

## Effects of Physical Training on Cardiac Modulation in Normal Weight, Overweight, and Obese Individuals: A Comparative Study

Thaís HRDS, Gastaldi AC, Izabela CC, João EA, Suenimeire V and Hugo CDS\*

Exercise Physiology Laboratory, Department of Biomechanics, Medicine and Rehabilitation, School of Medicine of Ribeirão Preto, University of São Paulo, Brazil.

\*Corresponding author: Hugo CDS, Department of Biomechanics and Rehabilitation, School of Medicine of Ribeirão Preto, University of São Paulo, 14049-900, Ribeirão Preto, SP, Brazil, Tel: (55)16-3315-4416; (55)16-98175-1017 Fax: (55)16-3315-4413; E-mail: [hugocds@fmrp.usp.br](mailto:hugocds@fmrp.usp.br)

Rec date: Oct 08, 2015; Acc date: Nov 17, 2015; Pub date: Nov 23, 2015

Copyright: © 2015 Thaís HRDS, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Abstract

Our study has assessed the effect of aerobic physical training on the heart rate variability (HRV) on sedentary women with different body mass indices (BMI; weight/height<sup>2</sup>). Forty-eight volunteers were divided into three groups according to their BMI as follows: NW group (normal weight), 18.0-24.9; OW group (overweight), 25.0-29.9; and OB group (obese), 30.0-39.9. HRV was assessed with the subjects at rest and during tilt test by means of spectral analysis before and after 16 weeks of an aerobic physical training protocol.

Prior to the aerobic physical training, OW and OB groups exhibited decrease in low frequency (LF, 0.4-0.15 Hz) and high frequency (HF, 0.15-0.5 Hz). After aerobic physical training all groups had similar HF oscillations, with only OB group exhibiting increase in LF oscillations. The HRV responses to tilt test obtained before and after aerobic physical training showed that NW group had no differences in LF ( $34 \pm 6\%$  vs.  $36 \pm 8\%$ ) and HF ( $-65 \pm 6\%$  vs.  $-60 \pm 7\%$ ) oscillations. However, OW group had an increase in LF ( $46 \pm 6\%$  vs.  $86 \pm 14\%$ ) and HF ( $-44 \pm 7\%$  vs.  $-61 \pm 7\%$ ) oscillations, whereas OB group had a decrease in LF ( $288 \pm 25\%$  vs.  $159 \pm 16\%$ ) and HF ( $-83 \pm 5\%$  vs.  $-70 \pm 4\%$ ) oscillations.

In conclusion, our results suggest that the increase in the percentage of body fat may impair the cardiac autonomic modulation in proportion to BMI, which is an important predictor of cardiovascular morbidity and mortality. In turn, the physical exercise can attenuate this negative effect, regardless of the reduction of body fat mass.

**Keywords:** Obesity; Physical training; Heart rate variability; Spectral analysis

### Introduction

Obesity is normally associated to the metabolic perturbations and it can be also accompanied by cardiovascular autonomic dysfunctions [1]. In this sense, analysis of the autonomic modulation of heart rate variability (HRV) has demonstrated that excess in body fat mass might promote concomitant reduction of LF and HF oscillations, characterizing low autonomic sympathetic and parasympathetic cardiac influences and consequently increasing the risk of cardiac morbidity and mortality [2-7].

Therefore, obesity and overweight have a relevant impact on longevity, and any kind of intervention aiming to reduce the body fat percentage yields long-term benefits that trend to be greater with earlier intervention [8].

By this reason the main recommendations regarding programs for reduction of body fat percentage are the following: reduction of energetic support, decrease in dietary fat ratio, and practice of physical training [9]. Hypocaloric diet yields expressive caloric deficit that leads to reduction of body weight, whereas physical training promotes important physiological adaptations, mainly metabolic and cardiovascular ones, thus reducing the risk of morbidity and mortality [10-12]. Additionally, some studies have shown evidence that improvement in cardiovascular autonomic control from physical

training would be one of the main adaptations, playing a positive role in energetic balance related to obesity subjects [2,13].

In fact, the benefits of aerobic physical training on cardiac autonomic control have already been documented by both experimental and clinical studies on different chronic degenerative diseases, such as hypertension [14-16]. In this sense, amongst the several cardiac autonomic function benefits, the regular physical exercises practice may promote a decrease in the sympathetic autonomic influence and an increase in the vagal influence [17-19]. In turn, the influence of physical training on cardiovascular autonomic control in women with different body fat percentages is not well known in the literature, specially related to the repercussion of life style changes on populations with no related comorbidities.

Therefore, the objective of the present study was to investigate and compare normal weight, overweight, and obese women presenting no comorbidities regarding the effects of aerobic physical training on autonomic modulation of HRV in different situations, that is, at rest in the supine position and during tilt test in order to evaluate the adaptative modulation response to posture changes.

### Methods and Procedures

#### Sampling

All participants were screened at the Exercise Physiology Laboratory of the Department of Biomechanics, Medicine, and Rehabilitation, School of Medicine of Ribeirão Preto, Brazil. Forty-

eight sedentary women with mean age of  $42 \pm 2$  years old were included for study. All subjects were physically inactive and were in reproductive phase, were non-smokers, had no signs or history of overt heart disease and had no muscular-skeletal alteration impeding the practice of physical training. We also excluded subjects that were taking medications known to alter heart rate, treated for cancer or diagnosed with chronic conditions (e.g., lupus, etc.).

