International Journal of Anatomy and Physiology ISSN: 2326-7275 Vol. 8 (11), pp. 001-005, November, 2019.
Available online at www.internationalscholarsjournals.org © International Scholars Journals

Full Length Research Paper

The effects of carotid baroreceptor stimulation on blood pressure variability in young male athletes and non-athletes

Ali Arasteh[1]*, Maliheh Hamzehzadeh Azar[2], Saeed Khamnei[3] and Yousef Mehmannavaz[4]

[1]Department of Physiology, Maragheh Branch, Islamic Azad University, Maragheh, Iran.
[2]Department of Nursing, Maragheh Branch, Islamic Azad University, Maragheh, Iran.
[3]Department of Physiology, Tabriz University of Medical Sciences, Tabriz, Iran.
[4]Department of Animal Science, Maragheh Branch, Islamic Azad University, Maragheh, Iran.

Accepted 15 May, 2019

The purpose of this study was to investigate the effects of carotid baroreceptor (BR) stimulation on blood pressure changes in young male volleyball players and non-athletes. Twenty five young male volleyball players and twenty-five young male non-athletes were recruited for voluntary participation in the present study. A neck suction device (Ekberg's model) was used for stimulation of carotid baroreceptor. In this study the duration of stimulation was 70 s, and the negative pressure used was -30 mm Hg. Blood pressures were recorded before, during and after ending of stimulation of carotid baroreceptors in sitting position. Indirect pressure assessment was utilized for data acquisition. According to the results of this study, systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse blood pressure (PBP) significantly decreased compared to baseline levels in two groups (P<0.001). This research showed that the maximum reduction in SBP and PBP occurred in the 25th second, while for DBP the maximum reduction time was seen in the 5th second during stimulation in two groups. Also, maximum reduction in mean arterial blood pressure (MABP) in the group of athlete occurred in the 5th second, while in the group of non-athlete, it occurred in the 25th second .Our data showed a more reduction in SBP as compared with DBP, which was more evident in control group, although was not statistically significant. It is concluded that in athletes carotid baroreflex becomes more efficient due to long term physiological adaptations of exercise.

Key words: Blood pressure, carotid baroreceptor, male athletes.

INTRODUCTION

It is known that the arterial BR play an important role in maintaining arterial pressure because a large fall in arterial pressure occurs with exercise in experimental animals whose arterial BR have been denervated (Melcher and Donald, 1981). It is well known that exercise can lower arterial pressure and restore or partially restore baroreflex control of heart rate and blood pressure to more normal values (Timmers et al., 2004). Another study reported that activation of the carotid sinus stimulator before or during exercise caused small reductions in heart rate and large reductions in mean arterial pressure (Joyner, 2006). On the other hand, it is reported that heavy endurance training and overtraining did not change baroreflex sensitivity or blood pressure variability during supine rest in female athletes (Uusitalo et al., 1998). Recent evidence suggests that increases in physical activity produce beneficial effects on the cardiovascular system in normal and diseased individuals through reductions in blood pressure and sympathetic outflow in humans (Cornelissen and Fagard, 2005). In addition, animal studies have shown that BR sensitivity is an independent variable that can influence organ damage in hypertension

*Corresponding author. E-mail: arasteh_cns@yahoo.com.

Abbreviations: SBP, systolic blood pressure; **DBP,** diastolic blood pressure; **PBP,** pulse blood pressure; **MABP,** mean arterial blood pressure; **HR,** heart rate; **BR,** baroreceptors.

(Shan et al., 1999). Previous studies have shown that baroreflex sensitivity increased (McDonald et al., 1993), decreased (Hyek et al., 1995) or be unchanged (Sheldahl et al., 1994) with endurance training in athletes. Studies have demonstrated that regular aerobic exercise lowers blood pressure in patients with essential hypertension (Hagberg et al., 2000). In addition, another study has shown that regular exercise decreased blood pressure, the systolic blood pressure variability and in total peripheral vascular resistance in hypertensive subjects (Izdebska et al., 2004).

