Exercise intensity alters postexercise hypotension

Linda S. Pescatello[a], Margaux A. Guidry[a], Bruce E. Blanchard[ab], Allison Kerr[a], Amy L. Taylor[b], Amy N. Johnson[a], Carl M. Maresh[a], Nancy Rodriguez[a] and Paul D. Thompson[b]

Objective Blood pressure (BP) is immediately lowered after a session of dynamic exercise, e.g. *postexercise hypotension* (PEH). The optimal exercise intensity needed to evoke PEH has not been established. We examined the effect of light (LITE) and moderate (MOD) exercise intensity on PEH.

Design Subjects were 49 men (mean ± SEM, 43.8 ± 1.4 years) with high normal to stage 1 hypertension (145.0 ± 1.5/85.8 ± 1.1 mmHg). Men randomly completed three blinded experiments: a control session and two cycle exercise bouts, one at 40% (LITE) and the other at 60% (MOD) of maximal oxygen consumption.

Methods Experiments began with a baseline period and were conducted at the same time of day and separated by $\geqslant$ 2 days. Subjects wore an ambulatory BP monitor after the experiments. Repeated measure analysis of variance (ANOVA) tested if BP and heart rate differed over time and between experimental conditions. Multivariate regression tested factors related to the BP response.

Results For 9 h after all experiments, average awake systolic blood pressure (SBP) increased and diastolic blood pressure (DBP) decreased compared with baseline ($P < 0.001$). Average awake SBP increased up to 6.9 mmHg less ($P < 0.001$) and DBP decreased 2.6 mmHg more ($P < 0.05$) after exercise versus control. For 5 h, PEH was greater after MOD; but over the course of 9 h, LITE was as effective as MOD in eliciting PEH. Baseline BP was the primary factor explaining the BP response ($\beta = -0.434$ to -0.718, $r^2 = 0.096-0.295$).

Conclusions LITE and MOD evoked PEH throughout the daytime hours. Lower intensity dynamic exercise such as walking, contributes to BP control in men with hypertension. *J Hypertens* 22:1881–1888 © 2004 Lippincott Williams & Wilkins.

Journal of Hypertension 2004, **22**:1881–1888

Keywords: blood pressure, exercise, hypertension, men, physical activity, prevention

[a]University of Connecticut, Storrs and [b]Hartford Hospital, Hartford, Connecticut, USA.

Sponsorship: This work was supported by an American Heart Association Grant-in-Aid No. 0150507N.

Correspondence and requests for reprints to Linda S. Pescatello, Associate Professor and Director of the Center of Health Promotion, University of Connecticut, School of Allied Health, 358 Mansfield Road, U-2101, Storrs, Connecticut 06269–2101, USA.
Tel: +1 860 486 0008; fax: +1 860 486 1588;
e-mail: Linda.Pescatello@uconn.edu

Received 3 November 2003 **Revised** 13 May 2004
Accepted 25 May 2004

See editorial commentary page 1859

Introduction

Hypertension (HTN) is a major public health problem that affects nearly 29% of US adults [1]. HTN is the most common primary diagnosis in Americans with 90% of middle-aged and older adults likely to develop HTN [2]. Those with high normal blood pressure (BP) are at highest attributable risk of cardiovascular disease (CVD) with 50% of related CVD events occurring in this group [3–5]. For these reasons, practice of healthy lifestyle behaviors such as habitual physical activity is recommended for the prevention and control of HTN [1,5,6].

Endurance exercise training lowers BP between 5 [7] and 10 mmHg [8,9] in those with HTN with the greatest reductions seen in those with the highest pre-exercise BP [10,11]. Although studies are limited in number and many lack adequate controls, moderate intensity aerobic exercise is professed to be at least as effective as vigorous intensity exercise in eliciting this response [8]. Hagberg *et al.* [9] have noted, however, 25% of the people with HTN do not lower their BP with dynamic exercise training.

Isolated aerobic exercise sessions produce immediate decreases in BP that can persist for 22 h after exercise [12]. These BP reductions below control levels following acute exercise have been named, postexercise hypotension (PEH) [13]. The immediacy by which PEH occurs suggests some if not all of the BP benefit attributed to endurance exercise training may actually be an acute phenomenon related to recent exercise [14]. The reductions in BP resulting from isolated exercise sessions as with those produced by long term

0263-6352 © 2004 Lippincott Williams & Wilkins

exercise training are greatest in people with the highest pre-exercise BP values [15]. Preliminary evidence indicates PEH may be a low intensity threshold event [12,16–20], meaning the BP reductions occur after low levels of physical exertion. However, not all people with HTN demonstrate PEH, and the reasons for this variable response are not clear [15,16,21].

The present study examined the influence of exercise intensity on PEH. Because PEH appears to be a low intensity threshold event, we compared the effectiveness of light and moderate intensity endurance exercise in eliciting PEH in men with high normal to stage 1 HTN. We hypothesized light and moderate intensity exercise would similarly evoke PEH, and the largest BP reductions would occur in men with the highest baseline BP.