The volunteers participants were divided into three groups according to their body mass index (BMI) as follows: NW group, normal weight ( $18.0\text{-}24.9 \text{ kg/m}^2$ ); OW group, overweight ( $25.0\text{-}29.9 \text{ kg/m}^2$ ); and OB group, obese ( $30.0\text{-}39.9 \text{ kg/m}^2$ ). This study was conducted in accordance with the ethical standards set forth in the Helsinki Declaration of 1975 and approved by the Human Research Ethics Committee of the Clinical Hospital of the School of Medicine of Ribeirão Preto, University of São Paulo, Brazil (case HCRP N. 13475/2009). The evaluations were performed before and after the physical training period and all volunteers provided their free and informed consent.

### Training protocol

All participants were submitted to supervise aerobic physical training protocol on a motor-driven treadmill 3 days a week during 16 weeks, with each session lasting 45 minutes. Cardiac rate was determined at 5% below respiratory anaerobic threshold by using ergospirometric testing (modified Bruce's protocol). At the end of the 16-week training phase, the aerobic capacity was evaluated again.

### Laboratory exams and body composition

Laboratory exams were conducted for serum glucose, triglyceride, and total/fractional cholesterol levels before and after the 16-week protocol. The volunteers were instructed to fast for 12 hrs, avoid alcoholic beverages 48 hrs before the exams, practice no physical activity, and keep their habitual diet. Their body composition was determined by means of bioimpedance analysis.

### Analysis of heart rate variability (spectral analysis)

The participants were also instructed to ingest no beverage containing either alcohol or caffeine, practice no physical activity, and keep their habitual diet for 48 hrs prior to the exam. Spectral analysis of HRV was performed from 9 o'clock a.m. to 10 o'clock a.m. according to the following protocol: the subjects were put in the supine position at rest on a tilting orthostatic table for 20 minutes, and then they were

passively put in 75-degree inclined position for further 20 minutes. HRV was measured at supine position and during tilt test by means of electrocardiography (ADinstruments, Australia), where time series were obtained from the time elapsed between adjacent R-R waves (RRi). Time series were divided into contiguous segments of 200 beats and then superposed over the segments of 100 beats from the earlier time series. After calculating mean and variation of each segment, these were submitted to autoregressive spectral analysis as described above [20-23]. The oscillatory components present in the stationary segments, beat-to-beat RRi, were calculated based on the Levinson-Durbin recursion according to the Akaike's criteria [20]. This procedure allows automatic quantification of both central frequency and influence of each relevant oscillatory component in the interval series. The oscillatory components were classified as low (LF) and high frequencies (HF), with oscillations occurring at frequency bands of 0.04-0.15 Hz and 0.15-0.5 Hz, respectively. The power of LF and HF components in the RRi variability was also expressed in normalized units, which were obtained by calculating the percentage of LF and HF variability oscillations ( $\text{HRV} < 0.04 \text{ Hz}$ ). The normalization procedure tends to minimize the effect of total force changes on absolute values in the variability of LF and HF components [20-23]. Finally, the LF/HF ratio was calculated to establish an index of autonomic modulation.

### Statistical analysis

Data are reported as mean  $\pm$  standard deviation (SD) of mean. The results of laboratory tests, body composition, and spectral analysis were assessed by two-way ANOVA followed by Tukey's post-hoc test. Significant differences were considered when  $p < 0.05$ .

### Results

Table 1 show all the values regarding subjects' characteristics as well as metabolic values and basal hemodynamic values before and after the 16-week aerobic physical training protocol. No significant differences in both age and height were observed between the groups studied. All groups exhibited differences in weight, BMI, and body fat percentage. In this sense, OB group had the highest values, whereas NW group had lower values for all variables. With regard to hemodynamic values, basal heart rate was similar in all groups before and after physical training, but only OB group showed a reduction after the 16-week protocol. As for the arterial pressure (AP), despite the fact that all subjects were normotensive, NW group showed the lowest values for all pre-training variables compared to other groups. Aerobic exercise training did not reduce AP in either group.

Participants	Normal weight		Overweight		Obese	
	Sedentary	Trained	Sedentary	Trained	Sedentary	Trained
Subjects characteristics						
Age, years	$34 \pm 7$	----	$32 \pm 6$	----	$33 \pm 7$	----
Height, m	$1.64 \pm 0.07$	----	$1.64 \pm 0.08$	----	$1.65 \pm 0.06$	----
Weight, kg	$63 \pm 6$	$62 \pm 5$	$75 \pm 7^a$	$74 \pm 6$	$100 \pm 8^{ab}$	$96 \pm 7$
BMI, $\text{kg/m}^2$	$22.9 \pm 2.4$	$22.7 \pm 2.2$	$27.9 \pm 2.9^a$	$27.5 \pm 2.7$	$37.3 \pm 3.7^{ab}$	$36.5 \pm 3.6$
Fat mass, %	$24.4 \pm 1.8$	$24.3 \pm 1.9$	$34.4 \pm 3.3^a$	$33.3 \pm 2.2$	$43.2 \pm 3.9^{ab}$	$41.4 \pm 2.9$