According to the data collected by the scientists, it was determined that exercise training may alter central neural regulation of blood pressure through the plasticity of GABAergic systems in the hypothalamus (Kramer et al., 2002). It has been reported that application of suction clearly evokes reflex bradycardia and a fall in blood pressure during supine exercise in humans (Bevegard and Shephered, 1966). In addition, it is also stated that at rest roughly one-third of the changes in arterial pressure during baroreflex stimulation were due to changes in heart rate/cardiac output and two-thirds were dependent on alterations in vascular resistance (Ogoh et al., 2003). However, less information is available about the effects of regular exercise and carotid stimulation of BR on blood pressure variability in male athletes compared with non-athletes. The purpose of this study was to determine the effects of regular exercise in male volleyball players on BR function and blood pressure changes with stimulation of carotid BR and compared with male non-athletes.

METHODS

Subjects

Twenty-five healthy male athletes (athlete group) and twenty-five healthy male non-athletes (control group) were recruited for voluntary participation in the present study. Demographic data for the subjects are presented in Table 1. Athletes were recruited from volleyball team and control subjects were recruited from the students of the University of Medical Sciences in Tabriz. Athlete group had been practicing this sport activity for more than 5 years. Control group did not practice any physical training activity. Athlete and control groups were healthy and free of any known cardiac abnormalities, and none of them were on any cardioactive medications at the time of study. Prior written, informed consent was obtained from the all subjects.

Neck suction method

Stimulation of the carotid BR was produced with a method similar to that used by Eckberg et al. (1975). Briefly, the neck chamber comprised an elliptical piece of sheet lead rimmed with sponge rubber. A pressure transducer and a pneumatic valve were mounted directly upon the chamber. A commercial vacuum cleaner was used to provide a continuous vacuum source whose intensity was controlled with a rheostat. Neck suction was initiated electronically by rotation of a solenoid valve. By application of negative pressure in front of neck, it stretches the soft cervical tissues including carotid sinus. This method is better than the pharmacological one since it is non aggressive and exerts a rapid stimulus directly on the neck. In this way, we avoid the stimulation of other regulatory systems (Dwain and Eckberg, 1977).

Procedure

All tests were carried out in the morning in a temperature controlled laboratory (22 to 24°C). We tried to adapt the subjects to the experimental procedure. Therefore in many cases the recordings were repeated on several occasions in order to avoid emotional tensional which greatly influences baroreflex sensitivity. Indirect pressure assessment was utilized for data acquisition. Blood pressure was recorded by mercury sphygmomanometer. We recorded SBP and DBP from subjects in two groups three times, according to the study protocol: 1) before of stimulation of BR by neck suction for 5 s periods for control levels, 2) during stimulation of BR by neck suction with -30 mmHg negative pressure for 70 s periods for studying effects of carotid BR stimulation on blood pressure variability and 3) after ending of the 70 s period of stimulation of BR. Resting measurements were performed in comfortable sitting position in athlete and control groups. PBP was calculated form difference between SBP with DBP. MABP was calculated as DBP plus one-third of PBP.

Statistical analysis

One-way analysis of variance (ANOVA) and Tukey tests were used to compare the effects of carotid BR stimulation on change of blood pressure in athlete and control groups statistically using the statistical Analysis System (SAS, 2002) software. P-values less than 0.05 were considered to be statistically significant.

RESULTS

SBP and DBP

The baseline characteristics for the subjects participating in this study has been summarized in Table 1. Table 2 shows changes of SBP and DBP levels responses to stimulation of BR by neck suction for 70 s in athlete and control groups. SBP and DBP significantly decreased compared to the Baseline values (control levels) ($P<0.001$) in two groups. Our data showed that during continuation of stimulation, the peak decrease in SBP in response to neck suction occurred in the 25th second, while for DBP the maximum reduction time was in the 5th second of stimulation in two groups. After ending of stimulation, SBP and DBP gradually returned towards control levels. There was no significant difference in SBP and DBP after ending of stimulation compared to base-line values. In addition the finding of the present study showed a more reduction in SBP as compared with DBP, which was more evident in non-athletes, although was not statistically significant.

PBP and MABP

The changes of PBP and MABP responses to 70 s of stimulation of BR are presented in Table 3. The results

Table 1. Characteristics of Athlete and control groups.

