THE EFFECTS OF STRETCHING EXERCISE ON HEMODYNAMIC RESPONSES AND POST-EXERCISE HYPOTENSION IN NORMOTENSIVE WOMEN STUDENTS

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Annotation. *Aim:* The aim of the present study was to evaluate acute effects of SE on post-exercise hemodynamic responses for 1-h in normotensive sedentary young women. *Methods:* Sixteen women (21.56±1.21yr; 159.6±0.5 cm; 54.53±6.02 kg) were randomly assigned to SE (n = 8) and control (C) groups (n = 8). SE group performed 20 stretches for the whole body. Each SE was repeated 2 times. Rest interval between repetitions and movement 10 s were considered. Systolic blood pressure (SBP), diastolic BP (DBP), mean arterial BP (MAP), rate pressure product (RPP), pulse pressure (PP) and heart rate (HR) were measured during 1-h (minutes: 0,15,30,45 and 60) in SE and C groups. *Results:* There were significant decreases ($P<0.05$) in SBP, DBP, and MAP, after SE from 15 to 30 min of recovery than baseline. RPP also decreased significantly ($P<0.05$) after SE from 15 to 45 min when compared with baseline. But SE did not cause any significant changes in PP when compared with C group. Hemodynamic responses were not altered during the control trial. *Conclusions:* The results of this study showed that an acute SE lead to PEH. Therefore, SE may be an interesting training strategy to acutely decrease BP in young women.
Keywords: stretching, blood pressure, hypotension, student, girl.

Introduction

High blood pressure or hypertension (HT) is a risk factor for developing cardiovascular disease (CVD) and according to the World Health Organization (WHO) reports, each year more than 7 billion people in the world will die due to this disease (Melo et al., 2006). According to the American College of Sports Medicine (ACSM), even a modest 3 mmHg drop in systolic blood pressure (SBP) and diastolic blood pressure (DBP) at rest is associated with reduced cardiac mortality by 5 to 9 percent (Pescatello et al., 2004). Although pharmacological interventions are effective for reducing the blood pressure (BP), but the side effects of antihypertensive drugs have been proposed (Chobanian et al., 2003). Therefore, a lifestyle change for instant exercise is very important for the management of BP.

It has been shown that active individual have a lower risk of becoming hypertensive than those that are sedentary (Cardoso et al., 2010). Evidence suggests implementing regular exercise leads to decrease BP and the risk of CVD (Corrick et al., 2013). BP may decrease below pre-exercise baseline levels after different muscular work, this response is known as post-exercise hypotension (PEH) (Halliwill et al., 2013). This phenomenon could be clinically significance, because it tend to maintain BP of hypertensive subjects transiently at lower levels during different times of the day, especially when BP is at its highest level (Moraes et al., 2011). PEH has been investigated with various exercise including aerobic exercise (Mohebbi et al., 2010), resistance exercise (Rezk et al., 2006), water exercise (Terblanche et al., 2012), and concurrent exercise (Keese et al., 2012). The mechanisms that cause PEH are complex and multifactorial. It may be mediated by decrease in sympathetic nervous system, cardiac output, and vascular resistance or changes in the release of vasodilator substances (MacDonald., 2002). The decrease in total peripheral resistance induced by exercise usually results from increased bioavailability of nitric oxide (NO, a potent vasodilator) produced by endothelial cells in response to autonomic, hemodynamic and humoral stimuli (Santana et al., 2013).

Special populations such as the elderly and obese may have physical or musculoskeletal limitations and some individual due to inaccessibility gymnasium and high costs, may not have the ability to participation in conventional exercise modalities. Stretching exercise (SE) is an important component of fitness training (Farinatti et al., 2011) and it used in exercise programs for muscle injury prevention (Cipriani et al., 2012). Mechanical stress induced by SE can affect hemodynamic responses (Farinatti et al., 2011). Although it is clear that SE enhance flexibility (Cipriani et al., 2012), but its effect on hemodynamic responses and PEH is unclear. Recently, vong et al. observed that eight weeks of SE resulted to decrease in BP in obese postmenopausal women (vong et al., 2013). A previous study has reported that stretch stimulus to rat's pulmonary vascular endothelial cells leads to an increase release of NO (Kuebler eta al., 2003). Hotta et al. also demonstrated that acute SE improves peripheral circulated by production of NO (Hotta et al., 2013). Given the role of SE in the production of NO (Hotta et al., 2013), this model of physical activity may lead to PEH. It has been shown that stretched muscle fibers activate mechanoreceptors, which elicit cardiovascular regulation through parasympathetic inhibition and sympathetic activation (Drew et al., 2008). The fall in heart rate (HR) immediately post-exercise is a sign of parasympathetic nervous activity (Cole et al., 1999). Because of the increased vagal activity is associated with a reduced risk of mortality (Cole et al., 1999), we assessed changes in HR during post-exercise recovery. Rate Pressure Product (RPP, index of myocardial oxygen consumption) or Robinson index is the product of HR and SBP (Nagpal et al., 2007). Decrease in RPP post-exercise, indicating improvement in cardiac function (Piotrowicz et al., 2009). Mean arterial pressure (MAP) is a product of cardiac output and systematic vascular resistance (MacDonald., 2002). Given that PEH May be result from reductions in cardiac output, total peripheral resistance, or both (Santana et al., 2013). Therefore, the calculation of this parameter can help us when discussing the results. Pulse pressure (PP) defined as the difference between SBP and DBP, is an indicator of arterial compliance and used in