Hemodynamic values						
Heart Rate, bpm	72 ± 12	69 ± 9	69 ± 7	67 ± 8	74 ± 7	67 ± 6 <sup>c</sup>
Systolic AP, mmHg	107 ± 7	103 ± 8	117 ± 6 <sup>a</sup>	111 ± 9	121 ± 6 <sup>a</sup>	118 ± 7
Diastolic AP, mmHg	71 ± 6	69 ± 6	81 ± 5 <sup>a</sup>	76 ± 8	82 ± 7 <sup>a</sup>	78 ± 8
Mean AP, mmHg	85 ± 6	83 ± 7	95 ± 5 <sup>a</sup>	90 ± 8	98 ± 6 <sup>a</sup>	94 ± 7
Metabolic values						
VO <sub>2 peak</sub> , mL.kg <sup>-1</sup> .min <sup>-1</sup>	30.2 ± 2.9	33.4 ± 3.0 <sup>a</sup>	28.2 ± 2.1	31.7 ± 2. <sup>b</sup>	22.4 ± 2.9 <sup>ab</sup>	26.0 ± 2.8 <sup>c</sup>
Total Cholesterol, mg/dL	189 ± 19	179 ± 12	209 ± 38	204 ± 30	209 ± 16	202 ± 26
HDL, mg/dL	51 ± 8	49 ± 7	48 ± 11	50 ± 9	43 ± 7	46 ± 7
LDL, mg/dL	103 ± 9	101 ± 10	111 ± 14	109 ± 13	112 ± 8	111 ± 16
Triglycerides, mg/dL	107 ± 21	105 ± 9	114 ± 14	109 ± 17	124 ± 37	108 ± 25
Glucose, mg/dL	84 ± 6	82 ± 5	94 ± 7	92 ± 6	94 ± 8	91 ± 6

**Table 1:** Clinical characteristics of study participants before and after physical training. All values were expressed as mean ± SD. <sup>a</sup>P<0.05 vs. NW sedentary; <sup>b</sup>P<0.05 vs. OW sedentary; and <sup>c</sup>P<0.05 vs. OB sedentary.

With regard to metabolic values, all the groups showed similar results in almost all variables before and after physical training, except for VO<sub>2peak</sub>, where the groups studied had an increase after the 16-week protocol. However, when compared, OB group showed the lowest values compared to other groups before and after the physical training.

### Spectral analysis of HRV supine rest position

Table 2 shows spectral parameters obtained in the supine rest position in all groups studied before and after physical training. Before physical training, it was observed that OW group had a decrease in LF

oscillations in absolute and normalized units as well as an increase in HF oscillations in normalized units compared to NW group. In turn, OB group showed a reduction in total variance and LF oscillations in absolute and normalized units compared to other groups, besides showing a decrease in HF oscillations in absolute units compared to NW group and an increase in normalized units compared to NW and OW groups. Additionally, it was observed that OW and OB groups had significant reduction in LF/HF ratio compared to NW group, with OB group showing the lowest value.

Parameters	Normal weight		Overweight		Obese	
	Sedentary	Trained	Sedentary	Trained	Sedentary	Trained
RRI, ms	834 ± 122	872 ± 88	868 ± 97	890 ± 81	815 ± 78	899 ± 66 <sup>c</sup>
Spectral Parameters, RRI						
Variance, ms <sup>2</sup>	1930 ± 489	1922 ± 436	1524 ± 323	1511 ± 434	917 ± 389 <sup>ab</sup>	1521 ± 406
LF, ms <sup>2</sup>	895 ± 184	629 ± 205	311 ± 195 <sup>a</sup>	301 ± 119	69 ± 72 <sup>ab</sup>	219 ± 99 <sup>c</sup>
LF, nu	66 ± 12	63 ± 10	52 ± 11 <sup>a</sup>	47 ± 14	29 ± 18 <sup>ab</sup>	33 ± 9
HF, ms <sup>2</sup>	457 ± 132	364 ± 46	317 ± 94	441 ± 170	208 ± 135 <sup>a</sup>	425 ± 141 <sup>c</sup>
HF, nu	34 ± 12	37 ± 10	48 ± 11 <sup>a</sup>	53 ± 14	71 ± 18 <sup>ab</sup>	67 ± 9
LF/HF ratio	2.1 ± 0.39	1.8 ± 0.33	1.1 ± 0.51 <sup>a</sup>	0.8 ± 0.51	0.4 ± 0.36 <sup>ab</sup>	0.5 ± 0.31

