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Original Article

Heart Rate Variability following Combined Aerobic and Resistance Training in Sedentary Hypertensive Women: A Randomised Control Trial

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ABSTRACT

Objective: To investigate the effect of combined aerobic and resistance training (CART) on heart rate variability in sedentary, hypertensive women.
Participants: A total of twenty-eight hypertensive (Stage 1 and 2) sedentary women (Age 40.54 ± 4.2 yrs; Height 155.14 ± 5.4 cm; Weight 70.96 ± 10.2 kg; BMI 29.6 ± 4.4; Duration of HTN: 6.43 ± 2.5 yrs) were recruited for the study.
Methods: Participants were randomly assigned to either the CART group that performed combined aerobic and resistance exercise of moderate intensity [aerobic exercise 50–80% of HR_{max} (maximum heart rate) and resistance exercises at 50–80% of 1 Repetition Maximum (RM)], 5 times/week for 4 weeks, or to the control group that followed conventional treatment without any supervised exercise intervention.
Main outcome measures: Resting blood pressure was measured and standard heart rate variability (HRV) indices were calculated using time domain (SDNN, pNN50, RMSSD) and frequency domain (LFnu, HFnu, LF/HF and TP) analysis.
Results: CART group demonstrated an increase in HFnu, TP, SDNN, and RMSSD, ($p < 0.05$) along with a significant decrease in LFnu, LF/HF ratio, systolic blood pressure, and diastolic blood pressure ($p < 0.05$).
Conclusion: CART showed significant improvement in HRV parameters indicating vagal dominance in middle-aged hypertensive women. Therefore, exercise training in combined form (aerobic and resistance) may be incorporated in the management programs of the patients suffering from hypertension in order to augment improvement in their cardiac autonomic control.

1. Introduction

More women's lives are claimed by cardiovascular diseases (CVDs) than any of the other diseases,[1] resulting in about one death per minute among women in the US.[2,3] Hypertension has been found to be one of the most important risk factors for CVD in women. Hypertension is a complex multifactorial disease that is characterized by a gradual increase in systolic blood pressure (SBP) by 140 mmHg and diastolic blood pressure (DBP) by 90 mmHg.[4] It has been observed that attainment of clinical blood pressure goals can markedly reduce cardiovascular morbidity and mortality.[5] Yet, treatments often seem to fail to realize those goals as approximately two-thirds of treated hypertensive women still have uncontrolled blood pressure.[2,3]

Autonomic imbalance is characterized by sympathetic branch of autonomic nervous system (ANS) dominating over parasympathetic branch.[6] It has been observed that women with hypertension usually have normal cardiac output and raised peripheral resistance causing adrenergic modulations and dysregulation of parasympathetic and tonic cardiovascular control.[7] HRV is used to evaluate autonomic imbalances, diseases, and mortality.[8] Increased efferent vagal activity is specified by increased variability of heart rate (HR),

Abbreviations: ANS, Autonomic Nervous System; HRV, Heart Rate Variability; CART, Combined Aerobic and Resistance Training; HR_{max}, Maximum Heart Rate; RM, 1 Repetition Maximum; BP, Blood pressure; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; SDNN, Standard deviation of the RR intervals; RMSSD, Square root of the mean squared differences between adjacent RR intervals; pNN50, Percentage of interval differences of adjacent RR intervals greater than 50 ms derived from differences between consecutive RR intervals; TP, Total Power; LFnu, Low Frequency normalize units; HFnu, High Frequency normalize units; LF/HF, Ratio of low and high frequency; SA node, Sino-atrial node; NN interval, Normal to Normal Interval; FFT, Fast Fourier Transformation; T2DM, Type 2 Diabetes Mellitus; HR, Heart Rate; BMI, Body Mass Index; ET, Endurance Training; ECG, Electro Cardiogram; SD, Standard Deviation.

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whereas sympathetic stimulation decreases HRV. Increased risk of cardiac events and death is associated with decreased HRV in healthy individuals.[9] Moreover, it is an indicator of abnormal autonomic control of the heart and is also related with increased risk for spontaneous ventricular arrhythmias and progression of coronary atherosclerosis and deaths caused by cardiac failure.[10] HRV is reduced in hypertensive population and reduced HRV is one of the most powerful predictor of death.[11,12]