Methods

Subjects

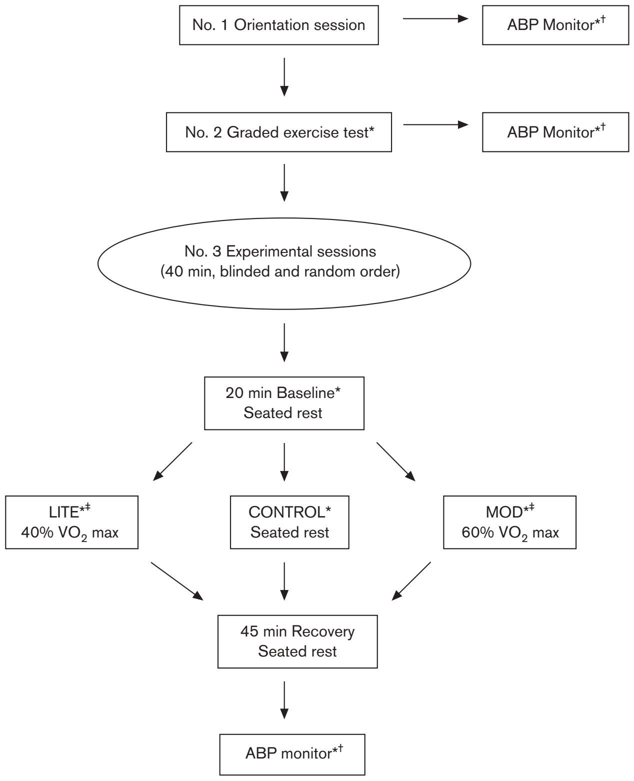
Volunteers were 49 men between 18 and 55 years, with high-normal to stage 1 HTN [systolic BP (SBP) ⩾ 130–159 and/or diastolic BP (DBP) ⩾ 85–89 mmHg]. Subjects were excluded if they had a SBP ⩾ 160 mmHg and/or DBP ⩾ 100 mmHg, CVD, diabetes mellitus, asthma, thyroid dysfunction, pancreatitis, acute illness and/or were on antidepressant medication. Volunteers had no physical limitations prohibiting exercise and were non-smokers. Subjects completed an informed consent approved by the Institutional Review Boards of the University of Connecticut and Hartford Hospital.

Any medications potentially influencing the BP response to exercise including antihypertensives, hyperlipidemics, anticoagulants, non-steroidal anti-inflammatory agents, nutritional supplements other than a one-a-day vitamin, cold medications, and herbal supplements were discontinued at least 4 weeks prior to testing. If medications were withdrawn, study investigators regularly monitored participants' BP during the washout period. Men in whom the withdrawal of antihypertensive medication resulted in resting SBP ⩾ 160 and/or DBP ⩾ 100 mmHg were excluded.

Procedures

The study design overview is shown in Figure 1. Subjects initially participated in an orientation session to familiarize them with the study and to ensure their BP met the study inclusion criteria. Volunteers were weighed on a standard balance-beam scale (Model 339; Detecto, Webb City, Missouri, USA) and height was recorded to calculate body mass index (kg/m^2). Waist circumference was obtained at the iliac crest with a Gulick tape measure. After a minimum of 5 min of seated rest, BP measurements were taken three times, 5 min apart in each arm by auscultation and averaged. An ambulatory BP (ABP) monitor (Accutracker II automatic noninvasive ambulatory BP monitor; Suntech Medical Instruments Inc., Raleigh, North Carolina) was then attached to each subject.

Fig. 1



Study design overview. ABP, ambulatory blood pressure; VO$_2$max, maximal oxygen consumption; * Blood pressure and heart rate taken throughout; † worn until waking the next morning; ‡ includes 5 min warm-up and 5 min cool down periods to total 40 min of exercise. LITE, 40%VO$_2$max; MOD, 60%VO$_2$max.

The ABP monitor was calibrated with a mercury sphygmomanometer until three successive ABP measurements were within 5 mmHg of measurements made by auscultation. In order to prevent the subject from anticipating the time of an ABP reading, the BP sampling interval was programmed to take three ABP measurements per hour at random intervals that were determined by the monitor to be between 15 to 20 min apart until 2300 h, and then ABP measurements were taken hourly thereafter. The mean time of day for ABP attachment time was 1230 h. Volunteers left the laboratory with instructions to proceed with their usual activities, not to engage in formal exercise, keep their arm extended and still at the time of measurement, and return the monitor the following day. They were given a journal to record activities performed during a measurement and any unusual events. The computerized recordings were considered acceptable if at least 80%

of the potential ABP readings were obtained. Values were deleted when they met the manufacturer's quality control rejection criteria. If orientation awake ABP averaged $< 135/85$ mmHg [22], subjects were excluded from further participation. The Accutracker II ABP monitor has demonstrated less disparity and closer levels of agreement with intraarterial BP than clinicians' measurements at rest and during exercise [22,23] and superior short and long term reproducibility over office determinations [24].