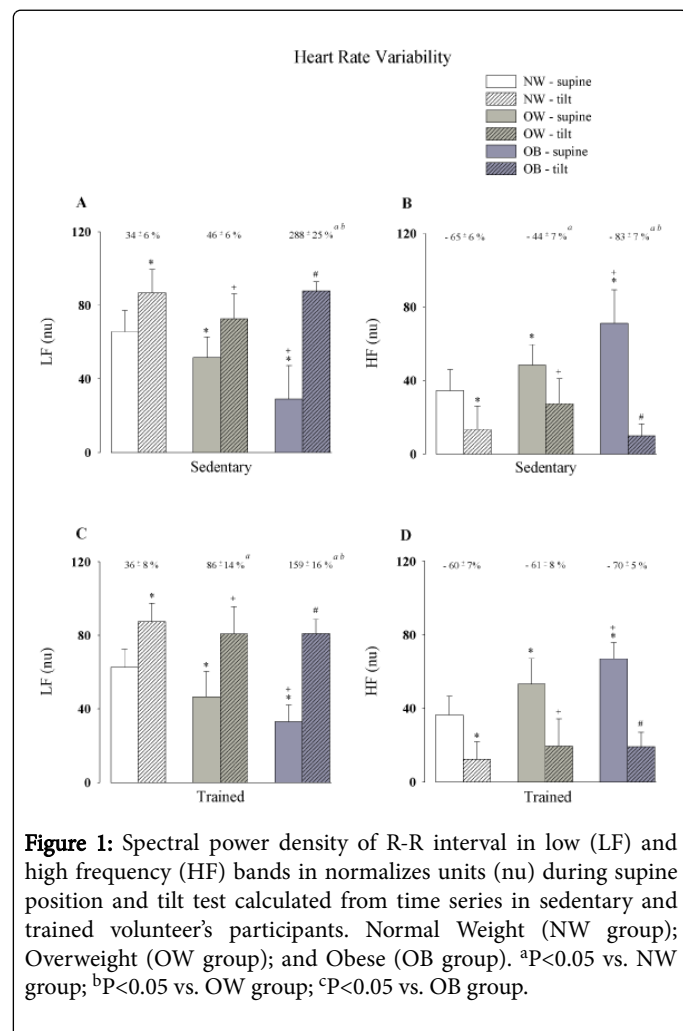
**Table 2:** Basal spectral parameters calculated from R-R interval (RRI) time series obtained in the supine position in normal weight (NW), overweight (OW) and obese (OB) participants. All values were expressed as mean ± SD. <sup>a</sup>P<0.05 vs. NW sedentary; <sup>b</sup>P<0.05 vs. NW trained; and <sup>c</sup>P<0.05 vs. OW sedentary. “nu” indicates normalized units.

Physical training did not change spectral parameters in NW and OW group in the supine position at rest. On the other hand, OB group showed increase in LF and HF oscillations, but only in absolute units.

### Spectral analysis of HRV-tilt testing

Table 3 shows the tilt test results for all groups of participants before the 16-week physical training (sedentary). Only OB group had an

increase in LF oscillations in absolute values. In turn, all groups studied had an increase in LF oscillations in normalized units (Figure 1), but OB group showed response percentage ( $288 \pm 25\%$ ) of basal supine values significantly greater than those from NW ( $34 \pm 6\%$ ) and OW ( $46 \pm 6\%$ ) groups.



With regard to HF oscillations, all groups presented reduction in absolute and normalized units, but when the response percentage was quantified (Figure 1), we observed differences between all groups (NW,  $-65 \pm 6\%$ ; OW,  $-44 \pm 7\%$ ; OB,  $-83 \pm 7\%$  groups).

After physical training (Table 4), we observed no change in the response of total variance and LF oscillations in absolute units, but all groups had different percentage increases in oscillations in normalized units (NW,  $36 \pm 8\%$ ; OW,  $86 \pm 14\%$ ; OB,  $159 \pm 16\%$  groups) as shown in Figure 1. In turn, HF oscillations were found to be reduced in all groups, both in absolute and normalized units (Table 4), but when tilt response percentage was compared (Figure 1), we observed no differences between the groups studied (NW,  $-60 \pm 7\%$ ; OW,  $-61 \pm 8\%$ ; OB,  $-70 \pm 5\%$  groups).

We have also observed that NW group showed no differences in LF ( $34 \pm 5$  vs.  $36 \pm 8\%$ ) and HF ( $-65 \pm 6$  vs.  $-60 \pm 7\%$ ) oscillations when HRV response percentages obtained before and after the 16-week training period were quantified. On the other hand, OW group had an increase in LF ( $46 \pm 6$  vs.  $86 \pm 14\%$ ) and HF ( $-44 \pm 7$  vs.  $-61 \pm 8\%$ ) oscillations, whereas OB group had a decrease in both, respectively,  $288 \pm 25$  vs.  $159 \pm 16\%$  and  $-83 \pm 5$  vs.  $-70 \pm 4\%$ . Figure 2 shows representative spectra of RRI for all groups studied in the supine position and during tilt test, before and after physical training.

## Discussion

The present study has shown that overweight and obese sedentary women had changes in HRV modulation, characterized by concomitant reduction in LF and HF oscillations. The main finding of this study was that aerobic physical training have induced HF oscillations in obese and overweight women, whose values were similar to those of normal weight women. However, physical training did not change LF oscillations in overweight women, only promoting a small increase in obese women, remaining further below to that values observed in normal weight women.

