

Yogesh Saxena¹, Rani Gupta¹, Arsalan Moinuddin², Ravinder Narwal³

¹Department of Physiology, HIMS, SRH University, Dehradun, Uttarakhand, ²Department of Physiology, NIMS University, Jaipur, Rajasthan, ³Department of PMR, BPS Government Medical College for Women, Sonipat, Haryana, India

Abstract

Context: Accumulated moderate physical activity (PA) for 30 min in a day is the only recommended treatment of prehypertension. **Objective:** We investigated autonomic modulation as a possible mechanism for the decrease in blood pressure (BP) during the rest periods in each 10 min session of PA. **Design, Setting, and Participants:** We conducted a single-blind randomized multi-arm control trial on 40 prehypertensive (pre-HT) young male adults. **Methods:** Participants were randomly divided by using random number table into four groups. Control (no intervention); Group 1 (walking at 50% of predicted VO₂ peak); Group 2 (walking at 60% of predicted VO₂ peak); Group 3 (walking at 70% of predicted VO₂ peak). BP, heart rate variability (HRV), and heart rate recovery 1 min (HRR 1 min) were measured at baseline and during the rest period after each session of 10 min over 30 min of accumulated physical activity (PAcumm). **Results:** Significant diastolic BP (DBP) reduction (*P* < 0.001) was observed during the rest period after each session of PAcumm in intervention groups. An average reduction in DBP was more in pre-HT undertaking PAcumm at 70% of predicted VO₂ Peak. Decrease in the mean value of low-frequency (LF) and LF/high-frequency ratio was observed following PAcumm in all intervention groups irrespective of the intensity of PA. No significant association of reduction of BP with HRV and HRR 1 s was observed. **Conclusion:** Reduction in BP was observed during the rest period after each 10 min session of PAcumm irrespective of the intensity of PA. No significant each 10 min session of PAcumm irrespective of the intensity of PA. No significant association of reduction in BP during the sessions.

Keywords: Accumulated physical activity, blood pressure, prehypertensive

Introduction

Primary prevention in hypertensive particularly in prehypertensive (pre-HT) must be promoted in population,^[1] as it is associated with an increase in cardiovascular morbidity and mortality.^[2] Lifestyle modifications, including regular physical activity (PA), are the only recommended treatment for prehypertension.^[3]

Lifestyle PA has been defined as a daily accumulation of at least 30 min of self-selected activity which may include occupational, at leisure, or household which is of at least moderate intensity.^[4] Although the role of PA in reducing morbidity and mortality

Address for correspondence: Dr. Yogesh Saxena, Associate Professor, Department of Physiology, HIMS, SRH University, Dehradun, Uttarakhand, India. E-mail: drysaxena@rediffmail.com

Access this article online				
Quick Response Code:	Website: www.jfmpc.com			
	DOI: 10.4103/2249-4863.192368			

has been well-established globally,^[1,5] lack of time is a constant barrier to PA.^[6] A recommendation that allows an individual to perform a short bout of activity throughout the day rather than a continuous time slot in a busy schedule is intuitively appealing.

Recent public health guidelines have endorsed the daily use of accumulated intermittent physical activity (PAccum) in short bouts for maintaining good health^[7,8] and for preventing progression of pre-HT to hypertension.^[1]

Reduction of blood pressure (BP) in hypertensive and high normal BP by structured exercise has been seen by several authors.^[9-11] Recently, studies have identified the accumulated use of structured exercise of a single bout as an effective way of improving fitness and altering the adverse health outcome.^[12,13]

For reprints contact: reprints@medknow.com

How to cite this article: Saxena Y, Gupta R, Moinuddin A, Narwal R. Blood pressure reduction following accumulated physical activity in prehypertensive. J Family Med Prim Care 2016;5:349-56.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

Accumulation of intermittent PA on BP reduction was found to be effective^[14,15] but the intensity of the accumulated PA, which shows best health benefit needs to be ascertained.

Review of the available literature has failed to elucidate any definitive mechanism(s) underlying postexercise hypotension (PEH), but it has been mainly attributed to decreased vascular resistance. Proposed mechanisms involved in the alteration of vascular sensitivity are a balance of neural, humoral, and vascular mechanisms.^[16] The neural mechanism may involve the modulation of barosensitive neurons by BP afferents and muscle afferents which may lead to a centrally mediated decrease in sympathetic efferent nerve discharge.^[17] Indirect indices of the autonomic nervous system by heart rate variability (HRV) have been widely utilized for sympathetic efferent nerve activity even in PEH.^[18]

Since the effectiveness of accumulated PA on BP reduction might be the residual effects of the successive several short bouts of PA sessions, autonomic modulation may, therefore, be a cause for BP response between the short sessions during the PAccum. The study was therefore planned to investigate the autonomic modulation as a possible mechanism for the decrease in BP during the rest periods following each 10 min PA session within accumulated physical activity (PAcumm) over a 30 min period in pre-HT adults.