Following the orientation session, volunteers completed a graded cardiopulmonary exercise stress test (GXT) to exclude occult coronary artery disease and determine the exercise workload. Maximal oxygen consumption (VO_2max) was measured by breath-by breath analysis of expired gases during testing via an open circuit respiratory apparatus (Sensormedics Vmax 29 Metabolic Cart; SensorMedics Corp., Yorba Linda, California, USA), while on an incremental resistance exercise test on a cycle ergometer (Monark Ergomedic 818E, Stockholm, Sweden). The exercise test consisted of continuous cycling at a cadence of 60 rev/min with resistance increased by 0.5 kp (30 W) every 2 min until volitional exhaustion. Heart rate (HR) was recorded continuously with a 12-lead ECG system (Marquette Case 8000; Jupiter, Florida, USA). SBP and DBP were measured every 2 min by auscultation. The GXT results were used to calculate the intensity of the experimental exercise sessions from a linear regression plot of work rate and HR as a function of VO_2. Subjects left the GXT attached to the ABP monitor to further acquaint them with the technology [25].

Subjects then completed three blinded experiments in random order that were separated by $\geqslant 2$ days. Experiments began between 0800 and 1100 h, depending on the subject's schedule. Once a starting time was chosen by the subject, all experiments were conducted at the same time of day for that subject. The experiments consisted of a control session of seated rest, and two cycle exercise bouts, one performed at light intensity (40% VO_2max, LITE) and the other at moderate intensity (60% VO_2max, MOD) (Fig. 1). Volunteers were instructed to abstain from exercise for 48 h, consume a standard meal and refrain from caffeinated food and beverages for 4 h before all experimental sessions.

Volunteers began each experiment with a 20-min baseline period in the seated position with HR and BP recorded every 2 min. HR was measured with a HR monitor (Model No. 1902750; Polar Electro Inc, Woodbury, New York, USA) and BP by auscultation. The same investigator measured BP before, during and after all experiments with a standard mercury sphygmomanometer in all subjects. Subjects were blinded to the experiment they were to perform, e.g. exercise or control, until the completion of the baseline period. The exercise sessions consisted of a 5-min warm up, 30 min of cycling at either LITE or MOD and a 5-min cool down, totaling 40 min of exercise. VO_2 was monitored during steady-state exercise (Sensormedics Vmax 29 Metabolic Cart; SensorMedics Corp.) to verify the designated exercise intensity. The average steady-state VO_2max maintained during the LITE phase was $40.3 \pm 0.0\%$, and $59.9 \pm 0.0\%$ for the MOD phase. The subjects also completed a 40-min control session of seated rest. HR and BP were measured at 5-min intervals during the experiments. Each experiment was followed by a 45-min recovery period in the seated position with BP and HR measured every 2 and 3 min, respectively. Following recovery, subjects were attached to the ABP monitor. Despite being told to keep the unit on until waking the next morning, many of the subjects had disconnected the unit by 0300 h, because of interrupted sleep.

Statistical analyses

Descriptive statistics were calculated on study variables. Repeated measure analysis of variance (RM-ANOVA) tested if BP and HR differed over time and between experimental conditions (control and exercise) and exercise intensities (LITE and MOD). There were significant BP × time interactions noted at 5 h after exercise between LITE and MOD, so that BP and HR results are reported for 5 h after the experimental sessions; and for 9 h, the observed duration of PEH when all men were awake and out-of-bed.

Pearson correlations examined the relationships between independent and dependent variables for 5 h and 9 h after exercise and control. Independent variables included age, baseline and orientation BP, body mass index (BMI), waist circumference, and VO_2max. Dependent variables were the mean BP response for 5 h and 9 h after control, LITE and MOD. Of these variables, baseline BP, orientation ABP, age and VO_2max were found to be significant correlates with the BP response.

Multivariate regression analyses then determined if baseline BP, orientation ABP, age and VO_2max were related to the BP response after, compared with before, control and exercise. In these regression analyses, the degree of collinearity among predictors was examined to ensure the covariances between the independent variables were low. Findings from the multivariate regression analyses are shown in Table 1 as reduced models containing only the predictors found to be significant. All statistical analyses were performed with the Statistical Package for Social Sciences Base 11.0 for Windows (SPSS Inc., Chicago, Illinois, USA) using two-

Table 1 Significant predictors of the mean awake blood pressure change from baseline for 5 and 9 h after control and exercise

	Condition	Variable entered	Beta	*t*	*P*	R^2
5 h SBP	Control	Baseline SBP	−0.587	−4.971	0.000	0.345
	LITE	Baseline SBP	−0.572	−5.162	0.000	0.447
		Age	−0.271	−2.445	0.018	
	MOD	Baseline SBP	−0.728	−6.330	0.000	0.478
		ASBP	0.431	3.749	0.000	
9 h SBP	Control	Baseline SBP	−0.606	−5.221	0.000	0.367
	LITE	Baseline SBP	−0.663	−5.697	0.000	0.426
		ASBP	0.265	2.275	0.028	
	MOD	Baseline SBP	−0.718	−6.299	0.000	0.488
		ASBP	0.487	4.271	0.000	
5 h DBP	Control	Baseline DBP	−0.600	−5.142	0.000	0.396
	LITE	Baseline DBP	−0.434	−3.016	0.004	0.186
		ADBP	0.321	2.264	0.028	
	MOD	Baseline DBP	−0.497	−4.248	0.000	0.423
		ADBP	0.413	3.523	0.001	
		Relative VO_2max	−0.386	−3.390	0.001	
9 h DBP	Control	Baseline DBP	−0.703	−5.742	0.000	0.423
		ADBP	0.258	2.112	0.040	
	LITE	Baseline DBP	−0.473	−3.446	0.001	0.260
		ADBP	0.422	3.081	0.004	
	MOD	Baseline DBP	−0.524	−4.034	0.000	0.339
		ADBP	0.320	2.472	0.017	
		Relative VO_2max	−0.292	−2.376	0.022	