The increase in body fat seems to impair significantly the cardiac autonomic modulation, since our study indicates that LF and HF oscillations are inversely related to body fat percentage [24]. However, this relationship seems to be more prominent for LF oscillations in the obese women group, since they had values 15 times lower than those observed for the group of normal body fat percentage. In this sense, the use of normalized data in order to minimize the effect of total power of absolute LF and HF values on HRV does not seem to apply to obese patients, including the LF/HF ratio, as we had already observed a paradox in which overweight and obese women had more expressive HF oscillation values in normalized units. Also, the LF/HF ratio suggests a more adequate modulation of autonomic balance compared to women with normal weight.

Parameters	Normal weight		Overweight		Obese	
	Supine	Tilt	Supine	Tilt	Supine	Tilt
RRI, ms	834 $\pm$ 122	640 $\pm$ 27 <sup>a</sup>	868 $\pm$ 97	716 $\pm$ 23 <sup>b</sup>	815 $\pm$ 78	735 $\pm$ 18 <sup>c</sup>
Spectral Parameters; RRI						
Variance, ms <sup>2</sup>	1930 $\pm$ 489	1692 $\pm$ 363	1524 $\pm$ 323	887 $\pm$ 444	917 $\pm$ 389 <sup>ab</sup>	1241 $\pm$ 382
LF, ms <sup>2</sup>	895 $\pm$ 184	911 $\pm$ 294	311 $\pm$ 195 <sup>a</sup>	257 $\pm$ 84	69 $\pm$ 72 <sup>ab</sup>	203 $\pm$ 87 <sup>c</sup>
LF, nu	66 $\pm$ 12	87 $\pm$ 13 <sup>a</sup>	52 $\pm$ 11 <sup>a</sup>	73 $\pm$ 14 <sup>b</sup>	29 $\pm$ 18 <sup>ab</sup>	88 $\pm$ 5 <sup>c</sup>
HF, ms <sup>2</sup>	457 $\pm$ 132	146 $\pm$ 99 <sup>a</sup>	317 $\pm$ 94	94 $\pm$ 41 <sup>b</sup>	208 $\pm$ 135 <sup>a</sup>	46 $\pm$ 19 <sup>c</sup>

HF, nu	34 ± 12	13 ± 13 <sup>a</sup>	48 ± 11 <sup>a</sup>	27 ± 14 <sup>b</sup>	71 ± 18 <sup>ab</sup>	12 ± 5 <sup>c</sup>
LF/HF ratio	2.1 ± 0.39	6.9 ± 1.4 <sup>a</sup>	1.1 ± 0.51 <sup>a</sup>	4.3 ± 1.2 <sup>b</sup>	0.4 ± 0.36 <sup>ab</sup>	5.1 ± 1.1 <sup>c</sup>

**Table 3:** Spectral parameters calculated from R-R interval (RRI) time series obtained in supine position and during the tilt test in normal weight (NW), overweight (OW) and obese (OB) sedentary participants. All values were expressed as mean ± SD. <sup>a</sup>P<0.05 vs. NW supine; <sup>b</sup>P<0.05 vs. NW supine; and <sup>c</sup>P<0.05 vs. OW supine. “nu” indicates normalized units.

Parameters	Normal weight		Overweight		Obese	
	Supine	Tilt	Supine	Tilt	Supine	Tilt
RRI, ms	872 ± 88	653 ± 29 <sup>a</sup>	868 ± 97	710 ± 22 <sup>b</sup>	899 ± 66	758 ± 46 <sup>c</sup>
Spectral Parameters; RRI						
Variance, ms <sup>2</sup>	1922 ± 436	1414 ± 332	1511 ± 433	877 ± 414	1521 ± 406	1574 ± 318
LF, ms <sup>2</sup>	629 ± 205	648 ± 147	301 ± 119 <sup>a</sup>	317 ± 75	219 ± 99 <sup>a</sup>	431 ± 192
LF, nu	63 ± 10	87 ± 10 <sup>a</sup>	47 ± 14 <sup>a</sup>	81 ± 15 <sup>b</sup>	33 ± 9 <sup>a</sup>	81 ± 8 <sup>c</sup>
HF, ms <sup>2</sup>	364 ± 46	94 ± 33 <sup>a</sup>	441 ± 170	74 ± 22 <sup>b</sup>	425 ± 141	84 ± 92 <sup>c</sup>
HF, nu	37 ± 10	13 ± 10 <sup>a</sup>	53 ± 14 <sup>a</sup>	19 ± 15 <sup>b</sup>	67 ± 9 <sup>ab</sup>	19 ± 8 <sup>c</sup>
LF/HF ratio	1.8 ± 0.33	7.3 ± 1.3 <sup>a</sup>	0.8 ± 0.51 <sup>a</sup>	4.1 ± 1.2 <sup>b</sup>	0.5 ± 0.31 <sup>ab</sup>	5.3 ± 1.2 <sup>c</sup>

**Table 4:** Spectral parameters calculated from R-R interval (RRI) time series obtained all trained volunteers in supine position and during the tilt test using autoregressive spectral analysis. All values were expressed as mean ± S.E.M. a P<0.05 vs. NW supine; b P<0.05 vs. NW supine; and c P<0.05 vs. OW supine. “nu” indicates normalized units.