Methods

Study design

The study was experimental randomized within-subject design to examine the effect of levels of PAaccum in pre-HT male volunteers aged 20–40 years and was approved by the Institute Ethical Committee and registered at the central registry of clinical trials no. CTRI/2015/11/006343.

Study population

The study participants were recruited from adult population in and around HIHT campus in Doiwala block, Dehradun because of the homogeneity of the residents regarding their environmental exposures, including lifestyle and nutritional factors and habitual dietary intake.

BP screening was done for the volunteers and those with the BP in the pre-HT range according to WHO/JNC VII classification^[1] (defined as systolic BP [SBP] of 120–139 mmHg and/or diastolic BP [DBP] of 80–89 mmHg) were examined for the exclusion criteria like history of clinical cardiovascular disease, diabetes mellitus, and chronic kidney disease, current use of anti-diabetic medications or insulin, and heavy alcohol consumption. Those found eligible were administered PA questionnaire (IPAQ).^[19] Volunteers with less than or equal to the moderate intensity of work by IPAQ were included as participants. Following written informed consent, these volunteers were called between 9 and 11 am from Monday to Thursday in the Department of Physiology, HIMS, SRH University.

Study protocol

A standard questionnaire was administered to the participants by trained staff at the point of entry to collect information on demographic characteristics, personal and family medical history. Three BP measurements were obtained in morning hours using standard protocol and predicted VO₂ peak was calculated using regression equation by 6 min walk test (MWT)^[20] on the same day.

Randomization

It is a unicentric unbalanced randomization (3:1) single blind controlled multi-arm parallel group study conducted at HIMS SRH University.

Study groups

On the next day (intervention period), unbalanced randomization (3:1) was done with participants grouped into control (no intervention) groups (n = 10) and experimental (PAaccum) group (n = 30).

a. Experimental group (n = 30) were randomly administered either of three different set of physical activities (PAaccum) Group I: 3 sessions of 10 min walk at 50% of predicted VO₂ peak

Group II: 3 sessions of 10 min walk at 60% of predicted VO₂ peak

Group III: 3 sessions of 10 min walk at 70% of predicted VO₂ peak

b. Controls group (10 participants) did not do any of the PA.

Following resting HRV and resting BP intervention was given to study participants. Heart rate recovery 1 min (HRR 1 min) during 2 min of cooling down period, BP and HRV recording were done after 30 min of rest postexercise between sessions and 30 min postintervention period. Volunteers were advised to report any change and symptom during the intervention period.

Parameters measured

Blood pressure

BP was measured with the participant in the sitting position after 5 min of rest. In addition, participants were advised to avoid consumption of alcohol, coffee, tea, cigarette smoking, and exercise for at least 30 min prior to their BP measurements. BP recording was done at rest, after 30 min of rest between sessions and during postinterventional period.

Predicted VO, peak

It was calculated for each of the individuals by 6 MWT^[20] using the predicted VO₂ peak equation (6 min walk distance). Participants in the group were given intervention by 50%, 60%, and 70% of their predicted VO₂ peak. The variance in the predicted VO₂ peak for each group was nearly the same (3.5, 4.7, and 4.2). Results



observed in the study may be interpreted in light of the predicted VO₂ peak and the variance.

Anthropometry

Body weight, height, and waist circumference were measured twice with the participant in light indoor clothing without shoes during their baseline examination, (waist circumference was measured 1 cm above the participant's navel during minimal respiration).

Heart rate recovery

During first 2 min of cooling period postexercise.

Autonomic function test

HRV analysis was done by Physiopac (Medicaid) at the department at the point of entry, in last 15 min of rest period of postexercise between sessions and last 15 min of postintervention rest period.

Procedure

Baseline period

The participant reported to the laboratory between 08:30 and 09:00 h approximately and following attachment of leads for the monitors, the participants completed a sitting baseline rest period for 15 min. Accumulation of short sessions of PA intervention was started. The duration of each short bout of PA was 10 min. Each short session was followed by the 60 min rest period in the seated position.

Intervention

Successive short PA sessions were proceeded by a 15 min baseline period. The study was focused on the three rest periods following 10 min of walking session. After every session, a 60 min rest period was given during which heart rate (via electrocardiogram [ECG]) and BP (via aneroid auscultation) by a single observer was measured during the last 15 min of the rest period. The whole test was conducted over a period of 3 h. Following random allocation into groups, based on the allocated group participants in the group was given intervention by 50%, 60%, and 70% of their predicted VO₂ peak. The mode of PA was walking on a motorized treadmill without any inclination, and the intensity of each PA session was at 50% of predicted VO₂ peak for Group II, 60% of predicted VO₂ peak for Group II, and 70% of predicted VO₂ peak for Group II. The speed of the treadmill was set according to the corresponding predicted VO₂ peak calculated from the speed of the 6 MWT.

