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EFFECT OF AEROBIC EXERCISE ON CARDIOVASCULAR FUNCTIONS AND REACTIVITY IN NORMOTENSIVE OFFSPRINGS OF NORMOTENSIVE AND HYPERTENSIVE PARENTS

Amruta Chauhan

Pioneer Physiotherapy College, Near Ajwa Cross Road, N.H. No.8, Ajwa Nimeta Road, At and Post Sayajipura, Vadodara, Gujarat, India

E-mail of Corresponding Author: amruta1748@gmail.com.

ABSTRACT

Introduction: Hypertension is a major health problem and when untreated, predispose to cardiovascular morbidity and premature death. Cardiovascular reactivity is the pattern of an individual's hemodynamic response to stress On the basis of genetic factor and environmental components, first degree relatives of essential hypertensive patients are known to be at risk of developing hypertension.

Objective: The objective of the study is to compare the effect of aerobic exercise on response of cardiovascular functions and reactivity in normotensive offsprings of normotensive and hypertensive parents.

Methodology: 30 individual with parental history of hypertension and 30 individual without parental history of hypertension were taken. At the beginning, baseline measures included body weights, heights were taken and BMI was calculated. After 5 minutes of rest, blood pressure reading was taken with mercury sphygmomanometer and heart rate reading was taken. Subjects performed aerobic exercise start with warm up in the form of slow walking for 5 minutes. Circuit training was given in the form of upper limb exercise, trunk mobility exercise, treadmill walking for 30 minutes. Subject performed exercise on RPE 6 and as much as can go above it on modified Borg scale with their maximum speed. Post parameter of heart rate and blood pressure was taken at immediately after, after 3 minute. Subjects performed cool down period in the form of slow walking for 5 minutes and again parameters were taken.

Results: The result shows that there is statistically significant difference in the reactivity and cardiovascular functions in normotensive offsprings of hypertensive parents compared to normotensive offsprings of normotensive parents.

Conclusion: The normotensive offsprings of hypertensive parents have increased reactivity and cardiovascular functions with delayed recovery compared to normotensive offsprings of normotensive parents.

Keyword: aerobic exercise, reactivity, offsprings

INTRODUCTION

Hypertension is a major health problem and when untreated, predispose to cardiovascular morbidity and premature death.[1] Seventh report of joint national committee on prevention, detection, evaluation, and treatment of high blood pressure (JNC 7 report) has introduced following classification for adults aged 18 and older [2]

- Normal: <120/<80 mmHg
- Pre hypertension: 120-139/80-89 mmHg

- Stage 1 hypertension: 140-159／ 90-99 mmHg
- Stage 2 hypertension : ≥160 ／ ≥100 mmHg

On the basis of genetic factor and environmental components, first degree relatives of essential hypertensive patients are known to be at risk of developing hypertension.[5] Reactivity is measured as the difference between baseline and initial stress response.[5]

Acute aerobic exercise may be potent stressor to the cardiovascular system. Stress activates the sympathetic nervous system directly causes increase in cardiovascular function. High sympathetic nervous system activity may interact with genetic factor, high sodium intake, obesity and lead to development of hypertension. Evidence supports that increased sympathetic nervous system activity in early hypertension and normotensive offspring of hypertensive parents. Both physical and mental stress causes increase in sympathetic activity.

Normotensive offsprings of hypertensive and normotensive parents have increased cardiovascular and sympathetic nervous system reactivity to stress, but offsprings of hypertensive parents respond grater increased in cardiovascular reactivity. There is controversy about whether normotensive offsprings of hypertensive parents have an increased sympathetic response to stress. Such a response could produce a hyperactive sympathetic reaction, lead to development of hypertension. Both genetic and environmental factor are thought to contribute to this hyper-responsiveness.