Regular physical activity, such as endurance and strength training have been shown to increase HRV presumably by increasing the vagal relative to the sympathetic influence on the heart.[13,14] Therefore, exercise may exert cardio protective effects and thus benefit the cardiovascular risk profile.[15,16] According to previous literature, exercise prescription and training-induced long-term physiological benefits depend on outcomes of cardiac autonomic activity.[17] A study reported improvement in cardiac autonomic function, following aerobic exercise program in obese hypertensive women.[16] Cozza et al.[15] also showed that aerobic exercise training improves HRV in hypertensive patients. Resistance exercise is also an important part of comprehensive wellness program but its effect with respect to HRV is still controversial.[18] Recommendations by American College of Sports Medicine (ACSM) prescribe both aerobic and resistance exercise in the regime of hypertensive patients, and combined exercise has shown more improvements in blood pressure (BP) than individual exercise forms.[19,20] Although the role of combined aerobic and resistance training (CART) in enhancing cardiac health is well established in scientific literature but its effect on cardiac autonomic control which is an important component of cardiac health and an important link in the patho-physiology of hypertension is not known. Therefore, the present study aimed to investigate the effect of CART on HRV in middle-aged, sedentary hypertensive women.

2. Materials and methods

2.1. Study design

This was a single-blinded study with two-arm comparative pretest-posttest design, with random allocation of subjects into experimental and control groups. Block randomization with variable block size was done in 1:1 ratio, using a list of random numbers generated by Ralloc software. The outcome assessor remained blinded to participant allocations and the interventions that each group received.

2.2. Participants

A total of twenty-eight hypertensive women participated in the study (Fig. 1). A pilot study on 10 subjects was conducted to calculate the sample size. Sample size was calculated by Cohen's Fischer's '*F*'distribution,[21] using between group changes in HF power of HRV of the 10 participants, with an effect size of 0.59, partial eta square of 0.26, alpha level of 0.05, and power of 0.90. Based on these effect estimates, a total sample size of 22 (11 in each group) subjects was found to be necessary. Considering drop-outs, we enrolled 28 participants (14 in each group).

As exercise performance is shown to be affected by age, to limit the age range in the study premenopausal sedentary women aged 30–50 years and diagnosed with either Stage 1 or Stage 2 hypertension were included. Premenopausal status was evaluated by taking a history of regular menstrual cycles. Participants were defined as sedentary if they were either not engaged in any leisure time physical activity or there was only one instance of low intensity ($<50\%$ HR_{max}) exercise or sports activity for less than 20 min in a week.[22,23] To avoid any complications, women with a history of recent cardiac infarction, unstable angina, valvular illness, pulmonary disease, heart failure, kidney failure, uncontrolled hypertension, and diabetes, were excluded from the study. Patients who used more than one antihypertensive drug and those involved in exercise training for more than 6 months were also excluded.[24]

Subjects were recruited through referrals from the university health center and nearby hospitals. They were assessed in the Human Performance Lab and trained in the gymnasium at the university clinic. The study was approved by the Institutional Ethical Committee, Jamia Millia Islamia, New Delhi, India, and a written informed consent was obtained from all participants. Ethical standards were maintained during the study according to the Declaration of Helsinki, 1964.

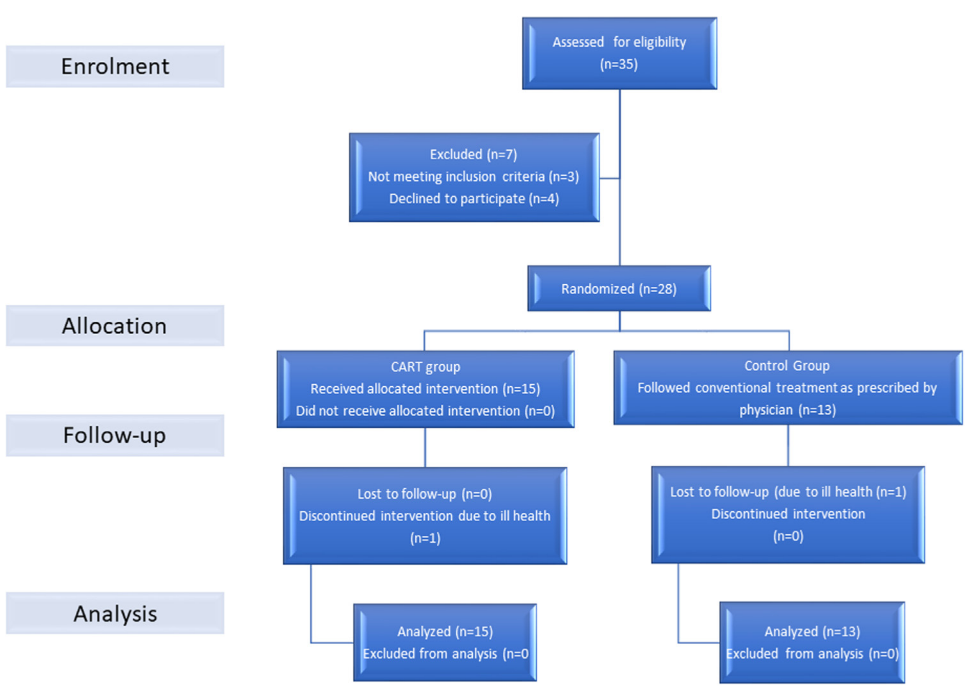

Fig. 1. CONSORT flow chart describing participant flow during the study.