Heart rate variability

The ECG data were then used for the power spectral analysis of HRV. HRV is a noninvasive tool to assess, not to directly measure, autonomic function. Frequency domain measures of HRV were assessed using the Fast Fourier Transform. The total power was calculated by the standard deviation (SD) of the R-R interval (00.1 Hz). HRV of the record was computed using the whole range of high-frequency (HF) power (0.15–0.40 Hz), low-frequency (LF) power (0.04–0.15 Hz), and very LF power (0.003-0.04 Hz). Although the physiological roles of its components are not yet fully understood but HF component is known to be related to parasympathetic activity, but the association of LF component and the LF/HF ratio is still controversial.^[17] Normalized (%) values were calculated. Normalized units represent the relative value of each power component in proportion to the total power minus the very LF component. The ratio of LF power to HF power was determined. Normalized LF power, HF power, and the ratio of low to HF were averaged for the baseline period and for the last 10 min of the rest periods between 10 min walking sessions.

Statistical analysis

Descriptive statistics are presented as mean values and SDs. Differences across groups were examined with one-way analysis of variance (ANOVA) and within the group by repeated measures ANOVA for continuous variables such as BP, HRR 2 min, and autonomic function measurements over the baseline and three sessions. Differences between the sessions were calculated by the *post hoc* test. The association of confounding variables (body mass index [BMI], body composition age, and predicted VO₂ peak) to a reduction in BP among experimental and control pre-HT were analyzed by linear regression analysis. P < 0.05 as significant.

Results

All 40 pre-HT participants were randomly divided into four groups of control, 50% of predicted VO_2 peak, 60% of predicted VO_2 peak, and 70% of predicted VO_2 peak. Demographic characteristics in Table 1 shows that anthropomorphic variables such as age, height, weight, BMI, and body fat percent did not differ statistically between the groups. Autonomic function test at baseline did not show any significant difference between the groups, but sympathetic predominance was found in all of them. Salt intake was also found to be higher than the recommended value as per "WHO" but remained almost the same in all the groups [Table 1].

Autonomic function variables and BP measurements during rest period following each session of 10 min exercise over a 30 min period of PAaccum in all four groups was compared in Table 2.

Blood pressure reduction

The decrease in the SBP was observed in the entire interventional group across the three sessions, and the change was significant when

Table 1: Anthropomorphic, blood pressure, autonomic
parameters, and daily salt intake/day among all the groups

,		, 0	0		
Controls (n=10)	Group I (n=10)	Group II (n=10)	Group III (n=10)	Р	
29±8.9	32±10.1	30.4±6.6	27.1±6.5	NS	
171.1±4.6	166.7±8	167.8±7.3	167.2±11.1	NS	
82.2 ± 21.5	73.0±13.9	70.8±13.4	68.9 ± 17.2	NS	
28.1 ± 6.7	25.9 ± 3.0	25.4 ± 4.0	25.3±4.3	NS	
28.4 ± 6.6	27.5 ± 3.6	24.9 ± 4.6	24.5±7.2	NS	
69.8 ± 12.4	76.6±9.3	70.7±7.7	70.8 ± 8.8	NS	
$28.4{\pm}11.5$	24.9±11.6	26.4 ± 6.2	28.6 ± 8.1	NS	
3.3 ± 2.5	3.8 ± 2.1	2.8 ± 0.9	2.78±1.2	NS	
131.9±6.5	127.7±5.9	129.6±5.2	129.4±4.2	NS	
80.9 ± 8.5	82.1±6.2	80.6 ± 5.3	83±4.9	NS	
9.6±3.3	11.3±4.0	10.6 ± 2.7	9.89 ± 2.0	NS	
	(n=10) 29±8.9 171.1±4.6 82.2±21.5 28.1±6.7 28.4±6.6 69.8±12.4 28.4±11.5 3.3±2.5 131.9±6.5 80.9±8.5 9.6±3.3	(n=10)(n=10) 29 ± 8.9 32 ± 10.1 171.1 ± 4.6 166.7 ± 8 82.2 ± 21.5 73.0 ± 13.9 28.1 ± 6.7 25.9 ± 3.0 28.4 ± 6.6 27.5 ± 3.6 69.8 ± 12.4 76.6 ± 9.3 28.4 ± 11.5 24.9 ± 11.6 3.3 ± 2.5 3.8 ± 2.1 131.9 ± 6.5 127.7 ± 5.9 80.9 ± 8.5 82.1 ± 6.2 9.6 ± 3.3 11.3 ± 4.0	(n=10)(n=10)(n=10)29±8.932±10.130.4±6.6171.1±4.6166.7±8167.8±7.382.2±21.573.0±13.970.8±13.428.1±6.725.9±3.025.4±4.028.4±6.627.5±3.624.9±4.669.8±12.476.6±9.370.7±7.728.4±11.524.9±11.626.4±6.23.3±2.53.8±2.12.8±0.9131.9±6.5127.7±5.9129.6±5.280.9±8.582.1±6.280.6±5.39.6±3.311.3±4.010.6±2.7	(n=10)(n=10)(n=10)29±8.932±10.130.4±6.627.1±6.5171.1±4.6166.7±8167.8±7.3167.2±11.182.2±21.573.0±13.970.8±13.468.9±17.228.1±6.725.9±3.025.4±4.025.3±4.328.4±6.627.5±3.624.9±4.624.5±7.269.8±12.476.6±9.370.7±7.770.8±8.828.4±11.524.9±11.626.4±6.228.6±8.13.3±2.53.8±2.12.8±0.92.78±1.2131.9±6.5127.7±5.9129.6±5.2129.4±4.280.9±8.582.1±6.280.6±5.383±4.9	