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assessing the risk of heart disease (Franklin et al., 1999). Among the indicators of cardiovascular risk assessment, most emphasis is on the PP (Franklin et al., 1999). Thus, evaluate the changes PP post-exercise may be useful. Therefore, because information on the effect of SE on the PEH seems to be lacking, the purpose of these study was to examine the effect of SE on PEH and post-exercise hemodynamic responses in normotensive sedentary young women.

Material and Methods

Subjects

Sixteen women volunteered for this study. All subjects completed the short version of the International Physical Activity Questionnaire (APAQ) and were considered sedentary. Subjects were randomly assigned to SE (n = 8) and control groups (C, n = 8). The inclusion criteria were: normotensive, weight normal, no regular exercise training in the previous 12 months. The exclusion criteria were: use of drugs that could affect cardiovascular function, (i.e. beta-blockers and inhibitors of angiotensin-converting enzyme), smoking, have any cardiovascular and osteomyoarticular problems that could affect performance during SE, being in menstrual bleeding cycle, and SBP and DBP greater than 139 and 89 mmHg respectively. Volunteers completed a thorough physical examination, including a medical history, BP assessment, anthropometric, and orthopedic evaluation prior to participation in the experimental sessions. Moreover, all volunteers signed a written consent and were informed about the risks and benefits of the present study which was approved by the ethical Committee of the Department of Sport Sciences, University of Guilan. Demographic characteristics of subjects are shown in Table 1.

Anthropometric, body fat percentage, and hemodynamic parameters

Height was measured using a wall-stadiometer, by precision of 1 mm. weight and body composition was determined by In Body System (0.3, Korea). SBP and DBP using a standard mercury sphygmomanometer (ALPK, Japan) and HR were measured by an electronic HR monitor (Polar PM80, Finland). The measuring procedure was in accordance with the recommendation of the American Heart Association (Fletcher et al., 1995) and was conducted by an experienced researcher. RPP, MAP and PP were estimated by conventional equations.

Acute exercise session

SE group completed familiarization program prior to testing. During the familiarization subjects were advised regarding proper performance of stretches. Experimental session was conducted in a temperature-controlled room (23 ± 1°C) between 9:00-11:00 am. Subjects refrained for at least 48-h from intake of caffeine or alcoholic beverage and severe activity that demanded high energy before testing. Before the exercise protocol, in order to measure baseline BP and HR, Subjects seated for 10 minutes and then 5 recordings were taken at 2 minute-intervals. The lowest and highest BP recorded values were removed and the remaining three were averaged to determine of baseline BP. At the end of 10 minutes, HR was measured only once. Immediately after the SE, BP and HR were measured at 0, 15, 30, 45, and 60 minutes with subjects seated. Same parameters were measured for C group without performing any type of exercise. Procedure of SE: researcher closely supervised the SE, which were conducted on a floor mat and focused on whole body major muscles: pectorals major and minor, latissimus dorsi, bicep brachial, triceps, deltoid, trapezius, illiopsas, gluteus, quadriceps, hamstring, leg adductors and gastrocnemius. The 20 stretches were performed in the standing, sitting, and lying position. Each SE was repeated 2 times. The stretched muscle was held for 30 s at point of maximal exertion (defined as RPE > 18). Rest interval between repetitions and movement 10 s were considered. Subjects counted from 1 to 30 during the 30 s stretch to avoid holding their breath during the stretching. Total time SE lasted approximately 38 minute.

Statistical Analysis

All data are presented as mean ± standard deviation (SD). Independent t-tests was used as appropriate and repeated measures analysis of variance was used to determine the degree of difference SBP, DBP, MAP, HR, RPP and PP between baseline values and at 0, 15, 30, 45, and 60 minutes in C and SE groups. Statistical significance was accepted at the $P<0.05$.

