Original Research Article

COMPARATIVE STUDY OF RECOVERY OF HEART RATE AND BLOOD PRESSURE POST EXERCISE IN HEALTHY MALE AND FEMALE ADULTS

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Abstract

Background: It is well-established that a reduction in arterial BP occurs following a single bout of land-based, acute exercise. Acute exercise elicits an increase in HR and arterial systolic blood pressure (SBP). The aim of the study was to compare the responses of women and men during recovery from dynamic exercise.

Material and methods: This observational study included 60 healthy, non-athletic adults of age 18-35 years (30 were males and 30 females). The participants were asked to perform the submaximal exercise on the treadmill test according to the guidelines given before and during the study procedure. The two study patterns of active and inactive recovery modes were performed randomly. Pre and post-exercise BP and Heart rate were measured before, during and post exercise inactive and active recovery after 1, 3 and 5 min by automatic electronic heart rate and BP monitor.

Results: At peak exercise, men showed higher MAP as compared to women participants. There was an immediate decrease in MAP after exercise during inactive recovery modes. In men, MAP returned to pre exercise levels at minutes 2–5 during the inactive recovery, while in women, MAP fell to below pre-exercise levels at 1–5 min of inactive recovery. During exercise, men exhibited lower peak exercise HR compared with women. During active recovery, HR decreased less compared with inactive recovery for both the genders.

Conclusion: This study concludes that women may have increased risk of post exercise orthostatic hypotension, and active recovery from exercise should reduce this risk.

Keywords: Cardiovascular Health, Exercise, Physical Activity, Postexercise Heart Rate.

Introduction

Regular physical exercise has proven to be an effective intervention to reduce cardiovascular risk in both men and women.[1,2] Various exercise modalities have been shown to increase aerobic fitness and induce a number of favorable hemodynamic changes, including the lowering of blood pressure (BP), heart rate (HR), total peripheral resistance (TPR), and regional sympathetic outflow, all of which contribute to decrease in risk of cardiovascular and all-cause mortality.[3] Acute exercise elicits an increase in HR and arterial systolic blood pressure (SBP), yet depending on the nature of the exercise, a post-exercise hypotensive effect has been reported by a number of investigators in both male and female participants.[4,5] This post-exercise hypotension (PEH) is characterized by a sustained reduction in BP below pre- exercise or resting levels. PEH has been observed following various types of aerobic exercises such as walking, running, leg ergometry, and arm ergometry.[6] This BP response is thought to contribute to the mechanism underlying the chronic, long-term beneficial effects of dynamic aerobic exercise which includes improved cardiovascular morbidity and mortality.[7,8]

Decreased responsiveness of cardiovascular mechanisms that normally contribute to regulation of arterial pressure and maintenance of cerebral blood flow increase the risk of syncope after exercise.[9] Several investigations found that women have a lower tolerance to various orthostatic challenges at rest when compared with men.[10,11] Also, some investigations report that women have less responsiveness in mechanisms that regulate arterial pressure as compared to men.[10,12] Frey and Hoffler suggested that men may respond to orthostatic challenges with greater sympathetic stimulation to the peripheral vasculature as compared to women, whereas women respond with greater vagally mediated increase in heart rate (HR) when compared to men.[13] Because women exhibit less orthostatic tolerance than men at rest, women may be more susceptible to post exercise orthostatic hypotension; however, only few studies have investigated the influence of gender on cardiovascular responses during inactive recovery from exercise. Therefore, the purpose of this study was to compare the responses of women and men during recovery from dynamic exercise. We hypothesized that women have greater decrease in arterial pressure during recovery from exercise as compared to men.

Material and Method

This observational study was conducted in a Medical College attached to a Tertiary Care Centre over a period of 2 years after obtaining approval from the institutional ethics committee. The study included total 60 healthy, non-athletic adult participants (30 males and 30 females) who were undergraduate and postgraduate medical students. The whole procedure of the study was explained and written informed consent was obtained from all the participants prior to their enrollment in the study.

Inclusion criteria:

- Male and female adults of age 18 to 35 years.
- Non-smokers, non-alcoholics, non-diabetic, non-asthmatic, non-hypertensive, apparently healthy and free of cardiovascular diseases and not

taking medications that could affect cardiovascular functions, such as heart rate or any other hormonal problems etc.

Exclusion criteria:

- Age below 18 years and above 35 years.
- Any physical deformity or inability to stand erect.
- Non menstruating at that time of test, in female participants.
- Those subjects who could not complete the exercise protocol and the exercise test has been stopped before the endpoint is reached with common complaints of leg fatigue, exhaustion, breathlessness and dizziness, irrespective of age.