2.3. Heart rate variability assessment

Twenty minutes of Electrocardiographic (ECG) activity was recorded, which was later analyzed for HRV. Participants were advised to avoid any caffeine containing substances (including tea, coffee, and cola drinks) for 12 h, and alcoholic beverages or smoking for at least 24 h prior to the assessment.[25] Participants were instructed to abstain from any kind of additional physical activity (beyond normal lifestyle activities) 48 h prior to their HRV assessment. During recording, participants were instructed to close their eyes and avoid any bodily movement or conversation. To ensure uniformity and minimize any confounding effects of fluctuating levels of female hormones, the ECG was recorded in the mid-follicular phase of the menstrual cycle for all the subjects. In supine position, ECG was recorded for 20 min after 5 min of rest at 24 °C. Heart rate of the participants was recorded by polar heart rate monitor during the rest period (Model T 34, Polar Electro Oy, and Finland). Time and frequency domain variables of HRV were analyzed by detection of R waves. AD instruments lab chart version 7.3.7 with HRV module version 1.4.2 using weltch window (Power Lab 8 SP, AD Instruments, and Australia) was used as data acquisition software for recording ECG, which calculated R-R intervals as the measure of difference between successive beats. Initially all R-R intervals were edited by visual inspection mainly to exclude all the undesirable beats/ectopic beats which counted for $<10\%$ in every subject. ECG signal was then amplified and digitized. Analysis was done on a time series of five consecutive minutes which were selected from the 20 min recordings. All calculated indices of HRV provided a good estimation of autonomic function. Time domain variables such as square root of the mean squared differences between adjacent R-R intervals (RMSSD) and number of interval differences of adjacent R-R intervals greater than 50 ms derived from differences between consecutive R-R intervals (pNN50) indicates the parasympathetic activity, whereas standard deviation of N-N intervals (SDNN) reflects overall variability in heart rate. Frequency domain indices include total power which indicates variations between the N-N intervals, low frequency power (LF: 0.04–0.15 Hz) which depicts combination of sympathetic and parasympathetic activity, high frequency power (HF: 0.15–0.40 Hz) which denotes vagal activity, and LF/HF ratio which represents sympathovagal balance. The combination of all these variables of HRV provides information on the amount of relative contributions from sympathetic and parasympathetic tonus to heart.[26]

2.4. Blood pressure measurement

SBP and DBP were measured by standard manual sphygmomanometer. Participants were strictly prohibited from consuming any caffeinated products, smoking, and exercising, 30 min prior to the BP measurement. Throughout assessment, patients remained in a relaxed seated state for 5 min. The cuff was placed on the left arm for all patients and BP was recorded according to standard guidelines. A second recording was also taken after 2 min on the same arm. If the measurements had a difference of $\geq$5 mmHg of BP, further readings were obtained until there were 2 consecutive stable measurements. Final reading recorded was considered as an average of the 2 stable measurements obtained.[27]

2.5. Training protocol

Measurements of HRV and BP were made before the beginning of training period. Following baseline assessment, subjects were divided into either the CART group or the control group. Participants in the CART group received combined aerobic and resistance exercise training, while those in the control group did not receive any supervised exercise intervention and continued to follow their usual medical routine and dietary recommendations as prescribed by their treating physician. Before the commencement of actual training, subjects were given 2 familiarization sessions. The HR_{max} of subjects was calculated using the equations specific for women, i.e., $206 - (0.88 \times \text{age})$, while 1 Repetition Maximum was calculated by Brzycki equation.[28,29]

CART group trained on an inclined (5.0%) treadmill 3 days/week for 4 weeks. In addition, resistance training was provided twice a week, resulting in a total of 5 training days/week. Each session commenced with a 5-min warm-up performed at 40% of HR_{max} on treadmill, followed by either an aerobic or resistance exercise bout. Aerobic training was performed at 50–80% of HR_{max} (intensity was progressed gradually from 50% to 80% HR_{max} across 4 weeks) for 20 min, followed by 5 min of cool down at 40% of HR_{max}. Resistance training constituted 3 sets of 10 repetitions of 5 exercises: bicep curls, triceps extensions, abdominal crunches, leg curls, and knee extensions, at an intensity of 50–80% of 1RM (intensity was progressed gradually from 50% to 80% of 1 RM over 4 weeks).[30,31] HR, SBP, and DBP were closely monitored during and after exercise. Exercise training was performed 5 times/week for 4 weeks.