Results

In C group, SBP, DBP, HR, MAP and PP, did not changed significantly. Figure 1 presents mean values of SBP, DBP and MAP during 1-h after the experimental session. The SBP, DBP and MAP was significantly decreased ($P<0.05$) at time points of 15 to 30 after SE when compared with the baseline values. Table 1 shows the changes in HR, RPP, and PP from baseline values to 1-h after experimental sessions. HR was significantly increased immediately after SE when compared with baseline value. However, HR at time points of 15 to 6o was significantly reduced. RPP decreased significantly at time points of 15 to 45 when compared with baseline value. But, SE did not any significant changes in PP.

Table 1

Demographic characteristic of subjects

Parameter	Exercise	Control
Age, yr	21.87±1.25	21.25±1.64
Height, cm	159.1±0.06	158.2±0.05
Weight, kg	55.86±4.40	53.20±7.37
BMI, kg/m^2	22.10±1.94	21.17±2.06
Fat, %	28.19±6.31	32.09±5.84

Values are shown as mean ± SD or numbers (%). BMI = body mass index

Table 2

HR, RPP and PP variability parameters in the C and SE groups

	Trials	HR	PP	RPP
Exercise	Pre-exercise	80±5.24	50.75±4.77	10105±536.4
	Post-exercise	86±6.41 *#	49.75±6.45	10490±368.6 #
	15 min	77±5.13 ¥	48.87±7.30	9222.5±484.2 *¥#
	30 min	75±5.13 ¥	50.25±4.86	9017.5±811.7 *¥#
	45 min	73.5±5.21 ¥#	49.12±5.44	8831±590.1 *¥#
	60 min	76.5±5.42 ¥€	51.12±4.32	9528±729.5 €
Control	Pre-exercise	84±8	47.25±8.41	10024±1027.5
	Post-exercise	84.2±7.9	47.23±8.44	10490±368.6
	15 min	83±5.13	47.25±7.62	9222±484.2
	30 min	82±6.05	46.25±9.76	9017±811.6
	45 min	82.5±6.02	46±8.34	9802±736.3
	60 min	81.5±6.02	46±8.41	9528±729.5

Values are shown as mean ± SD or numbers (%). HR = heart rate, PP = pulse pressure, RPP = rate pressure product
* Significant difference from pre-exercise (P<0.05)
€Significant difference from 45 min (P<0.05)
¥Significant difference from post-exercise (P<0.05)
Significant difference between the two group (P<0.05)

Discussion

The results indicate that SE induced a drop in BP and RPP when compared with the control session. The absence of any decrease in BP during the non-exercise control trial shows that, decreasing the BP levels after SE are due to the exercise effect and not to the normal diurnal BP variations. It was research has demonstrated that acute exercise (aerobic, resistance, water, and concurrent) induce PEH (Mohebbi et al. 2010; Rezk et al. 2006; Terblanche et al. 2012; Keese et al. 2012). But the effects of SE have not been investigated. The novelty of this study was the acute of SE lead to PEH. To the best of knowledge, this is the first report of the effects of stretching exercise on PEH. The mechanisms underlying the PEH are not fully understood. Changes in the neural control of the circulatory system and the release of vasodilator agents are may be related with the PEH (Lockwood et al., 2005). Lansman et al. showed that a single stretch stimulus to vascular endothelial cells instantly enhanced the opening frequency of Ca^{2+} channels in the cell membrane (Lansman et al., 1987). SE could increase Ca^{2+} inflow into vascular endothelial cells by stretch activated Ca^{2+} channels (Singer et al., 1982). This increase leads to augmented calcium-dependent NO production from vascular endothelium (Singer et al., 1982). In the present study, we have observed PEH in SBP, DBP and MAP. Therefore, the PEH induced by SE may be due to NO production from vascular endothelial cells. It has been shown that, when muscle stretching and contraction occur simultaneously (which is property of the static stretching method, due to the muscle spindle reflex), type III fibers and metaboreceptor activated, and may induce parasympathetic inhibition and baroreflex stimulation and contribute to an increase in the hemodynamic responses (Farinatti et al., 2013). It is accepted that during SE there is a parasympathetic withdrawal and sympathetic stimulation, resulting in increases of the HR (Goldberger et al., 2006). These effects are reversed during the post-exercise recovery (Goldberger et al., 2006). Our study also revealed that HR was significantly increased immediately after SE and then gradually returns to the baseline value. Although SE did not lead to significantly decrease in HR below baseline level during the recovery period, but RPP for 30 minutes was lower compared with baseline value. Also, the RPP is the product of HR and SBP, any changes of these parameters can affect the RPP value. However, reducing the RPP may be more related with reduction in SBP. The present study does not provide explanations about the mechanism by which SE simultaneously decreases post-exercise BP and RPP in normotensive young women. One possible mechanism underlying the effects of SE on BP and RPP may be related to the decrease in sympathetic nervous activity. Pagani et al. found a significant decrease in low-frequency component SBP (a marker of vascular sympathetic activity) after stretching training (Pagani et al., 1997). Another possible mechanism is an improved endothelial-mediate vasodilation. A recent study showed that a 15 min SE acutely improved NO-mediated vasodilation (Hotta et al., 2013). Nevertheless, it is possible that SE via releasing of NO and decrease in sympathetic nervous activity contributed to PEH and fall in RPP. The study has limitations that should be considered. The small sample size that was utilized and the voluntary in this study were normotensive women, limiting the extrapolation of these to individual with other characteristics. Moreover, PEH mechanisms were not investigated. Additionally, blood sample measures should be considered for future researches, including the analysis of vasodilator metabolites, particularly NO. The assessment of sympathetic nervous activity following SE.

