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Research Article

Males vs Females – Risk of Developing Early Hypertension?

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Abstract: Females are at less risk of coronary heart disease, and of serious arrhythmias as compared to their male counterparts, women lagging behind men in the incidence of sudden death by 20 years. In present study, 20 healthy males and 20 females with family history of hypertension were enrolled and underwent cycle ergometer exercise for 3 min. Their blood pressure was recorded and compared. Result of study revealed that males have higher systolic blood pressure as compared to females, though there was not much difference in diastolic blood pressure.

Keywords: Males, Females, Isotonic exercise, Family history of hypertension, Systolic hypertension, Diastolic hypertension.

INTRODUCTION

Hypertension, often called the silent killer, is a key risk factor for cardiovascular disease mortality and morbidity [1]. Persons with a parental history of hypertension are at a higher risk of development of this disorder [2]. Autonomic abnormality in the form of increased sympathetic tone has been demonstrated in young normotensive offsprings of hypertensive parents [3]. Compared to their male counterparts, women are at a lesser risk of developing coronary heart disease [4,5], and serious arrhythmias [6,7], with women lagging behind men in the incidence of sudden death by 20 years [8]. Only a few studies, with rather conflicting results, have focused on the influence of gender on cardiac autonomic modulation. Both heart rate and blood pressure response to exercise are important determinants of the healthy cardiovascular system. When females and males are exposed to an acute bout of exercise, responses differ between the sexes in terms of strength, cardiovascular, respiratory and metabolic changes. So present study was conducted to know which gender with positive family history for hypertension is prone for developing early hypertension.

MATERIALS AND METHODS

A total of 20 healthy males and 20 healthy females between the age group of 18-30 years, non smokers and with a normal Body Mass Index (BMI), were taken as subjects for the study. Brief history along with general and systemic examination was undertaken to rule out any systemic disease. Brief history of hypertension was taken from the medical prescription of the parents and anti hypertensive drugs prescribed to them.

They were divided into two groups, Group 1 – 20 males with family history of hypertension, Group 2 - 20 females with positive family history of hypertension

Exclusion criteria were any acute illness, smokers, alcoholics, physical disability impairing cycling, involvement in any physical training program, any history of breathlessness or orthopnea, history of osteoarthritis and rheumatoid arthritis or any recent illness during past 2 weeks, any menstrual abnormality, and married females.

Before exercise, subjects were given 10 min to acclimatize during which they were informed about procedure. Blood pressure and Heart rate was recorded at rest. Subjects were instructed to sit on cycle ergometer and start doing cycling at the rate of 60 revolutions per minute and record reading was taken from odometer on cycle. Slowly intensity of cycling was increased. Subjects were informed to continue the exercise up to 3 minutes. Blood pressure was recorded at the end of exercise and then every 5 minutes, for 10 minutes by sphygmomanometer. Systolic blood pressure (SBP) was taken as korotkoff phase 1(appearance of sounds) and diastolic blood pressure (DBP) was taken as korotkoff phase V (disappearance of sound) [9]. The data thus obtained was analyzed using student t test.

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RESULTS

SBP in males was more than females before exercise (p=0.0009, extremely statistically significant), after exercise (p=0.0086, very statistically significant),

5 min after the end of exercise (p=0.0105, statistically significant) and 10 min after end of exercise (p=0.0104, statistically significant) as shown in Table 1.

	Males	Females	Unpaired	p value
	Mean±S.D.	Mean±S.D.	t-value	(two sided)
	(n=20)	(n=20)		
Before exercise	128.89±7.88	112.36±10.23	3.9705	0.0009
				extremely statistically
				significant
After exercise	136.89±4.14	126.00±10.39	2.9464	0.0086
				very statistically
				significant
5 min after end of	134.44±7.26	123.09±9.93	2.8549	0.0105
exercise				statistically significant
10 min after end of	131.56±7.67	120.18±9.69	2.8593	0.0104
exercise				statistically significant

Table 1: Showing Comparison of SBP in males and females

DBP in males was more than females throughout the protocol but the difference was statistically significant after exercise (p=0.0036) and 5 min after the end of exercise (p=0.0484) but not quite statistically significant before exercise (p=0.0671) and 10 min after end of exercise (p=0.0570) as shown in Table 2.

Table 2: §	Showing (Comparison	of DBP in	males and females	
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	Males	Females	Unpaired	p value
	Mean \pm S.D.	Mean \pm S.D.	t-value	(two sided)
	(n=20)	(n=20)		
Before exercise	78.89±7.56	73.64±4.37	1.9485	0.0671
				not quite statistically significant
After exercise	82.22±8.09	75.64±4.54	2.3007	0.0036
				statistically significant
5 min after end of exercise	80.00±6.78	74.91±3.83	2.1177	0.0484
				statistically significant
10 min after end of exercise	79.78±7.10	74.73±3.82	2.0333	0.0570
				not quite statistically significant

DISCUSSION

In this study we compared the effect of exercise on SBP and DBP in both the genders with positive history of hypertension. Males had higher SBP at rest, after exercise 5 min after the end of exercise and 10 min after end of exercise as compare to females. This may be due to hormonal influences or a genetic background. The effects of sex hormones on blood pressure have been widely demonstrated: oestrogens have a favourable effect, possibly as a result of a mechanism involving the renin-angiotensin system and endothelium-derived relaxing factors (i.e. nitric oxide and prostaglandins) [10], the central nervous system [11], or the stimulation of natriuretic peptide production [10]. While testosterone increases blood pressure levels [12]. Furthermore, it is worth noting that (at least in animal models) the Y-chromosome itself is involved in the genesis of hypertension because it enhances sympathetic hyperactivity [13]. These results strongly suggest that the role of gender should be taken into account whenever studies of hypertensive offspring are

being evaluated. Cowan et al carried a study in 1994 to describe the effects of gender and age on heart rate variability (HRV) in healthy volunteers and found that HRV was significantly lower in healthy women compared with healthy men. The results of our study are also similar as females didn't demonstrate a higher BP at rest as well after exercise as compared to males [14]. This exercise-induced rise in diastolic BP in the pre hypertensive stage is similar to that described in high-risk subjects (with high-normal BP and family history of hypertension) [15] and borderline hypertensives [10] and can be explained by increased resting peripheral vascular resistance in the early stages of hypertension[16] and impaired capacity for exercise vasodilatation [15,17-19]. induced Diastolic abnormalities may be responsible for abnormal heart rate control by influencing the afferent pathways of the cardiopulmonary baroreceptors which, in their turn, could affect the variation in heart rate [28] and arterial baroreflex [29].

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