3. Statistical analysis

Data was analyzed using SPSS version 17.0. All variables were assessed by Shapiro-Wilk test for normality of distribution and those found to be non-normal (SBP, DBP, HFnu, pNN50) were log-transformed for further analysis. Independent *t*-test was used to compare the demographic characteristics as well as baseline criterion measures between both the groups. Analysis of Covariance (ANCOVA) was used to analyze the variables that were significantly different between the two groups at baseline (SBP and HFnu). 2×2 mixed model Analysis of variance (ANOVA) was employed to examine the difference between the groups (CART vs. Control) across 2-time points (pre- and post-training) as well as the interaction effect (Group X Time). Paired *t*-test was employed to investigate the within group change in outcome measures. Significance level was set at 0.05.

4. Results

Twenty-eight participants were assessed at baseline, of which, 2 dropped out (1 in CART and 1 in control group) due to health issues which were handled by intention to treat analysis (Fig. 1). The results of independent *t*-test comparing the demographic characteristics and outcome measures between the two groups at baseline, showed significant differences in height, SBP and HFnu (Table 1). The descriptives suggested that while the CART group recorded positive changes, the control group showed little difference (BMI and blood pressure) or deterioration (resting HR and measures of HRV) from the baseline values following 4 weeks (Table 2).

4.1. Heart rate and blood pressure

A 2×2 mixed model ANOVA yielded a significant interaction effect for weight, BMI, resting HR and DBP, denoting that the CART grouped showed greater improvements relative to the control group. SBP which was found to be significantly different between the two groups at baseline, was analyzed using ANCOVA using the pre-training value as a covariate and revealed similar findings emphasizing the main effect for both group and interaction ($p < 0.001$) (Table 2; Fig. 2).

Table 1
Comparison of demographic characteristics and outcome measures at baseline between the groups.

Variable	CART Mean (SD) n = 15	Control Mean(SD) n = 13	*P*
Age (years)	39.67(4.1)	41.54(4.25)	0.24
Height (cm)	157.2(5.29)	152.6(4.44)	0.02*
Weight (kg)	74.2(10.544)	67.2(8.729)	0.07
BMI(kg/m^2)	30.1(4.79)	30.9(3.94)	0.49
Duration (years)	6.33 (1.67)	6.54 (3.36)	0.84
SBP (mmHg)	141.6(4.63)	149.4(11.57)	0.03*
DBP (mmHg)	84.6(6.54)	85.0(3.75)	0.81
HR(bpm)	75.5(9.03)	80.3(9.42)	0.18
SDNN (ms)	33.0(9.42)	35.6(14.25)	0.22
RMSSD (ms)	27.6(24.20)	28.1(19.22)	0.82
pNN50 (%)	8.5(16.91)	14.2(15.89)	0.41
Total Power (ms^2)	1126.2(1.43)	1460.0(1.30)	0.44
LF(nu)	48.3(16.15)	36.3(19.25)	0.07
HF(nu)	48.84(13.16)	64.59(20.24)	0.02*
LF/HF ratio	1.15(0.69)	0.75(0.71)	0.48

BMI: Body mass index;SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HR: Heart rate; SDNN: Standard deviation of N-N intervals; RMSSD: Square root of the mean squared differences between adjacent RR intervals; pNN50: Number of interval differences of adjacent RR intervals greater than 50 ms derived from differences between consecutive RR intervals; LFnu: low frequency normalized units; HFnu: high frequency normalize units; LF/HF ratio: low frequency/high frequency ratio; TP: total power,*significant difference, Independent *t* test was applied.

Table 2
Comparison of CART and Control group on the outcome measures after 4 weeks.