SBP, systolic blood pressure; ASBP, ambulatory systolic blood pressure obtained after orientation; DBP, diastolic blood pressure; ADBP, ambulatory diastolic blood pressure obtained after orientation; VO_2max, maximal oxygen consumption; LITE, 40%VO_2max; MOD, 60%VO_2max.

tailed tests with $P < 0.05$ established as the level of statistical significance.

Results

Subjects

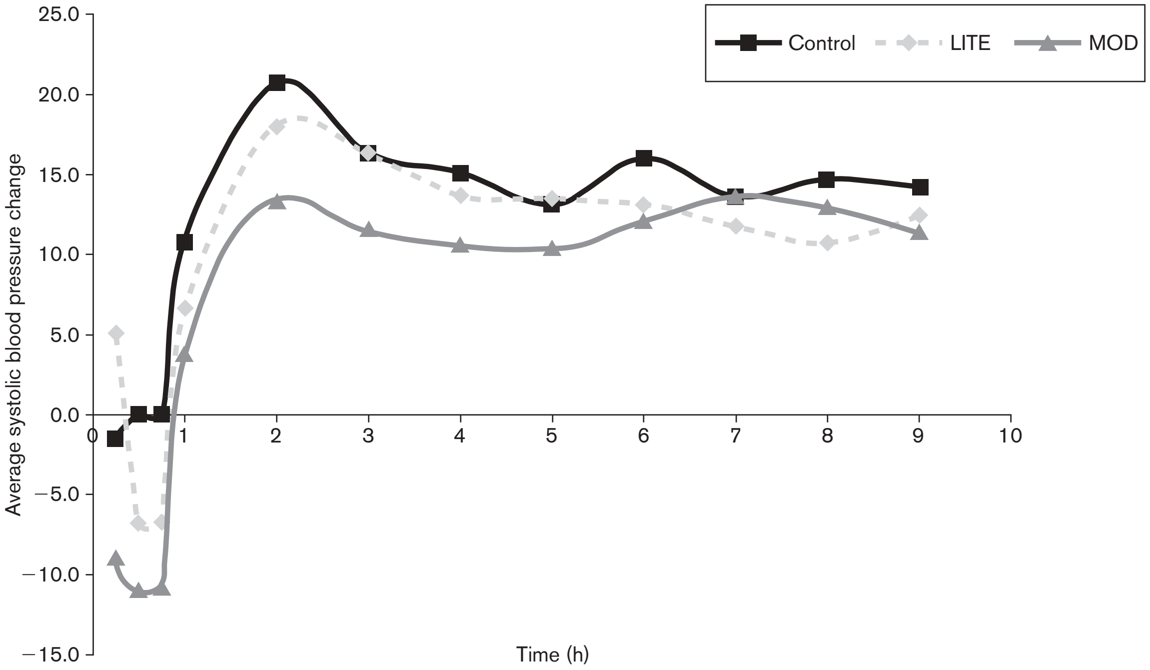
Participants ($n = 49$) were Caucasian men with a mean age of 43.8 ± 1.4 years, and an orientation mean awake ABP of 145.0 ± 1.5/85.8 ± 1.1 mmHg and night ABP of 125.7 ± 2.7/71.9 ± 1.3 mmHg. Subjects were overweight with a BMI of 29.4 ± 0.7 kg/m^2 and waist circumference of 101.8 ± 2.0 cm and had below average physical fitness (VO_2max, 31.3 ± 0.9 ml/kg per min) for men of their age [26]. Nearly half (49.0%) of the men reported a family history of HTN and 12 men discontinued antihypertensive or hyperlipidemic medications or both in order to participate.