Values in mean±SD; *P<0.05. SD: Standard deviation; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; BMI: Body mass index; LF: Low frequency; HF: High frequency; SD: Standard deviation; NS: Not significant compared to baseline. A significant change in SBP in rest period in between each session of walking was not observed. A significant decrease in SBP was also observed in the control group, but the decrease was less as compared to the interventional groups.

A significant decrease in DBP was observed across the three sessions in interventional groups, but a significant fall was not observed during the rest period in between each session of walking. The DBP was decreased in control group across all the three session of recording [Table 2 and Figure 1a].

Mean relative change in SBP and DBP following PAccum for 30 min was observed was more in interventional groups as compared to controls. No significant difference was observed, and the different grades of walking across the interventional group [Figure 1b].

Autonomic modulation

Autonomic modulation measured by HRV was averaged for the baseline period and for the rest period following short PA sessions of 10 min over a period of 30 min. The values of normalized HF power, normalized LF power, and the ratio of low to HF power during rest period following each session of 10 min exercise are summarized in Table 2. In the control group, LF and LF/HF ratio showed a decline in the values from the baseline after first two sessions, but the change was nonsignificant. HF values did rise after each session of rest, but the change was nonsignificant.

Groups	Parameter	Baseline	First session	ivity (walking) ove Second session	Third session	F	Р
Control	LF (nu)	69.8±12	62.4±16.9	62.1±9.0	71.2±10.8	1.4	NS
	HF (nu)	28.4 ± 11	34 ± 14.8	29.8 ± 10.7	31.8 ± 6.3	0.53	NS
	LF/HF	3.3 ± 2.5	2.5 ± 1.9	2.3±0.94	2.2 ± 0.61	0.33	0.02
	SBP (mmHg)	131.9 ± 6.5	128.7 ± 9.7	122.5±5.9**	124.7±5.4**	6.91	0.002
	DBP (mmHg)	80.9±8.5	76.3±8.1	74.9±5.5*	75.9±5.3	2.2	0.005
50% of predicted VO2 peak	LF (nu)	76.6±9.8	69.01 ± 10	72.46 ± 11.8	70.5 ± 8.8	1.07	NS
5070 of predicted 702 peak	HF (nu)	24.9±11	31.6±9.9	27.5±11.3	31.1±10.4	0.78	NS
	LF/HF	3.8 ± 2.0	2.8±1.4	3.2±1.6	2.6±1.3	1.4	NS
	SBP (mmHg)	127.7±5.7	119.1±10.7*	116.9±8.5**	116.1±8.6**	12.8	< 0.001
	DBP (mmHg)	82.1±5.9	76±8.2**	74.5±9.4**	74.5±10.2**	12.1	< 0.001
	HRR (beats)		21.7±4.1	21.5±3.6	23.2±6.6	0.77	NS
60% of predicted VO2 peak	LF (nu)	70.7 ± 7.7	71.7±10.1	67.8±10	64.4±14.7	0.81	NS
1 1	HF (nu)	26.4±6.2	24.8±9.1	29.3±10	32.2±13	1.06	NS
	LF/HF	2.8±1	3.5 ± 2.0	2.68 ± 1.2	2.7±1.5	0.47	NS
	SBP (mmHg)	129.6±5.0	120.8±9.7*	117.7±8** ^{,#}	117.7±7.1**	15.0	< 0.001
	DBP (mmHg)	80.6±5.1	74.1±7.7*	72.5±6.2*	75.9±7.8	5.6	0.007
	HRR (beats)		23.1±5.4	22.1±3.7	23.4±4.9	0.35	NS
70% of predicted VO2 peak	LF (nu)	70.8 ± 8.8	60.4±15	66.5±11.2	60.3±16.6	1.2	NS
	HF (nu)	28.6 ± 8.1	37.7±13.6	32.4±11	38.8±16.2	1.1	NS
	LF/HF	2.9±1.2	2.09 ± 1.5	2.4±1.3	2.2±1.5	0.51	NS
	SBP (mmHg)	128.4±4.1	118.8±7.4**	117.5±6.4**	117.8±5.4**	14.7	< 0.001
	DBP (mmHg)	83±4.7	73.6±5.3**	75.2±6.2**	75.5±6.3**	10.0	< 0.001
	HRR (beats)		25.6±7.2	24.5±7.1	23.3±5.6	0.64	NS