Dependent		CART Mean(SD)	Control Mean(SD)	Sources	*P*
Weight(kg)	Pre	74.2(10.54)	67.2(8.72)	Group(G)	0.10
	Post	72.8(10.82)	67.2(8.50)	Time(T)	0.001*
				Interaction(G × T)	0.001*
BMI(kg/m^2)	Pre	30.1(4.79)	28.9(3.94)	Group(G)	0.62
	Post	29.4(4.82)	28.9(3.83)	Time(T)	<0.001*
				Interaction(G × T)	<0.001*
†SBP(mmHg)	Pre	141.6(4.63)	149.4(11.57)	Group(G)	<0.001*
	Post	122.5(9.16)	149.0(10.53)	Time(T)	0.59
				Interaction(G × T)	<0.001*
DBP(mmHg)	Pre	84.6(6.54)	85.0(3.75)	Group(G)	0.007 *
HR(bpm)	Post	77.4(5.74)	86.3(6.35)	Time(T)	0.03*
	Pre	75.5(9.03)	80.3(9.42)	Interaction(G × T)	0.006*
	Post	71.2(8.90)	83.2(7.79)	Group(G)	0.01*
				Time(T)	0.02*
				Interaction(G × T)	<0.001*
SDNN(ms)	Pre	34.5(12.78)	35.6(14.25)	Group(G)	0.01*
	Post	54.5(12.16)	28.6(13.64)	Time(T)	0.02*
				Interaction(G × T)	<0.001*
RMSSD	Pre	27.6(24.20)	28.1(19.22)	Group(G)	0.26
	Post	37.9(17.85)	21.4(15.57)	Time(T)	0.41
				Interaction(G × T)	0.001*
pNN50	Pre	8.5(16.91)	7.8(12.21)	Group(G)	0.67
	Post	11.3(16.38)	10.9(15.83)	Time(T)	0.73
				Interaction(G × T)	0.97
LFnu	Pre	48.3(16.15)	36.3(19.25)	Group(G)	0.67
	Post	27.3(11.46)	44.3(20.01)	Time(T)	0.007*
				Interaction(G × T)	<0.001*
†HFnu	Pre	48.8(13.16)	64.5(20.24)	Group(G)	<0.001*
	Post	81.4(16.04.)	57.1(19.46)	Time(T)	<0.001*
				Interaction(G × T)	<0.001*
LF/HF Ratio	Pre	1.15(0.69)	0.75(0.71)	Group(G)	0.60
	Post	0.36(0.22)	0.99(0.78)	Time(T)	0.005*
				Interaction(G × T)	<0.001*
Total Power(ms^2)	Pre	1126.2(1.43)	1460.0(1.30)	Group(G)	0.68
	Post	1777.6(1.87)	1101.2(1.10)	Time(T)	0.19
				Interaction(G × T)	<0.001*

CART: combined aerobic and resistance training;SBP: Systolic blood pressure; DBP: Diastolic blood pressure;HR: Heart rate;SDNN: Standard deviation of N-N intervals; RMSSD: Square root of the mean squared differences between adjacent RR intervals; NN50: Number of interval difference of adjacent RR intervals greater than 50 ms derived from differences between consecutive RR intervals; pNN50: Percentage of interval differences of adjacent RR intervals greater than 50 msderived from differences between consecutive RR intervals; LF nu: Low frequency power normalize units; HF nu: High frequency power normalize units LF/HF ratio: Ratio of low and high frequency power; *significant difference, $p < 0.05$; Variables were analyzed using 2 × 2 mixed model ANOVA (analysis of variance) and ANCOVA (analysis of covariance)†.