Hemodynamic response

Over the course of 9 h, average SBP increased (Fig. 2) and DBP decreased (Fig. 3) from a mean baseline of 126.0 ± 1.8 and 87.1 ± 1.2 mmHg, respectively, following the experimental sessions ($P < 0.001$). The mean changes in awake SBP (Fig. 4) and DBP (Fig. 5) from baseline are shown for 5 h, the time point of the significant BP × Time experimental interactions; and for 9 h, the observed duration of PEH when all subjects were awake and ambulating. The mean increase in awake SBP from baseline for 5 and 9 h was less after exercise compared with control (Fig. 4) ($P < 0.05$), and the mean increase in awake SBP was less for 5 h after MOD than LITE ($P < 0.01$). Thus, average SBP was lower for 5 and 9 h after LITE (132.0 ± 1.5 and 134.0 ± 1.6 mmHg) and MOD (129.8 ± 1.6 and 133.0 ± 1.7 mmHg) compared with control (134.3 ± 1.7 and 136.1 ± 1.6 mmHg) ($P < 0.05$). For 5 h, the mean decrease in awake DBP was greater (Fig. 5) and average DBP lower after MOD (84.8 ± 1.2 mmHg) versus control (86.5 ± 1.2 mmHg) ($P < 0.05$). Although the mean decrease in awake DBP was not different after LITE than control ($P > 0.05$), average DBP tended to be lower after LITE (85.1 ± 1.3 mmHg) compared with control (86.5 ± 1.2 mmHg) ($P = 0.056$). Over the course of 9 h, the mean decrease in awake DBP was greater after exercise than control, but these differences did not achieve statistical significance ($P > 0.05$). Nonetheless, the decrease in awake DBP was sufficient to lower average DBP to a greater extent after LITE (84.1 ± 1.3 mmHg) ($P = 0.022$) than MOD (84.6 ± 1.2 mmHg) and control (85.6 ± 1.2 mmHg).

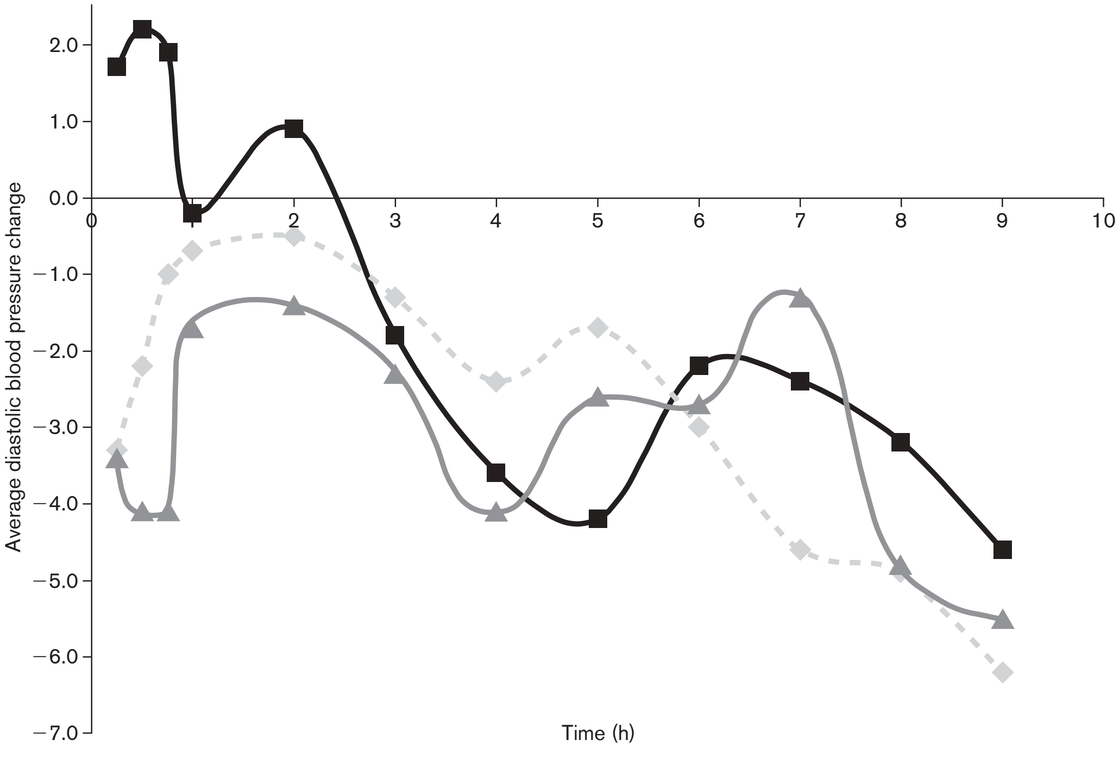
For 5 h, the mean increase in awake HR from baseline was more after exercise than control (Fig. 6) ($P < 0.05$), and average HR was higher after LITE (74.3 ± 1.7 bpm) and MOD (82.0 ± 1.8 bpm) compared with control (70.7 ± 1.5 bpm) ($P < 0.001$). In addition, the mean increase in awake HR and average HR were greater after MOD than LITE ($P < 0.001$). Over the course of 9 h, the mean increase in HR from baseline was greater after MOD than LITE and control ($P < 0.001$). Thus, average HR was higher after MOD (80.2 ± 1.7 bpm) than LITE (74.8 ± 1.6 bpm) and control (72.1 ± 1.5 bpm) ($P < 0.001$).