The participants were randomly selected based on the results of structural health, life style screening and medical history questionnaire. Participants were instructed not to consume beverages containing alcohol or coffee and not to eat a heavy meal or participate in any vigorous activity 24 hours before the test. The study was carried out on electronic treadmill machine in well ventilated room at normal room temperature of 26-27 °C. Participants were requested to come at study place between 7-11 AM on two separate days to study the recovery of heart rate and BP, during active and inactive post exercise recovery period.

Bruce protocol was employed for performing the Trademill exercise[14] and the participants were asked to perform the submaximal exercise on the treadmill. The two study patterns – active and inactive recovery mode were performed randomly as follows:

- *Active recovery:* The study participants walked on treadmill with a normal walking speed after exercise.
- *Inactive recovery:* The study participants stopped treadmill exercise and sat completely still on a chair.

Blood Pressure and Heart Rate Measurements - Before exercise test, age predicted maximum heart rate was determined for each participants by the following equation:[15]

HR max = 208 – (age x 0.7)

During exercise test, BP and HR were measured using automated BP device, at peak exercise and 1min, 3min and 5min post exercise recovery period for both active and inactive recovery modes. Each study participant performed symptom limited treadmill exercise test of intensity targeted at 70% of their age predicted maximal heart rate. The peak exercise BP and HR were defined as final values measured at the termination of exercise. From measured BP, Mean Arterial Pressure (MAP) was calculated for pre, peak and post exercise at 1, 3 and 5 min by following equation and used to compare BP response during and post exercise:

$$MAP = DBP + 1/3 (SBP – DBP)$$

Results

In the present study, the mean age of the male participants was 24±4 years and female participant was 23±3 years. There was no significant difference in the mean age, resting HR and DBP, whereas there was significant difference in the mean height, weight, and SBP between women and men. (Table 1)

Table 1: Descriptive Data Of Study Participants

VARIABLE	MEN (n=30)	WOMEN (n=30)	P VALUE
Age (years)	24 ± 4	23 ± 3	0.191
Height (cm)	169 ± 3	158 ± 5	0.0001*
Weight (kg)	63 ± 7	54 ± 4	0.0001*
SBP (mmHg)	118 ± 4	111 ± 4	0.0001*
DBP (mmHg)	70 ±5	68 ± 6	0.076
HR (bpm)	72 ± 5	73 ± 4	0.064

*Significant P value< 0.05

At baseline, there was no difference in the mean arterial pressure (MAP) between women and men, but at peak exercise, male participants showed higher MAP compared to female participants. All participants demonstrated an immediate decrease in MAP after exercise during inactive recovery modes. When measured 1 min after exercise, MAP decreased less during inactive recovery in men (9 ± 2 mmHg) as compared to women (15 ± 1mmHg). In men, MAP returned to pre exercise levels at minutes 2–5 during the inactive recovery, while in women, MAP fell to below pre exercise levels at 1–5 min of inactive recovery. Women and men both demonstrated ~5 mmHg less of a decrease in MAP during the active recovery. (Table 2)

Table 2: Mean Arterial Pressure (MAP) during active and inactive recovery after dynamic exercise

MAP		Baseline	Peak exercise	1 min	3min	5min
Inactive Recover ygroup	Male	85 ±4	101± 4	90± 5	89± 5	83± 4
	Female	82± 4	96 ±4*	81± 4*	79 ±5*	76± 4*
Active Recover ygroup	Male	86± 5	102± 6	94±6	91±5	87±5
	Female	82± 4	96± 4*	88± 3	86± 4	85± 5

*Significant P value< 0.05

During exercise, men exhibited lower peak exercise HR when compared with women (P<0.05). Both men and women demonstrated similar decrease in HR during 5 min of inactive recovery from exercise. No gender difference in HR existed during 5 min of active recovery. As expected, during active recovery, HR decreased less compared with inactive recovery in both the genders. (Table 3)

Table 3: Heart Rate (HR) during active and inactive recovery afterdynamic exercise

HR		Baseline	Peak exercise	1 min	3min	5min
Inactive Recoverygroup	Male	73± 5	144± 7*	95± 6	82± 4	74± 4
	Female	74± 6	153± 7	98 ±6	80± 5	74± 5
Active Recoverygroup	Male	72 ±5	144± 7	103± 6	88± 6	77± 6
	Female	74± 5	152± 6	105± 7	94± 6	78 ±4

*Significant P value< 0.05

Discussion

The results of this study supports the hypothesis that the reduction of MAP during
recovery from dynamic exercise is greater in women as compared to men. The reason behind this is that women has relatively greater reductions in cardiac output and less of an increase in TPR after exercise. During active recovery from exercise, skeletal muscle pumping was effective in the maintenance of