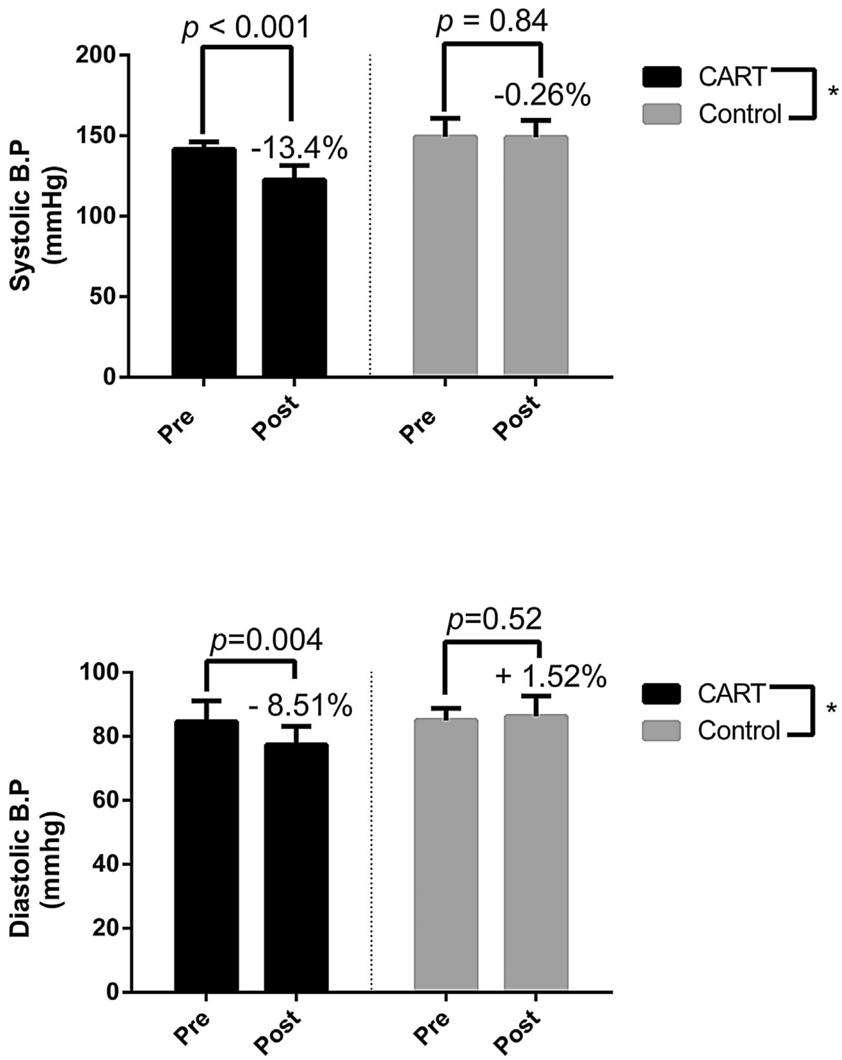

Fig. 2. Mean ± SD with percentage change in systolic and diastolic BP (blood pressure) before and after four weeks of combined aerobic and resistance training (CART) versus the control group, *significant difference.

4.2. Heart rate variability

4.2.1. Time domain measures

SDNN demonstrated a significant main effect for both time and group*time interaction implying that the CART group showed improvements with respect to values at baseline as well as the control group. Similar findings were obtained for RMSSD (time*-group interaction: $p = 0.001$), while pNN50 failed to demonstrate any significant changes (Table 2; Fig. 3).

4.2.2. Frequency domain measures

The results for variables signifying the frequency domain of HRV were, in agreement to those of time domain measures, with a significant interaction effect for LFnu, LF/HF ratio and total power ($p < 0.001$). HFnu which was analyzed using ANCOVA also revealed a significant difference between the CART and control groups, in favour of the CART group (Table 2; Fig. 3).

5. Discussion

The purpose of this study was to investigate the combined effect of aerobic and resistance training on cardiac autonomic control of middle-aged, sedentary, hypertensive women. The main findings suggested significant improvements in HRV and resting BP following 4 weeks of CART. In addition, there was a decrease in resting heart rate and BMI, indicating an enhanced overall fitness profile.

5.1. Effect of CART on resting heart rate and BMI

Heart rate (HR) is a familiar and accessible clinical variable. Based on epidemiologic data and inferences from clinical trials designed for other purposes, most physicians believe that tachycardia at rest is prognostically undesirable due to its association with cardiovascular disease risk and mortality.[32] It is well documented that when adjusted for age, HR in women averages 2–7 beats/min higher than in men, which might pose an increased mortality risk in women.[33,34] In the present study, in addition to BP, there was also a concomitant reduction in HR after exercise training ($p < 0.05$). Possible mechanisms which might have led to thus change in resting HR includes (1) increased parasympathetic tone[35,36]; (2) decreased responsiveness to beta-adrenergic stimulation[37,38]; (3) decreased intrinsic heart rate.[39,40] Previously, it has been proven that exercise training leads to reduction in intrinsic heart rate and thus may reduce CVD burden and mortality.[41]

