18]. Both groups were daily submitted to a standard protocol that included 10 min of rest in supine position followed by 4 min of respiratory exercises. During the resting period, the patients were always instructed to quietly relax, breathe spontaneously and remain awake. The respiratory exercises were done in the supine position and included diaphragmatic breathing pattern and deep breathing pattern. These respiratory exercises were only performed to
avoid possible pulmonary complication due to bed rest and hospitalisation. In addition, TG performed 5 days of phase I cardiac rehabilitation that consisted of a 5-step exercise programme, progressing from active assisted movements on the first day after AMI to walking in the final days of hospitalisation. This intervention was performed and supervised by a team of physical therapists.

The progression of TG through the exercise protocol was based on a daily clinical evaluation of each patient. Walking intensity was set at 20 beats per min above standing rest heart rate [16]. Additionally, if any signs and/or symptoms appeared during the progressive exercise training such as fatigue, chest pain, dyspnea, cyanosis, pallor, tachycardia (>120 beats per min), bradycardia, complex arrhythmias (i.e., those causing electrical and hemodynamic instability) or hypotension, the session was interrupted. However, none of these signs or symptoms occurred.

The instantaneous HR and RR interval were recorded during both standard and progressive exercise protocols with a digital telemetry system consisting of a transmitter placed on the patient’s chest and a HR monitor (Polar® S810; Polar Electro Oy, Kempele, Finland). The system detects ventricular...
depolarisation, corresponding to the R wave on the ECG, with a sampling rate of 500 Hz and a temporal resolution of 1 ms [19], and has been previously validated by Loimaala et al. [20]. After recording, the signals were transmitted to a receiver and interface connected to a computer for subsequent analysis. Additionally, blood pressure (BP) was measured each day by the auscultatory method before, during and after the standard and progressive exercise protocols. The medication dosage was noted daily and was not changed during the investigation in order to prevent any influence on the variables studied.

**Heart rate variability**

HR and RR interval data were obtained on the first (T0) and the sixth day of hospitalisation (T5) (i.e., after five days of progressive exercise training) during a 10-min period while subjects were in the resting supine position. Frequency domain analysis of HRV was performed with an autoregressive algorithm [21,22], which was applied to stable 256 RR interval series. The power spectral density was calculated for each RR series. Three spectral components were obtained: very low frequency (VLF), from 0 to 0.03 Hz; low frequency (LF), from 0.03 to 0.15 Hz; and high (HF), from 0.15 to 0.4 Hz. The spectral components were expressed in absolute (ms²) and normalised units and as low/high frequency ratio (LF/HF). Normalisation was computed by dividing the absolute power of a given spectral component (low or high frequency component) by the total power minus the power of the component, with a frequency range between 0 and 0.03 Hz (very low frequency), and then multiplying this ratio by 100 [21,22].

**Statistical analysis**

Data are reported as mean ± SE. Subject characteristics, medication dosages, HR, SBP, DBP and resting HRV indices at baseline (T0 = 1st day) were compared between groups using the t-test for independent samples. After that, the effect of time (T0 compared with T5 = 6th day), group (control compared with treated) and the interaction between time and group effects were evaluated by two-way ANOVA for repeated measures. When an interaction between time and group effects was found, the Student–Newman–Keuls Method was performed. Finally, intragroup analysis was performed using the paired t-test. These statistical analyses were carried out using Sigma Stat for Windows, version 2.03. The level of significance was set at \( p < 0.05 \).

**Results**

In each group studied, the autonomic heart rate modulation of patients with post-AMI was similar when divided by gender (male and female), ejection fraction (preserved and reduced), AMI topography (anterior and posterior) and clinical intervention received (with STK, without STK and with primary PTCA). Data were, therefore, pooled according to each condition (control or treatment). The patients’ characteristics are presented in Table I. No differences were found between CG and TG for these variables.

**Medications**

All administered medications were recommended in the ACC/AHA Guidelines [18] and the dosage of those that could affect HRV (β-blockers and angiotensin-converting enzyme-ACE inhibitors) was unchanged from the first (T0) to the sixth day of hospitalisation (T5) for both groups.