Table 2: Autonomic function, blood pressure, and heart rate recovery measurements for the baseline and rest period

*Groups compared by repeat measure ANOVA and *post hoc* test compare with baseline: *<0.05, **<0.01; "Comparison between first and second session; *Single tailed P<0.05. NS: Not significant; LF: Low-frequency; HF: High-frequency; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HRR: Heart rate recovery; ANOVA: Analysis of variance

Saxena, et al.: PAcumm lowers blood pressure in prehypertensive



Figure 1: (a) Change in systolic and diastolic blood pressure following different grades of accumulated physical activity over 30 min in prehypertensives. (b) Relative change in systolic and diastolic blood pressure following different grades of accumulated physical activity over 30 min in prehypertensives

Decline in the LF value and LF/HF value was observed in 50%, 60%, and 70% of predicted VO₂ peak group during the rest period after each session of walking, but the change was not statistically significant. HF values increased from the baseline in each session of walking in all intervention groups, but the change was nonsignificant.

Relative change in autonomic function analysis across all the groups showed that higher relative change in LF was observed in 70% of predicted VO_2 peak group following PAaccum of 30 min larger relative change in both LF and L/HF ratio was observed in all the interventional groups as compared to controls, but the change remained same in all grades of PA. When compared across the entire interventional group, the change was almost the same [Figure 2].

HRR 2 min following each session of PAaccum in the interventional group remained almost the same.

Association of relative change in SBP and DBP following different grades of PAcumm over 30 min with autonomic modulation such as HF, HRR, and LF/HF ratio did not show any significant relationship. The LF and LF/HF ratio was reduced in all intensity of walking but does not seem to be cause for the decline in BPs.

Discussion

Prehypertension is a critical population for prevention from progression to hypertension and lifestyle modification including the PAcumm is the only recommended treatment at this time.^[11] Moderate intensity of brisk walking accumulated toward the 30 min has been accepted and recommended for maintaining good health.^[21] PA walking was chosen as within present scenario of time constraints the only activity one can alter and incorporate is the one we do as a routine, i.e., walking. Grades of energy expenditure were set at predicted VO₂ peak and divided as 50%, 60%, and 70% of predicted VO₂ peak to study the effect of increasing intensity of walking on the BP reduction. Younger age group was studied as in present outlook they are more vulnerable

to risk factors associated with the development of hypertension. An acute study is justifiable as utilization of acute responses allows for a more efficacious study of possible variations in the accumulation of physical activity in lowering of the BP in prehypertension throughout the day. Finding reduction in BP as an immediate result may motivate the volunteer to continue incorporating the modification in PA in their daily routine.

In this study, we found a significant reduction in both SBP and DBP following PAcumm over a period of 30 min (P < 0.001). BP reduction during the rest period between short PA sessions in the accumulated PA over 30 min was also observed in pre-HT.

Reduction in SBP was observed after each of the sessions in rest period but was statistically significant only when compared to basal BP. A higher average reduction of SBP ($8.2 \pm 8 \text{ mmHg}$) was reported in the 1st session of PA of walking in 50%, 60%, and 70% of predicted VO, peak. Next two sessions did not show a major reduction (3.4 mmHg and 1.2 mmHg). Reduction in DBP was also observed following 30 min of PACumm (P < 0.001) and after each of the sessions in rest period but was statistically significant only when compared to basal BP. Major reduction was observed in the 1st session of 10 min in all grades of PA (1st session 7.3 mmHg, 2nd session 1.5 mmHg, and 3rd session 0.4 mmHg). A study done by Park et al. found a fall of SBP in all the session of PAcumm but the reduction of SBP in first two sessions was not significant when compared to baseline. Decrease in SBP in the third session was significant when compared to baseline (P = 0.03).^[22] Several other studies investigating the health outcomes like postexercise oxygen consumption during the rest periods found no significant reduction in SBP in first two sessions.[23]