MAP in both women and men. The factors that determine MAP includes stroke volume (SV), HR and TPR. Adjustments in these variables solely or collectively can explain the gender difference. Other investigations have demonstrated that orthostatic stress was associated with a greater decline in cardiac output and stroke volume[10] and greater vasoconstriction in women.[16,17]

Age group of participants in this study was 18-35 years. As there was no significant difference between male and females, they both showed PEH in inactive recovery but females showed greater decrease in magnitude. Age has been shown to affect the degree of PEH observed, with a greater magnitude in older (40-60 years old) compared with younger (20 to 39 years old) individuals.[18] However, this does not negate the fact that PEH appears to occur independent of age, with this phenomenon having been observed in young (aged 19-35),[19,20] middle aged (aged 35-60)[19] and older individuals.[21]

Forjaz et al.[22] reported that PEH is greater in subjects with a higher initial resting BP and those studies that have observed a decline in BP following exercise substantiate this finding, the average magnitude of PEH being more pronounced in pre-hypertensive (SBP/DBP ~14/9 mmHg) and hypertensive (SBP/DBP ~10/7 mmHg) compared with that of normotensive individuals (SBP/DBP ~8/9 mmHg).[65] In this study, the participants were normotensive.

The mean resting BP of male and female participants were 118/70 and 111/68 mmHg respectively. The average decrease in magnitude in PEH was 11/2 mmHg which is in accordance with the above mentioned study but the diastolic BP decreased very less as compared to systolic BP. Therefore mean arterial pressure was measured. It has been speculated that normal functional cardiovascular compensatory mechanisms that are activated in normotensive subjects but diminished in older, hypertensives, such as the baroreflex, may be responsible for the observed differential PEH response. However, Pescatello and Taylor,[23] have suggested that the correlation between pre-exercise BP status and the magnitude of the PEH response can be spurious because of mathematical coupling and regression-to-the-mean statistical artefacts.

In examining the effects of exercise intensity on PEH, the majority of studies have utilized protocols consisting of submaximal cycle ergometry or treadmill exercise at intensities ranging from 40-100% of maximal exercise capacity using end points including VO2max, predicted maximal heart rate, or HRR.[6,23,24] Direct comparisons of the effect of exercise intensity on PEH have mostly suggested that the degree of PEH is independent of exercise intensity. MacDonald, MacDougall, & Hogben[25] found no difference in the magnitude of

PEH (~8/5 mmHg) following 30 minutes of cycle ergometry at intensities of 50% and 75% of VO2max in young normotensives. Similar observations were made by Forjaz et al[22] and Pescatello et al[23] who found no significant difference in the magnitude of PEH in younger (20-30 years) and older individuals (35-45 years) respectively in their studies.

Conversely, another study observed larger magnitudes of PEH following high-intensity exercise (5 minute stages of 25 Watt increments) when compared to moderate- (5 minute stages of 12.5 Watt increments) and low- (constant 50 Watt) intensity exercise in a normotensive population. [24] This larger magnitude of PEH in young and older individuals following higher intensity exercise (70-80% VO2max) has also been observed by other investigators.[26]

Conflicting recently, evidence suggested that total work done, rather than intensity or duration of exercise alone, may determine the magnitude of PEH. In the study conducted by Jones et al,[26] similar magnitudes of PEH (-5 ± 3 mmHg versus -1 ± 7mmHg) were observed when participants exercised at 70% VO2 peak and at 40% VO2 peak with duration adjusted to correspond to the same total work. However, an increase in SBP (5 ± 6 mmHg) was observed when subjects were prescribed a low exercise dose (intensity x duration; 30 minutes at 40% VO2peak).

Therefore, a greater exercise dose can possibly increase the magnitude of the observed PEH. However, the results of this study showed that an increase in total work done does not increase the magnitude of PEH in prolonged exercise (150 minutes).[27] By extending the exercise dose and PEH response curve using two intensities (55 ± 5% and 75 ± 5% VO2max) during prolonged exercise (150 minutes), it was shown that the magnitude of PEH for both exercise intensities was similar, suggesting that the proposed "dose-response" relationship of intensity for PEH is not present following prolonged exercise.

Conclusion

In the present study, at peak exercise, men showed higher MAP as compared to women participants. There was an immediate decrease in MAP after exercise during inactive recovery modes. In men, MAP returned to pre exercise levels at minutes 2–5 during the inactive recovery, while in women, MAP fell to below pre-exercise levels at 1–5 min of inactive recovery.

During exercise, men exhibited lower peak exercise HR compared with women. During active recovery, HR decreased less compared with inactive recovery for both genders. Thus this study concludes that women exhibits significantly greater decrease in MAP than men during recovery from dynamic exercise and that the skeletal muscle pump (active recovery) is important in the maintenance of MAP during recovery from exercise.

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