Variable	Athletes (n = 25)	Control (n = 25)
Age (year)	21.6± 1.2	20.45± 2.3
Height (cm)	163.45±1.6	162.46 ±1.4
Weight (kg)	56.35± 2.1	57.35± o.7
Systolic blood pressure (mmHg)	120.75± 13.89	118 ± 10.80
Diastolic blood pressure (mmHg)	74.75± 8.503	73.25± 8.1

Data are presented as mean ±SD.

Table 2. Comparison of systolic- and diastolic blood pressure variability at during and after ending of stimulation of carotid baroreceptors with baseline values in athlete and control groups.

		Pre-stimulation (baseline)	During of stimulation			After ending of stimulation
			5th second	25th second	50th second	
SBP in athletes group (n= 25)	Mean±SD	120.750±13.886	112±14.179	108.75±14.76	115±14.69	120.90±13.64
	P		P<0.001	P<0.001	P<0.001	NS
SBP in control group (n= 25)	Mean±SD	118±10.80	107.80±11.423	103.5±11.932	112.5±10.699	120±10.38
	P		P<0.001	P<0.001	P<0.001	NS
DBP in athletes group (n=25)	Mean±SD	74.75±8.503	68.85±8.468	70.6±8.999	71.35±9.326	74.15±9.588
	P		P<0.001	P<0.001	P<0.001	NS
DBP in control group (n=25)	Mean±SD	73.25±8.156	67.6±8.852	69.6±8.081	71.55±8.37	71.55±17.169
	P		P<0.001	P<0.001	P<0.001	NS

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, NS: not significant.

Table 3. Comparison of pulse and mean arterial blood pressure (mmHg) variability during and after ending of stimulation of carotid baroreceiptors with baseline values in athlete and control groups.

	Pre-stimulation (baseline)	During stimulation			After ending of stimulation
		5th second	25th second	50th second	
Mean±SD	43.5± 11.367	41.65±13.148	36.15±12.721	41.4± 13.248	44.35± 11.5
P		P<0.001	P<0.001	P<0.01	NS
Mean±SD	44.75±7.691	40.90± 8.87	33.9± 9.999	40.95± 8.179	45.2± 8.92
P		P<0.001	P<0.001	P<0.001	P<0.001
Mean±SD	91.74± 8.958	84.562± 8.68	84.81± 8.74	85.722± 14.2	91.58± 9.06
P		P<0.001	P<0.001	P<0.001	NS
Mean±SD	88.172± 8.374	80.58± 9.377	79.146±10.13	81.274±11.429	89.84± 7.45
P		P<0.001	P<0.001	P<0.001	NS

PBP: Pulse blood pressure, MABP: mean arterial blood pressure, NS: not significant.

showed that PBP was significantly decreased in athlete and control groups during the period of stimulation of BR compared to baseline levels (P<0.001). The most reduction was seen in the 25th second of stimulation period and gradually retuned toward baseline levels. Our results showed that the decrease of MABP pressure was statistically significant during the period of stimulation of BR compared to the baseline levels in athlete and control groups (P<0.001).The lower value of MABP noticed in the 5th second in athlete group, while in control group it

occurred in the 25th second (Table 2). Our results showed that the level of MABP gradually returned toward baseline levels.