The effect of exercise on decreasing SBP became stronger with each additional walking session to eventually result in a clinically meaningful reduction. Since the change in SBP was not much in second and third session it could be explained by the assumption that over the period of PEH a reflexive response of baroreceptor may partially offset the observed reduction in BP. Although PEH lasted several hours after PAccum,



Figure 2: Relative change in high-frequency, low-frequency, and high-frequency/low-frequency ratio following different grades of accumulated physical activity over 30 min in prehypertensives

Park et al. observed that the residual effect of BP decreases across rest intervals between subsequent short sessions of aerobic exercise in pre-HTs over 40 min of PACumm.^[15] The potential mechanisms underlying hypotension after one bout of exercise have been theorized to reflect a functional product of changes in cardiac output and total peripheral resistance (due to local vasodilation in working muscles), both of which are affected by thermoregulation, nitric oxide, various vasoactive hormones, and autonomic activity to name a few.^[24] Muscle sympathetic nerve activity (MSNA) studies by Floras et al.[16] have documented a decrease in sympathetic activity following exercise in borderline hypertension and since they exhibit a higher than normal sympathetic activity in resting condition, a transient suppression of augmented sympathetic outflow may be related to hypotension. As sympathetic outflow governs the peripheral resistance, both regional and systemic resistance may be decreased below the preexercise levels hence fall in DBP.

Although the mean arterial pressure is related to the cardiac output and total peripheral resistance, the fall in SBP as observed by us may not be related to CO, which depends on HR which is increased postexercise.^[25] Decrease in SBP as observed in our study may be related to decrease circulating catecholamine because of decreased sympathetic activity.^[26]

The magnitude of a BP reduction after exercise is dependent on the presenting BP. That is, patients who present with higher BPs exhibit greater BP reduction.^[27] Since baseline BP may be a predictor of PEH^[28] relative/normalized reduction in BP was taken for measuring the change and for seeing the association with autonomic modulation and other covariates. The mean relative change in SBP in all the grade of PACumm (50%, 60%, and 70% of predicted VO₂ peak) over 30 min was 0.09 \pm 0.05 mmHg (absolute -11.4 \pm 6.4 mmHg). Mean relative reduction in DBP was 0.08 \pm 0.07 mmHg (absolute -6.6 \pm 7.0 mmHg) following PACumm over 30 min in all grade of walking. We observed a higher fall in BP (9.9 \pm 8.0/9.4 \pm 5.8 mmHg) after the 1st session following 70% of predicted VO2 peak as compared to other grades of PA. Padilla et al. on their work on PAccumm following lifestyle PA over 8-12 period in a day among normotensive, pre-HT found difference in SBP after PA only in pre-HT (6.6 \pm 2.3 mmHg) which persisted for the duration of 6 h when ambulatory BP was measured.^[14] They did not find a reduction in DBP in normotensives and pre-HTs following the lifestyle PA. Murphy and Hardman^[10] reported a reduction in SBP to be greater in women who walked three short bouts of 10 min (-7.4 ± 7.3 mmHg). A study by Lacombe et al. on older pre-HTs also observed a reduction of SBP - 4 ± 6 mmHg following equicaloric bouts of exercise in the interval (IE: $5 \times 2:2$ min at 85% and 40% maximal oxygen uptake). Effect on DBP observed was raised rather than reduced following postexercise and was similar to that of control during the rest period.^[29]

Dunn *et al.* reported a similar reduction in both SBP and DBP in adult engaged in structured exercise (-3.2/5.1 mmHg) which was almost similar to the lifestyle PA (-3.6/5.3 mmHg) but after a long period of exercise.^[11] A reduction in ambulatory BP after a single session of exercise of 5–8 mmHg for SBP over 11–12 h and 6–8 mmHg for DBP for 6–8 h has been reported by Wallace.^[27]

Higher change in the LF_{nu} and LF/HF was observed in our study in all grades of exercise over 30 min of PACumm as compared to control. Fall in the mean values was observed in rest period after each of the PA session, but the change was nonsignificant. Sympathetic drive decreased after each bout of PA with a maximal reduction in 70% of predicted VO₂ peak. Which may be due to the fact that pre-HT exhibited higher than normal LF in resting condition and observed fall in BP may be due to transient suppression of augmented sympathetic flow.^[16] It may be possible that with reduced BP may not be an adequate stimulus for decreased sensitivity of baroreceptors to cause a significant reduction in sympathetic efferent activity. Reduction in normalized LF as a marker of sympathetic activity was observed in the control group but was small compared to interventional groups. Similar decrement in MSNA activity was observed by Halliwill in normotensives.^[30] HF_{nu} value showed an increase after each session of PA in all grades of exercise, but relative change in HF_{nu} was not significant over 30 min of PAcumm. Moreover, the change was almost same in all grades of exercise and comparable to control group.