Fig. 2



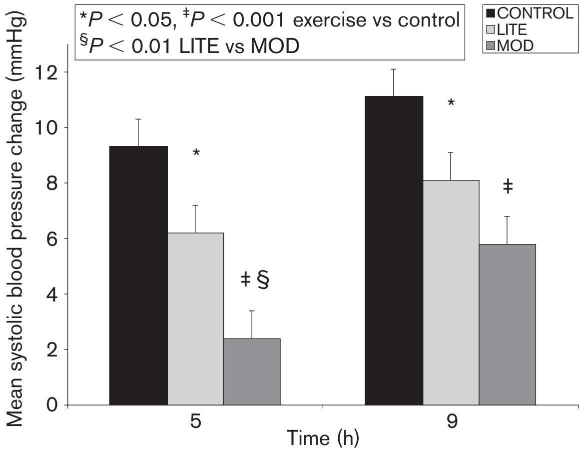
Average awake systolic blood pressure change from baseline (± SEM) at hourly intervals for 9 h after control and exercise. LITE, 40%VO_2max; MOD, 60%VO_2max.

Fig. 3



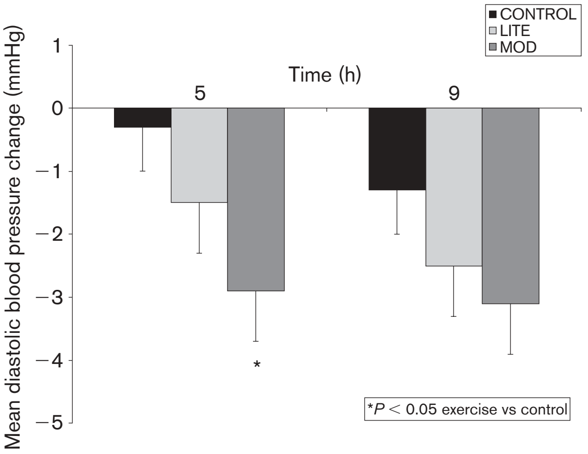
Average awake diastolic blood pressure change from baseline (± SEM) at hourly intervals for 9 h after control and exercise compared with baseline values. LITE, 40%VO_2max; MOD, 60%VO_2max.

Fig. 4



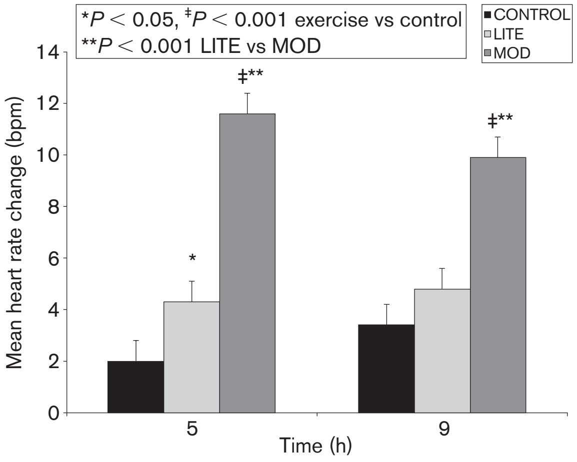
Mean change in awake systolic blood pressure from baseline (± SEM) for 5 and 9 h after control and exercise. LITE, 40%VO_2max; MOD, 60%VO_2max.

Fig. 5



Mean change in awake diastolic blood pressure from baseline (± SEM) for 5 and 9 h after control and exercise. LITE, 40%VO_2max; MOD, 60%VO_2max.

Fig. 6



Mean change in awake heart rate from baseline (± SEM) for 5 and 9 h after control and exercise. LITE, 40%VO_2max; MOD, 60%VO_2max.

Predictors of the BP response

Results from the multivariate regression analyses are displayed in Table 1 as reduced models with only significant predictors of the BP response retained. The strongest correlates with the mean awake BP change following exercise were baseline BP and orientation ABP, with 18.6 to 48.8% of the PEH variance accounted for by baseline BP and ABP (Table 1). The study participants having the highest baseline BP and lowest ABP experienced the greatest reductions in BP from baseline values after exercise; however, baseline BP was the stronger predictor of the two. One exception to these observations was baseline SBP and age were the significant predictors of the mean awake SBP response for 5 h after LITE with the older men having greater decreases in SBP than the younger men. An additional variable noted to be a significant correlate with the mean awake DBP change for 5 and 9 h after MOD was relative VO_2max with the mean decrease in DBP greatest in the more physically fit men. Baseline BP was the strongest correlate of the BP response following control, explaining 34.5 to 39.6% of the PEH variance.

Discussion

We examined the influence of LITE and MOD endurance exercise on PEH among 49 middle-aged men with high-normal to stage 1 HTN. For the first 5 h, the magnitude of PEH was greater after MOD; however, over the course of 9 h, LITE was as effective as MOD in eliciting PEH. Average awake SBP increased up to an average of about 6.9 mmHg less and DBP decreased an average of 2.6 mmHg more on the days the men exercised compared with the days they did not. Small decrements in SBP and DBP of 2 mmHg decrease the risk of stroke by 14% and 17% and the risk of coronary artery disease by 9% and 6%, respectively [27]. Our findings indicate public health benefits could be realized by people with HTN if they habitually engaged in lower levels of endurance exercise equivalent to a casual to brisk-paced walk.