**Heart rate, blood pressure, respiratory frequency, mean R–R and variance**

In both groups, resting HR and systolic blood pressure (SBP) were high and mean R–R was lower

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Control group (n = 16)</th>
<th>Treatment group (n = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (male/female)</td>
<td>13/3</td>
<td>17/4</td>
</tr>
<tr>
<td>Age (years)</td>
<td>54 ± 11</td>
<td>52 ± 12</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>73 ± 15</td>
<td>72 ± 12</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165 ± 7</td>
<td>168 ± 7</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27 ± 5</td>
<td>26 ± 3</td>
</tr>
<tr>
<td>Coronary heart disease risk factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>11 (69%)</td>
<td>13 (62%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>8 (50%)</td>
<td>8 (38%)</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>9 (56%)</td>
<td>5 (24%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1 (6%)</td>
<td>3 (14%)</td>
</tr>
<tr>
<td>Overweight and obesity</td>
<td>10 (62%)</td>
<td>11 (52%)</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td>13 (81%)</td>
<td>9 (43%)</td>
</tr>
<tr>
<td>Sedentary</td>
<td>7 (44%)</td>
<td>10 (48%)</td>
</tr>
<tr>
<td>Stress</td>
<td>12 (75%)</td>
<td>14 (67%)</td>
</tr>
<tr>
<td>Infarction topography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>8 (50%)</td>
<td>14 (67%)</td>
</tr>
<tr>
<td>Inferior</td>
<td>8 (50%)</td>
<td>7 (33%)</td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B-blockers</td>
<td>15 (94%)</td>
<td>15 (71%)</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>16 (100%)</td>
<td>9 (43%)</td>
</tr>
<tr>
<td>Lipid-lowering</td>
<td>12 (75%)</td>
<td>14 (67%)</td>
</tr>
<tr>
<td>Aspirin</td>
<td>15 (94%)</td>
<td>20 (95%)</td>
</tr>
<tr>
<td>Ticlopidine/Clopidogrel</td>
<td>15 (94%)</td>
<td>19 (90%)</td>
</tr>
</tbody>
</table>

BMI, body mass index; CAD, coronary artery disease; ACE, angiotensin-converting enzyme.
on the first day than the sixth day (time effect, \( p < 0.05 \)). No additional effects (group and interaction) were observed for these variables. Diastolic blood pressure (DBP) was higher in TG than in CG (group effect, \( p < 0.05 \)), but remained within the normal range (<80 mmHg). The variance of R-R intervals and respiratory frequency presented no significant effects (time, group or interaction). Only TG patients showed a lower SBP at discharge than on their first day of hospitalisation (\( p < 0.05 \)) (Table II).

Heart rate variability

All HRV indices presented a time effect (\( p < 0.05 \)), suggesting that these indices changed during hospitalisation. Additionally, LFnu, HFnu and LF/HF were influenced by an interaction between time (T0 vs. T5) and group (control group vs. treated group) (\( p < 0.05 \)). Thus, the progressive exercise protocol was able to decrease the LFnu and LF/HF ratio, as well as increase the HFnu in TG (\( p < 0.05 \)). For CG, however, the HRV indices remained unchanged (Table II). Intragroup changes were observed only in TG, which presented a higher HFnu and a lower LFnu and LF/HF at discharge (day 6) than on the first day of hospitalisation (\( p < 0.05 \)) (Table II).

Discussion

These results demonstrate that five days of progressive exercise improves vagal modulation and decreases sympathovagal balance while in the resting supine condition in patients with post-AMI. Considering that attenuated HRV is an independent risk marker of premature morbidity and mortality among patients with CAD [11], the present findings indicate that, when applied as a supplement to clinical treatment, progressive exercise contributes a protective effect during early recovery from AMI.

The imbalance in autonomic cardiac function that accompanies an AMI can be observed up to 6 months after the event [9]. This fact can be attributed to ventricular remodelling, which is divided into two phases: early, which occurs within hours of AMI onset, and late, which occurs 72 h after onset. Early ventricular remodelling is characterised by infarct expansion, which results in ventricular rupture or aneurism. Later remodelling involves the entire left ventricle (LV) and is associated with progressive dilatation, LV architecture alteration and myocyte hypertrophy, causing increasing wall stress and damage to contractile function. Disturbances in circulatory hemodynamics activate the sympathetic adrenergic system, which stimulates catecholamine synthesis and activates the rennin–angiotensin–aldosterone system [23]. An increase in afferent sympathetic activity would cause reflex inhibition of vagal activity, leading to sympathetic modulation predominance on the sinus node [24]. Consequently, the structural alterations observed in post-infarction LV remodelling can cause persistently reduced HRV [23,24].

Abe et al. [24], studying patients after reperfused first anterior AMI, verified that an HRV index (SDNN) was independently associated with LV end-systolic volume index. These authors suggested that autonomic alteration consequent to post-infarction LV remodelling might result in depressed HRV.