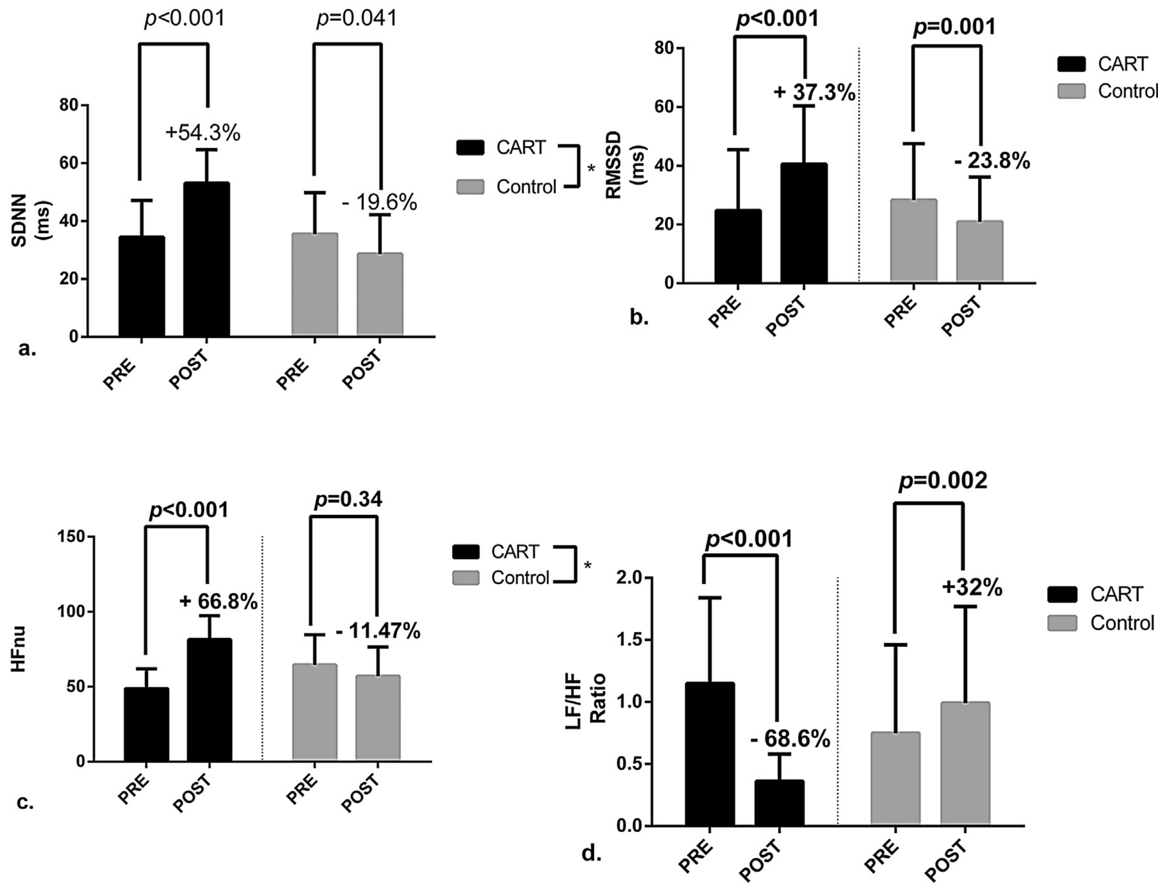

Fig. 3. Mean ± SD with percentage change in SDNN (standard deviation of N-N intervals), RMSSD (square root of the mean squared differences between adjacent RR intervals), HF (high frequency power), LF/HF (ratio of low and high frequency power) for hypertensive females before and after four weeks of combined aerobic and resistance training (CART) versus the control group, *significant difference.

BMI is an easy to calculate, simple tool to measure body composition, and a well-known marker of health-related physical fitness. Association of BMI with cardio-metabolic and global risk of diseases is well established by previous research.[42] Therefore, a reduction in BMI as demonstrated by the present study might be a positive adaptation in at-risk population such as hypertensive, sedentary females.

5.2. Effect of CART on blood pressure

The management of high BP is considered a primary objective in prevention of CVD. The implementation of exercises such as aerobic exercise results in increased oxygen consumption with respect to the intensity of the imposed activity while, resistance training acts as wellness program for the development of muscular strength, endurance and mass.[23] In the current study, CART group showed significant improvement in SBP (13.4% decrease, $p < 0.001$)and DBP (8.51% decrease, $p = 0.004$).These findings are consistent with those of previous researches which showed that 16-weeks of combine aerobic and resistance training is safe and effective in reducing BP in elderly hypertensive women.[20] Collier et al.[43] demonstrated that 4-weeks of aerobic training is effective in reducing systolic and diastolic BP. Results of present study reinforces the fact that CART can produce substantial beneficial effects in reducing BP. The possible reason for reduction in BP and HR might be reduced vasomotor tone and increase in vagal tone post-exercise training.[44]