There are many studies on effect of mental stress of normotensive offspring of hypertensive parents. Little attention has been given to the importance of altered cardiovascular response in normotensive offsprings of hypertensive parents caused by physical exercise. Aerobic exercise includes arm exercise, trunk exercise and leg exercise so the whole body cardiovascular response can be checked.

So, the need of the study is to compare the effect of aerobic exercise on response of cardiovascular functions and reactivity in normotensive offspring of hypertensive and normotensive parents.

MATERIALS AND METHODOLOGY

Study Design: A cross-sectional comparative study

Source of Data: The study was conducted in Physiotherapy College.

Study Duration: Total duration of study was 5 month

Sample Design: Random sampling

Sample Size: Total 60 subjects were taken:

Group A: 30 subjects with parental history of hypertension

Group B: 30 subjects without parental history of hypertension

Inclusion Criteria:

Age: 20-25 years

Gender: male and female

BMI within normal limits

People willing to participate

GROUP A: Subjects with parental history of hypertension

GROUP B: Subjects without parental history of hypertension

Exclusion Criteria:

Doing regular exercise

Any associated systemic involvement

Any musculoskeletal problem

Any neurological problem

Any respiratory and cardiovascular diseases

Data collection and procedure

Materials Used

Consent form, Data collection sheet, Pen, pencil, paper, Chair, Stop watch, Measure tap

Appartus: Motorized treadmill, Weighing machine, Cuff sphygmomanometer

Outcome Measure: Heart rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP)

Procedure: The entire subjects of this study were evaluated as per assessment format. Those who

fulfilled all inclusion criteria were taken up for the study. The procedure was explained to all subjects. A written consent of all participants was taken.

At the beginning, baseline measures included body weight, height was taken and BMI was calculated. After 5 minutes of rest, blood pressure reading was taken with mercury sphygmomanometer and heart rate reading was taken.

Subjects performed aerobic exercise start with warm up in the form of slow walking for 5minutes.Circuit training was given in the form of upper limb exercise, trunk mobility exercise, treadmill walking for 30 minutes. (photograph 1-3). Subject performed exercise on RPE 6 as much as can go above it on modified Borg scale with their maximum speed. Post parameters of heart rate and blood pressure were taken at immediately after, after 3 minute.

Subjects performed cool down period in the form of slow walking for 5 minutes and again parameters were taken. Statistical tests were used to compare the data of both groups. Level of significance was kept at 5%.

RESULT

The study comprises the two groups with 30 subjects in each. In this study, all the descriptive analysis was done using software graph pad prism - 5. Group A and group B shows no statistical difference in age and body mass index value. (Graph 1-2)

In this study, both group A and B performed aerobic exercise. Reactivity is measured as the difference between baseline and initial stress response of HR, SBP and DBP[5]. The reactivity of HR, SBP, and DBP were taken. For comparison of reactivity between group A and B Mann-Whitney U was used. Table 1-4 shows significant difference in reactivity compared to group B at 5% level of significance.

The value of mean and mean differences of HR responses is as shown in table 5 & 6. The value of mean and mean differences of SBP responses are as shown in table 8 & 9. The value of mean and mean differences of DBP responses are as shown in table 11 & 12. For comparison between group A and B Mann-Whitney U was used at immediately after exercise and at 3 minute after exercise. Table 7, 10, 13 shows statistically significant difference in of HR, SBP and DBP compared to group B respectively at 5% level of significance. After cool down period again Mann-Whitney U was used between two groups. There was no significant difference in group A and group B.

DISCUSSION

The present study was done to compare the effect of aerobic exercise on response of cardiovascular functions and reactivity in normotensive offsprings of hypertensive parents (group A) with the normotensive offsprings of normotensive parents (group B). Both groups in this study consisted of age, sex, and BMI matched, young, normotensive subjects. They differed only in the genetic propensity. The result shows that physical stress causes enhanced reactivity and increased immediate response of HR and BP in normotensive offsprings of hypertensive parents compared to normotensive offspring of normotensive parents.