DISCUSSION

The main finding of our study was that stimulation of carotid BR reduced the levels of SBP, DBP, PBP and MABP in both groups. It is important to note that our data showed that as compared with athlete group, control group had 1) a higher percent decrease of blood pressures (SBP, DBP, PBP, MABP) during of stimulation of carotid BR; 2) a more reduction in SBP as compared with DBP; 3) maximum reduction in MABP in the 25th second of stimulation. It is known that the BR reflex constitutes a powerful mechanism of negative retrograde arterial pressure regulation that aims at normalizing its changes. This is achieved directly by a reflex inhibition of sympathetic activity, activation of the parasympathetic system and increase of vascular resistance and HR and indirectly by renin and vasopresin secretion that, in turn, influence arterial pressure regulation (Davos et al., 2002). Studies have shown that exercise training reduces the elevated firing rate of caudal hypothalamic neurons (Beatty et al., 2005). These changes are associated with blood pressure reductions and a restoration of GABAergic transmission in this brain region of the spontaneously hypertensive rats (Kramer et al., 2001) and exercise training also affects measures of nitric oxid synthase activity in the paraventricular nucleus of spontaneously hypertensive rats (DiCarlo et al., 2002). Our findings showed that the greatest changes in blood pressure during stimulation of carotid BR were observed in the group of non-athlete. It seems that response to exercise compromises an increase of cardiac output, vasodilation in the active muscles, vasoconstriction in the vicera, inactive muscles, and the skin, with a reflex increase in the tone of the venous capacity vessels (Poderys et al., 2001). Based on the results presented in this study, it was observed that systolic, diastolic and pulse pressures decreased during period of stimulation in two groups, which was more evident in non-athletes, although was not statistically significant. It was found that after long-term athletic training, left ventricular diastolic cavity dimensions, wall thickness and mass will increase (Fagard et al., 1984). It is demonstrated that endurance training reduces resting and submaximal exercise systolic, diastolic and mean arterial blood pressure (Huston et al., 1985). On the other hand, endurance training has been reported to increase vagal tone and maximal oxygen uptake (Seals and Chase, 1989), which are correlates of baroreflex sensitivity (Sleight et al., 1995). It is well understood that exercise is accompanied by major cardiovascular alterations, including marked tachycardia, increases in cardiac output and in arterial and atrial pressures, and a reduction in total peripheral resistance, it could be expected that a cardiovascular regulating mechanism as important as the arterial BR reflex would play a significant role in mediating and modifying the exercise response (Bernardi et al., 1997). When we looked at the findings of our study and compared changes of blood pressure between athlete and control groups, we found a more reduction in systolic pressure as compared with diastolic pressure, which was more evident in control groups, although was not statistically significant. Therefore improving BR sensitivity, this may result in a more efficient arterial pressure regulation in volleyball players by the baroreflexes. On the other hand we found that short-term training could induce autonomic adaptations, with a reduction in sympathetic activity and an increase in parasympathetic activity and long-term aerobic training, eliciting arterial and ventricular dilation, would induce intrinsic electrophysiological adaptations and enhance parasympathetic activity (Aubert et al., 2003). When we analyzed our data, we observed that maximum reduction in MABP in group of athletes occurred in 5^{th} second, while in group of non-athletes it occurred in 25^{th} second. According to previous studies and our data, some mechanisms may explain the physiological adaptations and increased BR gain sensitivity in young male volleyball players or after exercise training including: increase of intrinsic aortic compliance in rats (Kingwell et al., 1997), arterial compliance in humans (Kingwell et al., 1995), reduction of muscle sympathetic nerve activity (Grassi et al., 1994), increase of the vasodilatory response to acetylcholine in hypertensive rats (Yen et al., 1995) and increase of brachial and femoral artery compliance (Kool et al., 1992).

Our data confirmed previous studies that demonstrated that regular moderate dynamic exercise reduces systolic, diastolic, and mean BP in humans with high BP (Laterza et al., 2007). It seems that the reduction in sympathetic nerve activity, decrease in central angiotensin II concentration, increase in central NO production (Liu et al 2000), resting bradycardia and increase in peak oxygen uptake (Hickson et al., 1977; Katona et al., 1982) are considered important markers of exercise training adaptation in humans. In this regard experimental data have suggested that the baroreflex function may be influenced by a stochastic resonance phenomenon (Hidaka et al., 2000), resetting phenol-menon (Seagard et al., 1992), central influences, humoral factors, cardiopulmonary reflex, chemoreflex, metabo-reflex (Di Rienzo et al., 2008), peripheral somatosensory inputs and activation of skeletal muscle afferent fibres by physical exercise (Potts and Mitchell, 1998). In conclusion, stimulation of carotid BR by neck suction induces a decrease in SBP, DBP, MABP and PBP levels in two groups. Furthermore, our results showed a more reduction in SBP as compared with DBP, which was more evident in non-athletes. Therefore, it seems that in young male volleyball players carotid baroreflex become

more efficient due to long term physiological adaptations of exercise.