The results of the present investigation expand upon those of our earlier work [15,16]. They establish PEH occurs in men with high-normal to stage 1 HTN after LITE and MOD for the remainder of the daytime hours; and affirm baseline BP predicts PEH such that the men with the highest baseline BP experience the largest postexercise reductions in BP. Two new preliminary observations from this study are that LITE lowered SBP to the greatest degree in the older men; and MOD reduced DBP to the lowest levels in the more physically fit men. Thus, LITE is a sufficient stimulus to evoke PEH among those with HTN, particularly in older men, and MOD seems to work best as antihypertensive therapy in the men who are more physically fit. These findings indicate clinicians can tailor exercise prescriptions to optimize the health, safety and enjoyment needs of their patients with HTN while maximizing BP benefit [28,29].

Surprisingly, men with lower orientation ABP elicited PEH to the greatest degree after exercise. Baseline BP and orientation ABP were positively correlated in linear regression analyses, yet when entered into the multivariate model they differentially impacted the postexercise BP response. One explanation for these findings is that the men with the highest baseline BP and lowest orientation ABP, or those exhibiting white-coat HTN, responded most favorably to exercise as antihypertensive therapy. Another possible explanation is that the first time the subjects wore the ABP was after the orientation session. Initial use of an ABP monitor results in higher BP compared to successive monitoring sessions due to an ABP monitor habituation effect [25]. Our observations are consistent with these reports. Study participant mean awake BP was highest after the orientation session compared with any of the other ambulatory monitoring sessions. These data indicate wearing of the ABP monitor appeared to initially serve as a BP perturbation event, possibly explaining the difference we observed in the relationships among PEH and baseline BP and orientation ABP. ABP habituation effects as found in this study indicate the importance of subjects completing multiple ABP monitoring sessions prior to performing any experimental interventions in PEH study designs.

Another unexpected finding of this investigation was exercise intensity differentially modulated PEH depending on the duration of the observation. For the first 5 h, PEH was greater after MOD exercise; but for the remainder of the day LITE appeared to be as effective as MOD in evoking PEH. Reasons for these observations are unclear, but may reside in the proposed mechanisms for PEH which include exercise-induced alterations in baroreceptor function, i.e. either a shift to a lower BP operating point or an increased responsiveness to changes in BP during the postexercise period [30]. It is plausible that exercise-induced alterations in baroreceptor function contributed to the more pronounced BP-lowering effects of MOD initially; however, over time other PEH mechanisms became operative that augmented the BP benefits of LITE for the remainder of the daytime hours. Another probable candidate for the greater BP-lowering effects of MOD than LITE for the first 5 h after exercise is that sympathetic inhibition or functional sympatholysis, defined as an intensity-dependent blunting of responsiveness to α- and β-adrenergic receptor stimulation and/or reduced noradrenaline release, was more pronounced after MOD than LITE over this time period [31,32]. As these and other PEH mechanisms become more clearly understood, they may provide insight into reasons for the exercise intensity cross-over effects we observed between LITE and MOD.

Our inclusion criteria were purposely restrictive so as to limit the strong confounding effects of age, gender, medication and disease, but they also limit the generalization of our findings to the population studied: white, middle-aged men with high-normal to stage 1 HTN. Despite our narrowly defined inclusion criteria, the differential BP responses we found between LITE and MOD and among men of varying BP, age and levels of physical fitness indicate the importance of the study design issues that need to be considered before undertaking studies examining the influence of exercise on HTN. These considerations include the need for a resting control condition, a baseline BP stabilization period, sufficient numbers of subjects, and accurate determination of HTN status with multiple BP assessments made prior to experimentation, ideally with ABP monitoring.

In summary, MOD evoked PEH to greater levels initially; but over the duration of the awake hours, LITE was as effective a PEH stimulus as MOD exercise among men with high-normal to stage 1 HTN. LITE appeared to lower BP to the greatest levels in the older men and MOD in the more physically fit men in this study sample, however, further investigation is needed to confirm these preliminary observations. Lower intensity dynamic exercise should be prescribed for men with elevated BP because it maximizes BP benefit in a population predisposed to the risks of more vigorous exercise. The acute BP-lowering effects of exercise are immediate and do not require prolonged exercise training. Consequently, important public health benefits could be achieved if people with HTN habitually engaged in lower levels of endurance exercise equivalent to a casual to brisk-paced walk.

Acknowledgements

We thank the subjects who volunteered their time; corporate and municipal agencies who enabled subject recruitment; University of Connecticut graduate students who assisted with data collection; Dr Jonna Kulikowich from Pennsylvania State University and Dr Ann O'Connell from the University of Connecticut for their statistical expertise; and Dr Richard Seip for his insightful review of the manuscript.

References

1 Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1998–2000. *JAMA* 2003; **290**:199–206.

2 Vasan RS, Beiser A, Seshadri S, Beiser, A, Sudha S, Larson M, *et al*. Residual lifetime risk for developing hypertension in middle-aged women and men: the Framingham Heart Study. *JAMA* 2002; **287**:1003–1010.

3 World Health Organization-International Society of Hypertension. 2003 World Health Organization (WHO)-International Society of Hypertension (ISH) statement on the management of hypertension. *J Hypertens* 2003; **21**:1983–1992.

4 European Society of Hypertension. European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* 2003; **21**:1011–1053.