In our study, the magnitude of change in autonomic parameters of LF_{nu} , HF_{nu} , and LF/HF ratio was not associated with the relative change in SBP and DBP the autonomic modulation did not mirror the change in BP. A study by Park *et al.* also could not find any association of autonomic parameters with a reduction in BP following PAcumm at 50% of predicted VO₂ peak over 30 min. They also could not find any significant difference in the autonomic parameters during the rest after each session of PA.^[22] A study by Liu *et al.*^[31] reported a decrease in the LF and HF following a single session acute exercise for 30 min at

65% of VO₂ max, and the difference was significant from the baseline suggesting a rebalancing of sympathovagal balance to heart,^[1] although parameters were recorded immediately after exercise during the lowest BP levels. A study by Lacombe *et al.* also observed that both HRV parameters LF (ms²) and HF (ms²) were reduced following equicaloric five sets of 2 min exercise and 2 min of active recovery at 85% maximal oxygen uptake (VO₂ max) and 40% VO₂ max, respectively, but in old pre-HTs and the difference of HF (ms²) was significant.^[29] Our findings are similar to those reported by Legramante *et al.* who found no significant changes in autonomic modulation, although they elicited a significant 11.9 mmHg reduction in SBP and a 5.3 mmHg reduction in DBP. ^[32] Postexercise (graded exercise to 87% of predicted maximum heart rate) BP and HRV were measured for 5 min between 60 and 90 min following a single session of exercise.^[32]

We observed a minimal rise in HF following PAcumm over 30 min, which was similar in all grades of exercise to that in control. Pober *et al.*^[33] found no significant increase in parasympathetic activity at 1, 3, and 6 h after 60 min of cycle ergometry at 65% of VO₂ peak. They did, however, find a significant decrease in sympathetic activity between 6 and 22 h following the exercise as observed in our study. It appears as though the change in autonomic modulation lags behind the change in early stage of PEH (1–2 h following the exercise). Halliwill^[30] suggested that a PEH is associated with a 30% reduction in sympathetic vasoconstrictor nerve activity to skeletal muscle, likely mediated by the arterial baroreceptors.

As observed in our study, grades of exercise do not seem to affect change in BP. The intensity of exercise of 50, 60, and 70% of predicted VO_2 peak and duration seems to be a weak predictor of BP reduction after acute exercise.^[24]

Therefore, an average reduction in BP of 11/6.6 mmHg is physiologically important though reduction following each bout of PA could not establish statistically significant levels. The reduction in resting sympathetic activity following 30 min of PAcumm was evident and was present after each session of activity which may suggest a decrease in vascular tone, hence fall in DBP. Change in the parasympathetic activity was not significant in rest period during the three sessions of PA (Same change in HRR 2 min and no change in HF_{nu}) but could be observed over a longer duration of PAcumm.

Conclusion

Autonomic function mechanisms underlying BP decrease during rest between several short periods of exercise over 30 min remain as yet unclear and could be different from those proposed for the longer standing PEH.

Limitation

It was a small group study, and the calculated predicted VO_2 peak was used instead of VO_2 max. The study results may be interpreted in light of the variance in the predicted VO_2 peak and

limited to the study group. Due to lack of funds, we could not procure the machine for the recording of VO_2 max and hence an indirectly calculated method which can only predict the value of VO_2 peak was used.

Acknowledgment

Authors acknowledge the HIMS, SRH University for providing finance and logistics for the work.

Financial support and sponsorship

HIMS, SRH University, Dehradun, Uttarakhand, India.

Conflicts of interest

There are no conflicts of interest.

References

- 1. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr., *et al.* Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension 2003;42:1206-52.
- 2. Qureshi AI, Suri MF, Kirmani JF, Divani AA, Mohammad Y. Is prehypertension a risk factor for cardiovascular diseases? Stroke 2005;36:1859-63.
- 3. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, *et al.* Primary prevention of hypertension: Clinical and public health advisory from the National High Blood Pressure Education Program. JAMA 2002;288:1882-8.
- 4. Dunn AL, Andersen RE, Jakicic JM. Lifestyle physical activity interventions. History, short- and long-term effects, and recommendations. Am J Prev Med 1998;15:398-412.
- 5. Lee IM, Skerrett PJ. Physical activity and all-cause mortality: What is the dose-response relation? Med Sci Sports Exerc 2001;33 6 Suppl: S459-71.
- 6. Trost SG, Owen N, Bauman AE, Sallis JF, Brown W. Correlates of adults' participation in physical activity: Review and update. Med Sci Sports Exerc 2002;34:1996-2001.
- 7. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, *et al.* Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Med Sci Sports Exerc 2007;39:1423-34.
- 8. At least five a week. Evidence on the impact of physical activity and its relationship to health. A report from the Chief Medical Officer. London, Department of Health, 2004 Available from: http://www.bhfactive.org.uk cmos-report-at-least-five-a-week.pdf. [Last accessed on 2015 Feb 19].
- 9. Moreau KL, Degarmo R, Langley J, McMahon C, Howley ET, Bassett DR Jr., *et al.* Increasing daily walking lowers blood pressure in postmenopausal women. Med Sci Sports Exerc 2001;33:1825-31.
- 10. Murphy MH, Hardman AE. Training effects of short and long bouts of brisk walking in sedentary women. Med Sci Sports Exerc 1998;30:152-7.
- 11. Dunn AL, Marcus BH, Kampert JB, Garcia ME, Kohl HW 3rd, Blair SN. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: A randomized trial. JAMA 1999;281:327-34.
- 12. Ebisu T. Splitting the distance of endurance running: On