Similarly, the tilt test also showed very interesting results. The group of obese women had a small increase in the LF oscillations as well as almost total suppression of HF oscillations during tilt test, in absolute units, even with baseline HF oscillations very reduced. In turn, the highest LF oscillations (288%) and the lowest HF oscillations (-83%) were observed in percentage values. The group of overweight women showed intermediate values ranging between those of obese women and those of normal weight women, indicating that the impairment of autonomic modulation of HVR is related on body fat percentage.

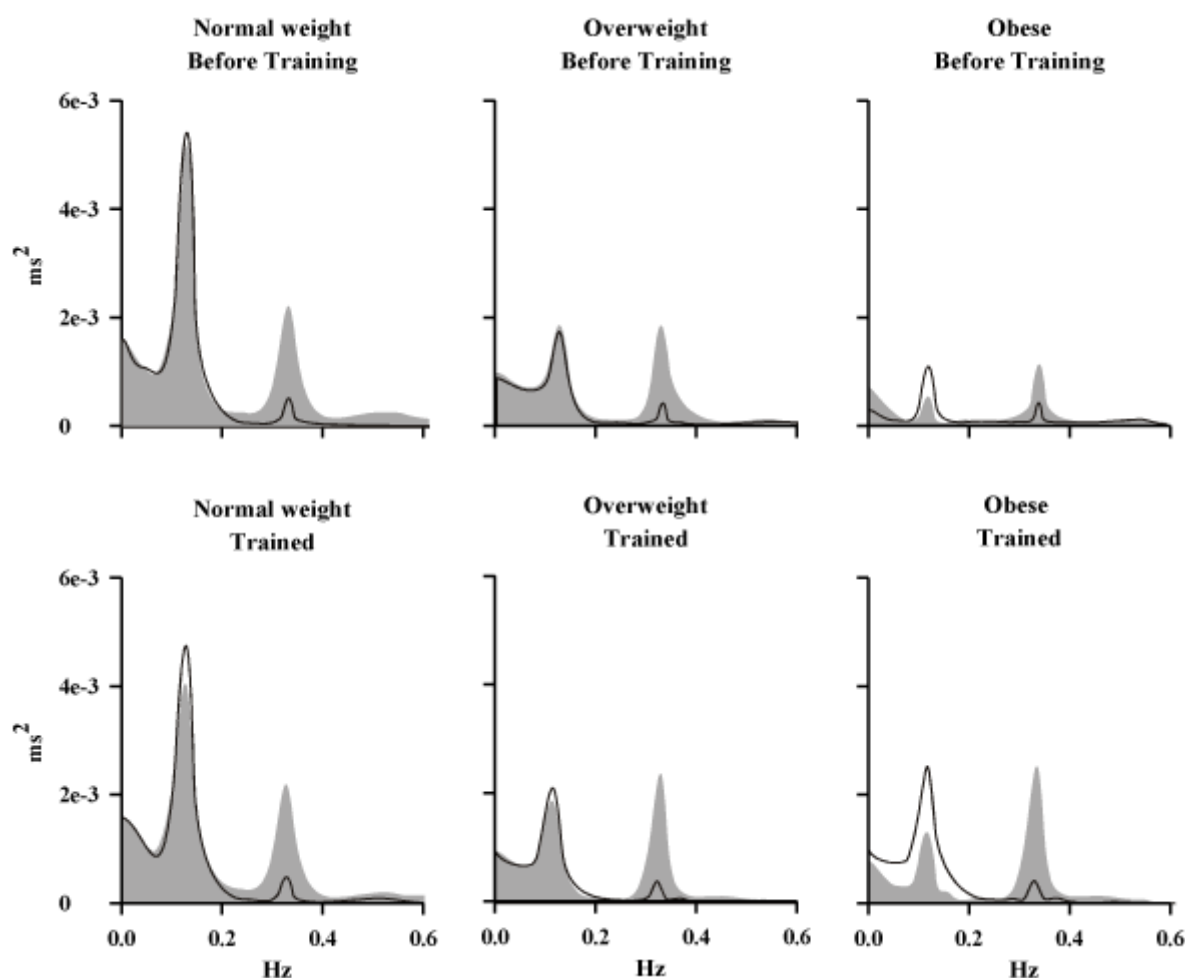
The causes of such disorders are not fully known. One of the hypotheses is that the development of obesity is accompanied by a progressive reduction in the sympathetic autonomic activity, resulting in a low basal metabolism [5]. In addition, some authors have also pointed to the occurrence of a concomitant increase in the parasympathetic autonomic component activity, thus contributing directly to a hyperplasia and hyperinsulinemia [15]. Therefore, this hypothesis suggests that changes in the autonomic nervous system may also contribute to the development of obesity [7]. The origin of this phenomenon is unknown and it has been suggested that low cardiac autonomic sympathetic activity in obese individuals might have a cardio-protective function, although the impact on the total of energy expense is controversial [6,19]. In addition, it is also important to emphasize that the impact of autonomic modulation on the *arrhythmogenesis* in obesity is still unclear, in part, because the cardiac vagal modulation was found to be decreased in our study, thus corroborating findings elsewhere [18,25].