REFERENCES

Aubert AE, Seps B, Beckers F (2003). Heart rate variability in athletes. Sports Med., 33: 889-919.

Beatty JA, Kramer JM, Plowey ED, Waldrop TG (2005). Physical exercise decreases neuronal activity in the posterior hypothalamic area of spontaneously hypertensive rats. J. Appl. Physiol., 98: 572-578.

Bernardi L, Passino C, Robergs R (1997). Acute and persistent effects of a 46-kilometer wilderness trail run at altitude: cardiovascular autonomic modulation and baroreflexes. Cardiovas. Res., 34: 273-280.

Bevegard BS, Shephered JT (1966). Circulatory effects of stimulating the carotid arterial stretch receptors in man at rest and during exercise. J. Clin. Invest., 45: 132-142.

Cornelissen VA, Fagard RH (2005). Effects of endurance training on blood pressure, blood pressure-regulating mechanisms, and cardiovascular risk factors. Hypertension, 45: 667-675.

Davos CH, Davies LC, Piepol M (2002). The effect of baroreceptor activity on cardiovascular regulation. Hellenic J. Cardiol., 43: 145-155.

Di Rienzo M, Castiglioni P, Iellamo F, Volterrani M, Pagani M, Mancia G, Karemaker JM, Parati G (2008). Dynamic adaptation of cardiac baroreflex sensitivity to prolonged exposure to microgravity. J. Appl. Physiol., 105: 1569-1575.

Dicarlo SE, Zheng H, Collins HL, Rodenbaugh DW, Patel KP (2002). Daily exercise normalizes the number of diaphorase (NOS) positive neurons in the hypothalamus of hypertensive rats. Brain Res., 955: 153-160.

Dwain L, Eckberg DL (1977). Adaptation of the human carotid baroreceptor-cardiac reflex. Physiology, 269: 579-589.

Eckberg DL, Cavanaugh MS, Mark AL, Abboud FM (1975). A simplified neck suction device for activation of carotid baroreceptors. J. Lab. Clin. Med., 85: 167-173.

Fagard R, Aubert AE, Staessen J (1984). Cardiac structure and function in cyclists and runners: comparative echocardiographic study. Br. Heart. J., 52: 124-129.

Grassi G, Seravalle G, Calhoun DA, Mancia G (1994). Physical training and baroreceptor control of sympathetic nerve activity in humans. Hypertension, 23: 294-301.

Hagberg JM, Park JJ, Brown MD (2000). The role of exercise training in the treatment of hypertension. Sports Med., 30: 193-206.

Hickson RC, Bomze HA, Holloszy JO (1977). Linear increase in aerobic power induced by a strenuous program of endurance exercise. J. Appl. Physiol., 42: 372-376.

Hidaka I, Nozaki D, Yamamoto Y (2000). Functional stochastic resonance in the human brain: noise induced sensitization of baroreflex system. Phys. Rrv. Lett., 85: 3740-3743.

Huston TP, Puffer JC, Rodney WM (1985). The athletic heart syndrome. N. Engl. J. Med., 313: 24-32.

Hyek MF, Szilagyi JE, Tate CA (1995). A chronically instrumented rat model to assess the altered baroreflex due to exercise. Med. Sci. sports Exerc., 27: 1339-1344.

I zdebska E, Cybulska I, Izdebski J, Makowiecka-ciesla M, Trzebski A (2004). Effects of moderate physical training on blood pressurevariability and hemodynamic pattern in mildly hypertensive subjects. J. Physiol Pharmacol., 4: 713-724.

Joyner MJ (2006). Baroreceptor function during exercise: resetting the record. Exp. Physiol., 91: 27-36.

Katona PG, Mclean M, Dighton DH, Guz A (1982). Sympathetic and parasympathetic cardiac control in athletes and nonathletes at rest. J. Appl. Physiol., 52: 1652-1657.

Kingwell BA, Arnold PJ, Jenning GL, Dart AM (1997). Spontaneous running increases aortic compliance in wistar-kyoto rats. Cardiovasc. Res., 35: 132-137.