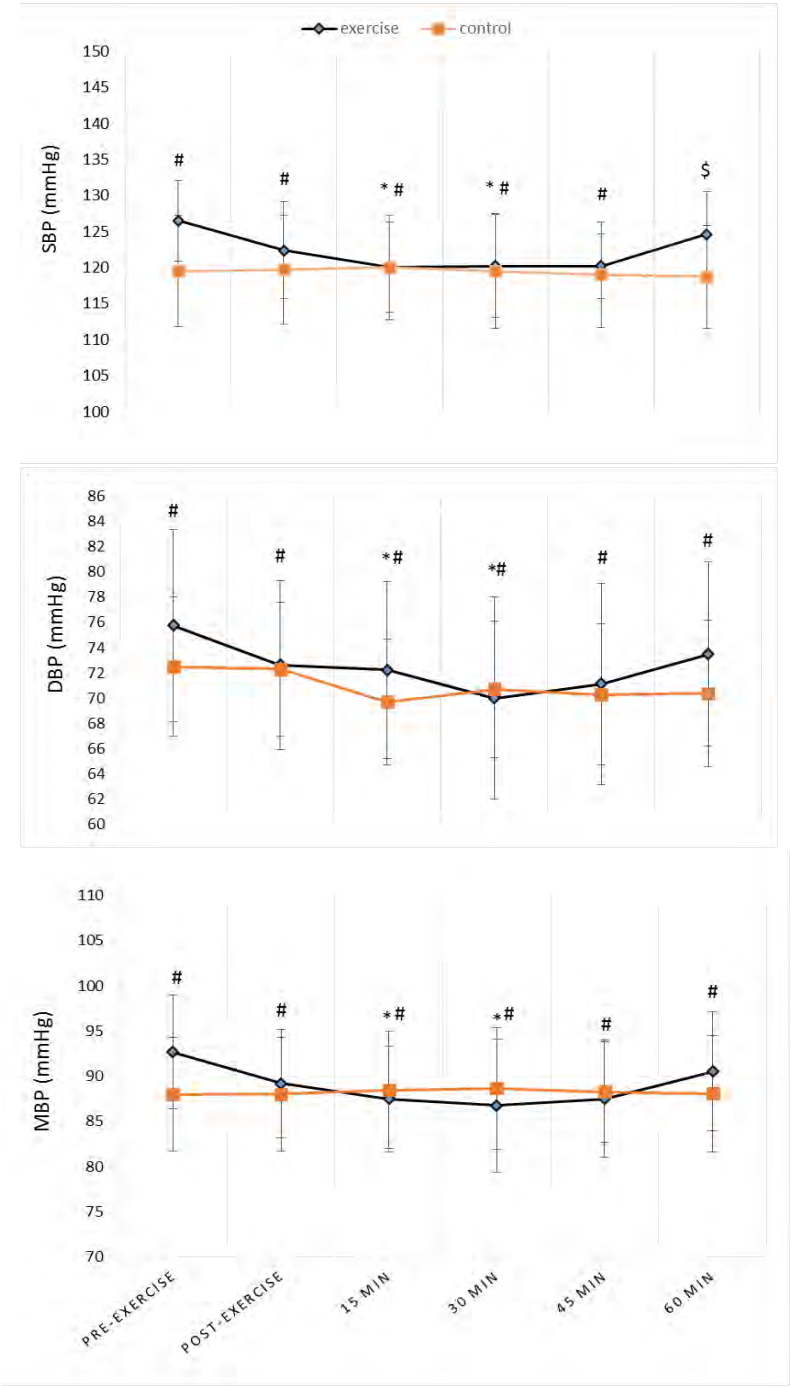

Fig. 1. SBP, DBP, MBP changes following exercise
* Significant difference from pre-exercise (P<0.05)
$ Significant difference from 15 min (P<0.05)
# Significant difference between the two group (P<0.05)

Conclusions

In normotensive women, SE decreases BP and RPP during the post-exercise period. This decrease persist for 30 min. These findings, if reproducible in hypertensive subjects, may have clinical implications.

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