Other possible causes of HVR modulation dysfunction is related to a decrease in the cardiac adrenergic receptor sensibility, lack of the

parasympathetic tonus and/or increase in the sympathetic activity, or reduction in the baroreflex sensibility [26-28]. Additionally, indirect evaluation of the sympathetic autonomic influence on the heart by means of plasma concentration of catecholamines (i.e., cardiac NE spillover rate) showed that normotensive obese individuals have serum levels 50% lower than those of normotensive individuals with normal weight. On the other hand, the cardiac autonomic sympathetic influence was twice higher in obese hypertensive individuals than in obese normotensive ones and 25% higher compared to normal weight individuals [1,6].

In fact, all these results show that the causes of impairment in cardiac autonomic modulation are still unclear, thus requiring further investigation on the correlation between autonomic dysfunctions and possible metabolic disorders; however this supposition was not addressed in the present study [29-30]. On the other hand, our study has shown that physical training can attenuate the cardiac autonomic modulation impairment in both obese and overweight women. In this sense, some studies have suggested that physical training can improve cardiovascular autonomic control and consequently it can influence the energetic balance related to obesity [7-8]. The causes of the autonomic control improvement are controversial, but one study of obese women concluded that 12-week moderate physical training increased significantly the total, LF, and HF variances, with such changes being related to a decrease in adiposity [8]. Nevertheless, in our study no reduction in adiposity was observed. Also, other authors showed that aerobic training improved the insulin sensibility in obese and overweight individuals regardless of changes in body weight, body fat percentage, or concentration of inflammatory markers [31].





**Figure 2:** Examples of representative autoregressive spectra calculate from series of R-R interval ( $\text{ms}^2$ ) in sedentary (top) and trained (bottom) volunteer's participants. Normal Weight (NW group); Overweight (OW group); and Obese (OB group).

Other investigations have indicated that the increase in the cardiac autonomic modulation in obese patients might be a result from augmented baroreflex sensibility [8,32]. This increase may be related to adaptations in the cardiovascular control sites such as hypothalamus, nucleus tractus solitarius (NTS), and rostral-ventrolateral medulla (RVLM) and these adaptations seem to occur by means of both neural remodeling and endogens factors such as nitric oxide [33-36]. In this sense, these central adaptations would improve the regulation of sympathetic and parasympathetic autonomic influences on the heart; however we cannot exclude the fact that physical exercise can also promote intrinsic cardiac adaptations, thus contributing to a better autonomic influence on HRV [35-37].

In conclusion, our results suggest that the regular physical activity has beneficial effect on the cardiac autonomic system function-which is a relevant predictor of cardiovascular morbidity and mortality-by attenuating the negative effect of obesity.

## Acknowledgments

This study was supported by FAPESP (2008/07561-0)

## Disclosure

The authors declared no conflict of interest.

## References

1. Davy KP, Orr JS (2009) Sympathetic nervous system behavior in human obesity. *Neurosci Biobehav Rev* 33: 116-124.
2. Mølgaard H, Sørensen KE, Bjerregaard P (1991) Attenuated 24-h heart rate variability in apparently healthy subjects, subsequently suffering sudden cardiac death. *Clin Auton Res* 1: 233-237.
3. Magnus KA, Matroos A, Strackee J (1979) Walking, cycling, or gardening with or without seasonal interruption, in relation to acute coronary events. *Am J Epidemiol* 110: 724-733.
4. Tsuji H, Venditti FJ Jr, Manders ES, Evans JC, Larson MG, et al. (1994) Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. *Circulation* 90: 878-883.
5. Bray GA (1991) Barriers to the treatment of obesity. *Ann Intern Med* 115: 152-153.
6. Vaz M, Jennings G, Turner A, Cox H, Lambert G, et al. (1997) Regional sympathetic nervous activity and oxygen consumption in obese normotensive human subjects. *Circulation* 96: 3423-3429.