Kingwell BA, Cameron JD, Gillies KJ, Jennings GL, Dart AM (1995). Arterial compliance may influence baroreflex function athletes and hypertensives. Am. J. Physiol., 265: 411-418.

Kool MJF, Struijker-boudier HA, Wijnen JA, Hoeks A, Bortel LM (1992). Effects of diurnal variability and exercise training on properties of large arteries. J. Hypertens., 10: 49-52.

Kramer JM, Beatty JA, Plowey ED, Waldrop TG (2002). Exercise and hypertension: a model for centeral neural plasticity. Clin. Exp. Pharmacol. Physiol., 29: 122-126.

Kramer JM, Beatty JA, Little HR, Plowey ED, Waldrop TG (2001). Chronic exercise alters caudal hypothalamic regulation of the cardiovascular system in hypertensive rats. Am. J. Physiol. Regul. Integr. Comp. Physiol., 280: 389-397.

Laterza MC, de Matos LD, Trombetta IC, Braga AM, Roveda F, Alves MJ, Krieger EM, Negrao CE, Rondon MU (2007). Exercise training restores baroreflex sensitivity in never-treated hypertensive patients. Hypertension, 49: 1298-306.

Liu JL, Irvine S, Reid IA, Patel KP, Zucker IH (2000). Chronic exercise reduces sympathetic nerve activity in rabbits with pacing-induced heart failure: a role for angiotensin II .Circulation, 102: 1854-1862.

Mcdonald MP, Sanfilippo AJ, Savard GK (1993). Baroreflex function and cardiac structure with moderate endurance training in normotensive men. J. Appl. Physiol., 74: 2469-2477.

Melcher A, Donald DE (1981). Maintained ability of carotid baroreflex to regulate arterial pressure during exercise. Am. J. Physiol., 241: 838-849

Ogoh S, Fadel PJ, Nissen P, Jans O, Selmer C, Secher NH, Raven PB (2003). Baroreflex-mediated changes in cardiac output and vascular conductance in response to alterations in carotid sinus pressure during exercise in humans. J. Physiol., 550: 317-324.

Poderys J, Trinkunas E, Grunovas A (2001). Cardiovascular function and muscle blood flow during incremental stepwise exercise. J. Human. Kinetics, 6: 39-46.

Potts JT, Mitchell JH (1998). Rapid resetting of carotid baroreceptor reflex by afferent input from skeletal muscle receptors. Am .J. Physiol., 275: 2000-2008.

SAS Institute Inc. (2002). SAS/STAT User's Guide: Version 9. 5th Ed. SAS Institute Inc., Cary, North Carolina.

Seagard JL, Gallemberg LA, Hopp FA, Dean C (1992). Acute reseting in two functionally different types of carotid baroreceptors. Circ. Res., 70: 559-565.

Seals DR, Chase PB (1989). Influence of physical training on heart rate variability and baroreflex circulatory control. J. Appl. Physiol., 66: 1886-1895.

Shan ZZ, Dai SM, Su DF (1999). Relationship between baroreceptore reflex function and end-organ damage in spontaneously hypertensive rats. Am. J. Physiol., 277: 200-206.

Sheldahl LM, Ebert TJ, Cox B, Tristani FE (1994). Effect of aerobic training on baroreflex regulation of cardiac and sympathetic function, J. Apple. Physiol., 76: 158-165.

Sleight P, Rovere MT, Mortara A (1995). Physiology andpathophysiology of heart rate and blood pressure variability in humans: is power spectral analysis largely an index of baroreflex gain. Clin. Sci., 88:103-109.

Timmers HJ, Wieling W, Karemaker JM, Lenders JW (2004). Cardiovascular responses to stress after carotid baroreceptor denervation in humans. Ann NY. Acad. Sci., 1018: 515-519.

Uusitalo AL, Uusitalo AJ, Rusko HK (1998). Endurance training, overtraining and baroreflex sensitivity in female athletes. Clin. Physiol., 18: 510-520.

Yen MH, Yang JH, Sheu JR, Lee YM, Ding YA (1995). Chronic exercise enhances endothelium mediated dilation in spontaneously hypertensive rats. Life Sci., 57: 2205-2213.