### Table II. Cardiovascular and respiratory variables with heart rate variability data during resting supine condition for both groups studied.

<table>
<thead>
<tr>
<th></th>
<th>CG (n = 16)</th>
<th></th>
<th>TG (n = 21)</th>
<th></th>
<th>( p ) Values</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T0</td>
<td>T5</td>
<td>T0</td>
<td>T5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular and respiratory variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>71 ± 2.0</td>
<td>62 ± 2.0</td>
<td>69 ± 1.7</td>
<td>66 ± 1.7</td>
<td>( p = 0.006 )</td>
<td>NS</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>108 ± 3.3</td>
<td>100 ± 3.3</td>
<td>110 ± 2.9*</td>
<td>103 ± 2.9</td>
<td>( p = 0.029 )</td>
<td>NS</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>68 ± 2.3</td>
<td>67 ± 2.3</td>
<td>76 ± 2.0</td>
<td>71 ± 2.0</td>
<td>NS</td>
<td>( p = 0.042 )</td>
</tr>
<tr>
<td>RF (res/min)</td>
<td>19 ± 3.4</td>
<td>18 ± 2.8</td>
<td>19 ± 3.1</td>
<td>20 ± 2.5</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Time domain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (ms)</td>
<td>865 ± 0.15</td>
<td>981 ± 0.14</td>
<td>898 ± 0.1</td>
<td>942 ± 0.2</td>
<td>( p = 0.02 )</td>
<td>NS</td>
</tr>
<tr>
<td>Variance (ms(^2))</td>
<td>120 ± 12.2</td>
<td>109 ± 8.6</td>
<td>113 ± 15</td>
<td>60 ± 4.6</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Frequency domain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LF (nu)</td>
<td>51.4 ± 21.0</td>
<td>54.4 ± 22.4</td>
<td>58.9 ± 21.4*</td>
<td>32.5 ± 24.1</td>
<td>( p = 0.012 )</td>
<td>NS</td>
</tr>
<tr>
<td>HF (nu)</td>
<td>39.9 ± 19.4</td>
<td>39.3 ± 23.0</td>
<td>35.9 ± 19.5*</td>
<td>65.2 ± 25.4</td>
<td>( p = 0.036 )</td>
<td>( p = 0.005 )</td>
</tr>
<tr>
<td>LF/HF</td>
<td>2.2 ± 1.7</td>
<td>2.6 ± 2.3</td>
<td>3.1 ± 4.0*</td>
<td>1.0 ± 1.5</td>
<td>( p = 0.0034 )</td>
<td>( p = 0.003 )</td>
</tr>
</tbody>
</table>

CG: control group; TG: treatment group; T0 = control condition (1st day); T5 = after 5 days of cardiovascular physiotherapy treatment (6th day); HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; RF: respiratory frequency; LF: low frequency; HF: high frequency; nu: normalised units; ANOVA two way: T = time effect (T0 vs. T5); G = group effect (control group vs. treated group); I = interaction between group and time effects.

*\( t \)-test: significant difference T0 vs. T5 for TG.
cardiac autonomic modulation appears to involve the consequence of the progressive exercise protocol. Observed in TG at discharge could be seen as a modification in HRV indices. Thus, considering the studies previously cited and the fact that patients were submitted to six months of moderate-intensity exercise [27,28], it is unlikely that short-term exercise training, i.e., five days of progressive exercise, would be able to attenuate ventricular remodelling. In addition, available data on the benefits of exercise with respect to post-MI ventricular remodelling have come from studies carried out during phase II–III cardiac rehabilitation, in which patients were submitted to six months of moderate-intensity exercise [27,28]. Considering the points discussed above, the improvement in autonomic imbalance observed in TG was probably mediated by mechanisms other than an attenuation of ventricular remodelling.

It is also important to note that there are some factors responsible for HRV recovery in post-AMI: the spontaneous recuperation of HRV without medication in the months following the event [29,30]; the use of β-blockers (class I in AMI treatment) [18] and angiotensin-converting enzyme (ACE) inhibitors [3,31,32] and the association between β-blockade therapy and exercise training in phase II cardiac rehabilitation [15]. Lurje et al. [31] observed that β-blockade therapy increases HRV in patients with post-AMI. The authors followed up patients for 3 months (from the 3rd to 6th month after the event) and did not detect any additional increase in HRV, with medication dosages unaltered during the study. Similarly, Carpeggiani et al. [33] evaluated 349 patients with post-AMI who had been admitted to CCU and observed no HRV changes between their admission (HFnu = 27, LFnu = 54 and LF/HF = 3) and their discharge (HFnu = 29, LFnu = 57 and LF/HF = 3) (13 ± 7 days). Moreover, these patients received no physiotherapeutic intervention, and their doses of β-blockers and ACE inhibitors remained unchanged.