5 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. The 6th Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. (JNC VI). *Arch Intern Med* 1997; **157**:2413–2446.

6 Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, *et al*. Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. JNC 7 – Complete Version. *Hypertens* 2003; **42**:1206–1252.

7 Whelton, SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med*. 2002; **136**:493–503.

8 American College of Sports Medicine. Position Stand. Exercise and Hypertension. *Med Sci Sports Exerc* 2004; **36**:533–553.

9 Hagberg, JM, Park JJ, Brown MD. The role of exercise training in the treatment of hypertension: an update. *Sports Med* 2000; **30**:193–206.

10 Fagard RH. Physical fitness and blood pressure. Exercise characteristics and the blood pressure response to dynamic physical training. *Med Sci Sports Exerc* 1993; **11**(**suppl**):S47–S52.

11 Fagard RH. Exercise characteristics and the blood pressure response to dynamic physical training. *Med Sci Sports Exerc* 2001; **33**(**suppl**):S484–492.

12 Rhonda UPM, Alves MJNN, Braga AMFW, Teixeira OTUN, Barretto ACP, Krieger EM, *et al*. Postexercise blood pressure in elderly hypertensive patients. *J Am Coll Cardio* 2002; **39**:676–682.

13 Kenney MJ, Seals DR. Postexercise hypotension: key features, mechanisms, and clinical significance. *Hypertens* 1993; **22**:653–664.

14 Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Med Sci Sports Exerc* 2001; **33**:S438–445.

15 Pescatello LS, Kulikowich JM. The after effects of dynamic exercise on ambulatory blood pressure. *Med Sci Sports Exerc* 2001; **33**: 1855–1861.

16 Pescatello LS, Fargo AE, Leach CN Jr, Scherzer HH. Short-term effect of dynamic exercise on arterial blood pressure. *Circulation* 1991; **83**: 1557–1561.

17 Hagberg JM, Montain SJ, Martin WH. Blood pressure and hemodynamic responses after exercise in older hypertensives. *J Appl Physiol* 1987; **63**:270–276.

18 Quinn TJ. Twenty-four hour, ambulatory blood pressure responses following exercise: impact of exercise intensity. *J Hum Hypertens* 2000; **14**:547–553.

19 Wallace JP, Bogle PG, King BA, Krasnoff JB, Jastremski CA. The magnitude and duration of ambulatory blood pressure reduction following acute exercise. *J Hum Hypertens* 1999; **13**:361–366.

20 Taylor-Tolbert NS, Dengel DR, Brown MD, McCole SD, Pratley RE, Ferrell RE, *et al*. Ambulatory blood pressure after acute exercise in older men with essential hypertension. *Am J Hypertens* 2000; **13**:44–51.

21 Pescatello LS, Miller B, Danias PG, Werner M, Hess M, Baker C, *et al*. Dynamic exercise normalizes resting blood pressure in mildly hypertensive premenopausal women. *Am Heart J* 1999; **138**:916–921.

22 Schettini C, Bianchi M, Nieto F, Sandoya E, Senra H. Ambulatory blood pressure normality and comparison with other measurements. *Hypertens* 1999; **34**(part 2):818–825.

23 White WB, Lund-Johansen P, Omvik P. Assessment of four ambulatory blood pressure monitors and measurements by clinicians versus intraarterial blood pressure at rest and during exercise. *Am J Cardiol* 1990; **65**:60–66.

24 Mansoor GA, McCabe EJ, White WB. Long-term reproducibility of ambulatory blood pressure. *J Hypertens* 1994; **12**:703–708.

25 Calvo C, Hermida R, Ayala DE, López JE, Fernández J, Dominguez MJ, *et al*. The 'ABPM effect'gradually decreases but does not disappear in successive sessions of ambulatory monitoring. *J Hypertens* 2003; **21**:2265–2273.

26 American College of Sports Medicine. *ACSM's Guidelines for exercise testing and prescription*, 6th edn. Baltimore, Maryland: Lippincott Williams & Wilkins; 2000.

27 Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotechen TA, *et al*. Primary prevention of hypertension clinical and public health advisory from the National High Blood Pressure Education Program. *JAMA* 2002; **288**:1882–1888.

28 Giri S, Thompson PD, Kiernan FJ, Francis J, Clive J, Fram D, *et al*. Clinical and angiographic characteristics of exertion-related acute myocardial infarction. *JAMA* 1999; **282**:1731–1736.

29 Shaper AG, Wannamethee G, Walker M. Physical activity, hypertension and risk of heart attack in men without evidence of ischaemic heart disease. *J Hum Hypertens* 1994; **8**:3–10.

30 Hallliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc Sports Sci Rev* 2001; **29**:65–70.

31 Brownley K, Hinderliter AL, West SG, Girdler SS, Sherwood A, Light KC. Sympathoadrenergic mechanisms in reduced hemodynamic stress responses after exercise. *Med Sci Sports Exerc* 2003; **6**:978–986.

32 Halliwill JR, Dinenno FA, Dietz NM. Alpha-adrenergic responsiveness during postexercise hypotension in humans. *J Physiol* 2003; **550**: 279–286.