cardiovascular endurance and blood lipids. Jpn J Phys Educ 1985;30:37-43.

- 13. DeBusk RF, Stenestrand U, Sheehan M, Haskell WL. Training effects of long versus short bouts of exercise in healthy subjects. Am J Cardiol 1990;65:1010-3.
- 14. Padilla J, Wallace JP, Park S. Accumulation of physical activity reduces blood pressure in pre- and hypertension. Med Sci Sports Exerc 2005;37:1264-75.
- 15. Park S, Rink LD, Wallace JP. Accumulation of physical activity leads to a greater blood pressure reduction than a single continuous session, in prehypertension. J Hypertens 2006;24:1761-70.
- 16. Floras JS, Sinkey CA, Aylward PE, Seals DR, Thoren PN, Mark AL. Postexercise hypotension and sympathoinhibition in borderline hypertensive men. Hypertension 1989;14:28-35.
- 17. Kulics JM, Collins HL, DiCarlo SE. Postexercise hypotension is mediated by reductions in sympathetic nerve activity. Am J Physiol 1999;276(1 Pt 2):H27-32.
- MacDonald JR, Hogben CD, Tarnopolsky MA, MacDougall JD. Post exercise hypotension is sustained during subsequent bouts of mild exercise and simulated activities of daily living. J Hum Hypertens 2001;15:567-71.
- 19. Booth M. Assessment of physical activity: An international perspective. Res Q Exerc Sport 2000;71 Suppl 2:114-20.
- 20. Cahalin L, Pappagianopoulos P, Prevost S, Wain J, Ginns L. The relationship of the 6-min walk test to maximal oxygen consumption in transplant candidates with end-stage lung disease. Chest 1995;108:452-9.
- 21. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, *et al.* Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA 1995;273:402-7.
- 22. Park S, Rink L, Wallace J. Accumulation of physical activity: Blood pressure reduction between 10-min walking sessions. J Hum Hypertens 2008;22:475-82.

- 23. Almuzaini KS, Potteiger JA, Green SB. Effects of split exercise sessions on excess postexercise oxygen consumption and resting metabolic rate. Can J Appl Physiol 1998;23:433-43.
- 24. MacDonald JR. Potential causes, mechanisms, and implications of post exercise hypotension. J Hum Hypertens 2002;16:225-36.
- 25. Headley SA, Claiborne JM, Lottes CR, Korba CG. Hemodynamic responses associated with post-exercise hypotension in normotensive black males. Ethn Dis 1996;6:190-201.
- 26. Tipton CM. Exercise, training, and hypertension. Exerc Sport Sci Rev 1984;12:245-306.
- 27. Wallace JP. Exercise in hypertension. A clinical review. Sports Med 2003;33:585-98.
- 28. Pescatello LS, Guidry MA, Blanchard BE, Kerr A, Taylor AL, Johnson AN, *et al.* Exercise intensity alters postexercise hypotension. J Hypertens 2004;22:1881-8.
- 29. Lacombe SP, Goodman JM, Spragg CM, Liu S, Thomas SG. Interval and continuous exercise elicit equivalent postexercise hypotension in prehypertensive men, despite differences in regulation. Appl Physiol Nutr Metab 2011;36:881-91.
- Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. Exerc Sport Sci Rev 2001;29:65-70.
- 31. Liu S, Goodman J, Nolan R, Lacombe S, Thomas SG. Blood pressure responses to acute and chronic exercise are related in prehypertension. Med Sci Sports Exerc 2012;44:1644-52.
- 32. Legramante JM, Galante A, Massaro M, Attanasio A, Raimondi G, Pigozzi F, *et al.* Hemodynamic and autonomic correlates of postexercise hypotension in patients with mild hypertension. Am J Physiol Regul Integr Comp Physiol 2002;282:R1037-43.
- 33. Pober DM, Braun B, Freedson PS. Effects of a single bout of exercise on resting heart rate variability. Med Sci Sports Exerc 2004;36:1140-8.