Therefore, our results agree with these authors, since no changes were observed in any HRV index of CG (6th month after the event) and did not detect any additional increase in HRV, with medication dosages unaltered during the study. Therefore, the improvement of HRV observed in TG at discharge could be seen as a consequence of the progressive exercise protocol.

The positive effect of physical exercise on cardiac autonomic modulation appears to involve adaptations in peripheral and central neural pathways [13]. Habitual exercise results in increased compliance in large arteries, which would act to augment pressure stimulus transductions, afferent responsiveness and, as consequence, baroreflex sensitivity [34]. Since arterial stiffness and baroreflex dysfunction would tend to reinforce sympathetic hyperactivity and potentially contribute to reduced HRV, associated improvements in HRV due to exercise training could be the result of both greater blood vessel distensibility and better signal transduction in barosensitive areas [35]. Besides its well-known influence on vascular homeostasis, nitric oxide may also be involved in cardiac autonomic control, since it exerts a facilitatory effect on afferent-mediated baroreflex activity in the central nervous system and also increases central and peripheral vagal neuronal activity [36]. However, the effects of exercise training on nitric oxide-mediated increases in vagal activity need further investigation. While it cannot be assumed that neuronal nitric oxide synthesis is influenced by physical exercise, accumulating scientific research data indicate that nitric oxide may modulate the vascular effects of physical training [36]. Hambrecht et al. [37] observed significant attenuation of coronary vasoconstriction in response to acetylcholine and shear stimuli in patients with CAD, suggesting an enhancement in the ability of the vascular endothelium to synthesize nitric oxide. Nevertheless, we can only speculate about the mechanisms by which exercise could be associated with increased HRV, mainly because most of the positive results came from studies involving greater periods and intensities of physical training [14,38,39] than those here presented. There is strong evidence that increased vagal modulation protects the heart against arrhythmias induced by cardiac electrophysiological imbalance [40]. The present findings showed that progressive exercise applied during early recovery from AMI decreased sympathovagal balance, which is a protective factor against cardiovascular complications.

The literature reports that bed rest causes a decrease in physical capacity, muscular tone, orthostatic tolerance and increased HR response to exercise [41]. Physiotherapeutic intervention by means of progressive exercise in phase I cardiac rehabilitation is an effort to decrease deleterious bed rest effects, evaluate the clinical response to increasing effort, establish the intensity of daily activities, reduce the length of hospitalisation and reduce cardiopulmonary complications [16,17,41]. Consequently, the progressive exercise protocol involves postural changes associated with a gradual increase in effort intensity, allowing early mobilisation of the bedridden patient (1st day) and preparing patients for a return to daily activities after discharge (6th day). In the present study, we observed that patients...
gradually adapted to each protocol stage, since they performed progressively higher intensity exercise in different postures. Finally, none of the TG patients showed signs and/or symptoms of exercise intolerance at any stage of progressive exercise training, and no clinical intercurrence was observed.

The present study has some limitations. First, bi-dimensional echocardiography was not performed due to logistic problems and, consequently, the process of ventricular remodelling could not be analysed. Second, due to the sample loss during the study, the final number of patients studied in each group was small. However, the number was sufficient to detect differences in HRV after the progressive exercise programme.

Conclusion

The present findings suggest that, as a supplement to clinical intervention, a progressive physiotherapeutic exercise protocol carried out during phase I cardiac rehabilitation caused an increase in vagal modulation and a decrease in sympathetic modulation and sympathovagal balance in the resting supine condition. Furthermore, the implemented progressive exercise protocol was well-tolerated by the patients and cause neither signs nor symptoms of exercise intolerance.

Acknowledgments

The authors are grateful to the clinical management of the local hospitals for authorising the collection of data at their institutions. This study was supported by the Fundação de Amparo a Pesquisa do Estado de São Paulo, Brazil (FAPESP, Proc. 04/05788–6; Proc.05/54838–9) and the Conselho Nacional de Desenvolvimento Científico e Tecnológico, Brazil (CNPq, Proc. 478799/2003–9; Proc.PDE: 200717/2008–1).

References