5.3. Effect of CART on cardiac autonomic control

HRV is a non-invasive reproducible measure corresponding to the balance between sympathetic and parasympathetic components of ANS function on sinoatrial node. The spectral measures of HRV for ECG sampling (20 min) are analyzed by fast Fourier transformation.[26] The main components of Fast Fourier indices are total power, LFnu and HFnu.[45] Total power is an estimate of global activity of ANS. HFnu (0.15 Hz–0.4 Hz) is the marker of cardiac autonomic parasympathetic nervous system activity, LFnu (0.04–0.15 Hz) reflects the combination of sympathetic and parasympathetic output and the ratio of two i.e. LF/HF ratio is considered as a marker of sympathovagal balance.[26,46] In the present study, there was significant improvement in Total power (22.3% increase, $p = 0.01$), HFnu (66.80% increase, $p < 0.001$) and LF/HF ratio (68.9% decrease, $p < 0.001$) in CART group which indicates vagal modulation. Although, there is lack of evidence for the effect of CART on cardiac autonomic function in hypertensive, there are studies that have explored the role of structured combined exercise training on HRV in other lifestyle diseases like type 2 diabetes mellitus (T2DM). Findings of our investigation are in accordance with previous literature where 12-weeks of combined training program in T2DM significantly increased HFnu and decreased LF/HF ratio.[47] Another study showed that aerobic exercise training changes resting autonomic balance by decreasing LF/HF ratio and increasing vagal modulation after 4 weeks in pre-hypertensive or stage 1 hypertensive individuals.[43] Possible speculations for improved cardiac autonomic function in training group are presence of mediators such as nitric oxide which have positive effect in increasing cardiac vagal tone after exercise and angiotensin II which is known as an inhibitor of cardiac vagal activity. However, these are mere speculations since the present study did not investigated these markers but previously researches have established that exercise training improves nitric oxide bioavailability and decreases angiotensin II levels.[48] No change and slight deterioration was observed in HFnu (11.47% decrease, $p = 0.34$) and LFnu (22.03% increase, $p = 0.03$) respectively in the control group probably due to the natural course of disease.

Time domain measurements plot HRV as the variations in normal R wave to R wave (N-N) intervals over time.[49] The most widely used measurements among time domain variables are SDNN that represents overall HRV, RMSSD and pNN50 computed over the entire recording represents pure vagal influence on HRV.[26] Present study documented significant improvement after exercise training in time domain variables, SDNN (54.3% increase, $p < 0.001$), and RMSSD (37.3% increase, $p = 0.001$) but non-significant changes ($p = 0.76$) were observed in pNN50. The reason behind discrepant results observed in time domain variables might be the short duration of ECG record used in this study since ideal method to trace changes in time domain variables of HRV has been suggested to be a 24 h ECG recording.[26] The improvement in majority of variables indicating vagal activity (SDNN and RMSSD) is in accordance with previous literature which reported an increase in these variables after 12-weeks of CART in diabetic population.[47] Another study concluded that aerobic exercise training in 144 obese hypertensive women and men significantly improve HRV.[16] The possible explanation for the improvement in time domain HRV variables in CART group may be adaptation in ANS in favour of parasympathetic dominance and decreased SNS activity after training sessions.[50] Deterioration was observed in the autonomic function of control group participants with a 23.8% and 19.6% decrease in RMSSD and SDNN, respectively.

5.4. Strengths and limitations

In the present study, all testing procedures were performed using standardized protocols as per guidelines. Moreover, the training sessions were supervised and monitored carefully. Assessment of HRV for all the participants was taken only after controlling all the extrinsic confounders affecting ANS function. The limitations of the present study are the short duration of training and use of only one measure of autonomic control. Longer duration of training with use of other autonomic function parameters such as baroreflex sensitivity and heart rate recovery might have elicited more precise results. Furthermore, we included a very small number of participants in this study which is an important limitation and in future researches this question should be addressed on a larger sample for extrapolation of findings to the population. It would also be of interest to compare these findings with the response of post-menopausal women.

6. Conclusion

Our results demonstrated that CART might improve HRV in hypertensive women indicating increased vagal cardiac activity. Altered cardiac autonomic control is observed in hypertensive women that further increases the risk of CVD and CART might act as an effective exercise protocol in its management. Results of the present study will help clinicians in designing exercise training protocols for female patients suffering from hypertension. However, there is need for more studies to investigate the effect of CART on cardiac autonomic function in hypertensive women (especially post-menopausal) on a larger sample to validate the current findings.

Conflict of interest

The authors report no conflict of interest.

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What was already known

It is known that physical exercise modulates cardiac autonomic control in patients with hypertension, however, literature is insufficient on specific form of physical exercise that should be followed. Recommendations by international bodies supports the incorporation of combined aerobic and resistance training (CART) but whether this form of combined exercise leads to any meaningful change in HRV of hypertensive women is still questionable.

What this study adds

Findings of the present study demonstrated that physical exercise in combined form (CART) lead to positive adaptations in the cardiac autonomic control of sedentary hypertensive women. Significant changes were observed, which means that 4 weeks of CART may reduce cardiovascular disease risk in sedentary young women to a certain extent by improving their HRV.

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