7. Ravussin E, Tataranni PA (1996) The role of altered sympathetic nervous system activity in the pathogenesis of obesity. *Proc Nutr Soc* 55: 793-802.
8. Mark AL (2008) Dietary therapy for obesity: an emperor with no clothes. *Hypertension* 51: 1426-1434.
9. McInnis KJ (2000) Exercise and obesity. *Coron Artery Dis* 11: 111-116.
10. Macor F, Fagard R, Amery A (1996) Power spectral analysis of RR interval and blood pressure short-term variability at rest and during dynamic exercise: comparison between cyclists and controls. *Int J Sports Med* 17: 175-181.
11. Miller WC, Kocaja DM, Hamilton EJ (1997) A meta-analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. *Int J Obes Relat Metab Disord* 21: 941-947.
12. Gielen S, Schuler G, Hambrecht R (2001) Exercise training in coronary artery disease and coronary vasomotion. *Circulation* 103: E1-6.
13. Amano M, Kanda T, Ue H, Moritani T (2001) Exercise training and autonomic nervous system activity in obese individuals. *Med Sci Sports Exerc* 33: 1287-1291.
14. Shibao C, Gamboa A, Diedrich A, Ertl AC, Chen KY, et al. (2007) Autonomic contribution to blood pressure and metabolism in obesity. *Hypertension* 49: 27-33.
15. Zahorska-Markiewicz B, Mizia-Stec K, Jastrzebska-Maj E, Mandecki T, Bilewicz-Wyrozumska T, et al. (2003) Tilt table testing in obesity. *Int J Cardiol* 88: 43-48.
16. Cozza IC, Di Sacco TH, Mazon JH, Salgado MC, Dutra SG, et al. (2012) Physical exercise improves cardiac autonomic modulation in hypertensive patients independently of angiotensin-converting enzyme inhibitor treatment. *Hypertens Res* 35: 82-87.
17. Grassi G, Seravalle G, Dell'Oro R, Turri C, Bolla GB, et al. (2000) Adrenergic and reflex abnormalities in obesity-related hypertension. *Hypertension* 36: 538-542.
18. Arone LJ, Mackintosh R, Rosenbaum M, Leibel RL, Hirsch J (1995) Autonomic nervous system activity in weight gain and weight loss. *Am J Physiol* 269: R222-225.
19. Rumantir MS, Vaz M, Jennings GL, Collier G, Kaye DM, et al. (1999) Neural mechanisms in human obesity-related hypertension. *J Hypertens* 17: 1125-1133.
20. Malliani A, Pagani M, Lombardi F, Cerutti S (1991) Cardiovascular neural regulation explored in the frequency domain. *Circulation* 84: 482-492.
21. Rubini R, Porta A, Baselli G, Cerutti S, Paro M (1993) Power spectrum analysis of cardiovascular variability monitored by telemetry in conscious unrestrained rats. *J Auton Nerv Syst* 45: 181-190.
22. [No authors listed] (1996) Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation* 93: 1043-1065.
23. Dutra SG, Pereira AP, Tezini GC, Mazon JH, Martins-Pinge MC, et al. (2013) Cardiac autonomic modulation is determined by gender and is independent of aerobic physical capacity in healthy subjects. *PLoS One* 3: e77092.
24. Piccirillo G, Vetta F, Viola E, Santagada E, Ronzoni S, et al. (1998) Heart rate and blood pressure variability in obese normotensive subjects. *Int J Obes Relat Metab Disord* 22: 741-750.
25. Piccirillo G, Elvira S, Bucca C, Viola E, Cacciafesta M, et al. (1997) Abnormal passive head-up tilt test in subjects with symptoms of anxiety power spectral analysis study of heart rate and blood pressure. *Int J Cardiol* 60: 121-131.
26. Ramaekers D, Ector H, Aubert AE, Rubens A, Van de Werf F (1998) Heart rate variability and heart rate in healthy volunteers. Is the female autonomic nervous system cardioprotective? *Eur Heart J* 19: 1334-1341.
27. Fraley MA, Birchem JA, Senkottaiyan N, Alpert MA (2005) Obesity and the electrocardiogram. *Obes Rev* 6: 275-281.
28. Alvarez GE, Davy BM, Ballard TP, Beske SD, Davy KP (2005) Weight loss increases cardiovagal baroreflex function in obese young and older men. *Am J Physiol Endocrinol Metab* 289: E665-669.
29. Scherrer U, Sartori C (1997) Insulin as a vascular and sympathoexcitatory hormone: implications for blood pressure regulation, insulin sensitivity, and cardiovascular morbidity. *Circulation* 96: 4104-4113.
30. Bobbioni-Harsch E, Sztajzel J, Barthassat V, Makoundou V, Gastaldi G, et al. (2009) Independent evolution of heart autonomic function and insulin sensitivity during weight loss. *Obesity (Silver Spring)* 17: 247-253.
31. Nassis GP, Papantakou K, Skenderi K, Triandafilopoulou M, Kavouras SA, et al. (2005) Aerobic exercise training improves insulin sensitivity without changes in body weight, body fat, adiponectin, and inflammatory markers in overweight and obese girls. *Metabolism* 54: 1472-1479.
32. Gulli G, Cevese A, Cappelletto P, Gasparini G, Schena F (2003) Moderate aerobic training improves autonomic cardiovascular control in older women. *Clin Auton Res* 13: 196-202.
33. Zheng H, Li Y, Cornish KG, Zucker IH, Patel KP (2005) Exercise training improves endogenous nitric oxide mechanisms within the paraventricular nucleus in rats with heart failure. *Am. J. Physiol* 288: 2332-2341.
34. de Abreu SB, Lenhard A, Mehanna A, de Souza HC, Correa FM, et al. (2009) Role of paraventricular nucleus in exercise training-induced autonomic modulation in conscious rats. *Auton Neurosci* 148: 28-35.
35. Souza HC, De Araújo JE, Martins-Pinge MC, Cozza IC, Martins-Dias DP (2009) Nitric oxide synthesis blockade reduced the baroreflex sensitivity in trained rats. *Auton Neurosci* 150: 38-44.
36. Sant'Ana JE, Pereira MG, Dias da Silva VJ, Dambrós C, Costa-Neto CM, et al. (2011) Effect of the duration of daily aerobic physical training on cardiac autonomic adaptations. *Auton Neurosci* 159: 32-37.
37. Nottin S, Vinet A, Stecken F, Nguyen LD, Ounissi F, et al. (2002) Central and peripheral cardiovascular adaptations during a maximal cycle exercise in boys and men. *Med Sci Sports Exerc* 34: 456-463.