Stress related cardiovascular reactivity is assumed to a marker of autonomic or sympathoadrenal functions.[6] Enhanced reactivity reflects a more general hyperadrenergic state, with elevation of neurohormones leading to increased risk for hypertension. BP reactivity may be a measure of poor endothelial function or the inability of the endothelium to counteract adequately the vasoconstrictive force induced by sympathetic stimuli.[7]

Folkow hypothesized that repeated episodes of stress over time cause a change in the wall-to-lumen ratio of arterioles in subjects with genetic predisposition to hypertension, ultimately eventuating in a fixed increase in peripheral resistance and future hypertension. Thus, both genetic predisposition and environmental interaction may play necessary roles in this

process.[8] Increased sympathetic activity may probably have a key role in the pathogenesis of hypertension.[9] In this study, normotensive individuals with a genetic predisposition for hypertension showed increased sympathetic activity in response to physical stress. Noll et al. found excessive sympathetic activation in response to mental stress in normotensive offsprings of hypertensive parents.[10] In the study of EG Ciolac, young normotensive women offsprings of hypertensive parents had increased resting, exercise, and recovery norepinephrine, and also exercise and recovery epinephrine than women with normotensive parents[11] which may result in increased sympathetic activity.

Increased endothelin-1 secretion play important role in development of hypertension. The enhanced vasoconstrictor response to endothelin-1 during exercise may be the result of an attenuated release of NO, a potent inhibitor of vascular contraction evoked by endothelin1. Additional explanation for enhanced vasoconstrictor response to endothelin- 1 may involve abnormality in endothelin receptor. In this study, the BP response to physical stress was exaggerated. This is supporeted by Enrico Mangieri, et al. handgrip increases endothelin-1 secretion in normotensive young male offspring of hypertensive parents.[12] In other study Carmel M. et al assessed response to handgrip exercise before and after infusion of endothelin receptor antagonist and found that vasodilator response to exercise was markedly enhanced in hypertensive but not in noremotensive.[3]

Folkow had proposed that peripheral vascular adaptation to stress is an initiating factor in the hypertensive disease process. He argued that cardiovascular response to stress place pressure on the arterial vessel walls, initiating hypertrophy of vascular smooth muscle. This increases the vessel wall/lumen ratio, causing a vascular hyper-reactivity to vasoconstrictor agents released during stress.[13]

The kidney may play a crucial role in development of genetic hypertension, perhaps in response to alteration in renal sympathetic activity. The possible mechanism would be early changes in glomerular function and vasoactive hormone execretion.[14,15] O´ Connor DT *et al.* found that early allteration in glomerular reserve in humans at genetic risk of essential hypertension. [16]

Another possible mechanism of exaggerated BP response may be the altered baroreceptor sensitivity. The baro-receptor reflex is a negative feedback system acting to reduce BP variability, with the adaptive end of preserving transcapillary pressure for maintain vital tissues which may cause increased BP response.[17] The study of L. R. Davrath, they found reduced baro-receptor sensitivity during the active change of posture from supine to standing position in the young adult normotensive offspring of hypertensive parents.[18]

In our study, exercise response causes enhanced reactivity & delayed recovery since exercise causes potent stress on the cardiovascular system. This is in supported by Harmer M that acute exercise & mental challenges causes blunted cardiovascular response in offspring of hypertensive parents.[19]

The long standing hypothesis that normotensive individuals who show large response to mental and physical challenge are at risk for hypertension. Karen A. tested this hypothesis on BP reactivity to psychological stress and concluded that larger BP response may be at risk for hypertension.[7] In contrast, the study of Manuck found no difference in HR and BP reactivity between parental history groups.[20] In our study, Group A showed statistically significant difference at 3 minute after exercise in HR, SBP, and DBP compared to group B. There is no significant difference found in HR, SBP, and DBP after cool down in between group A and group B.

The result suggests that increased response and delayed recovery present in normotensive offsprings of hypertensive parents compared to normotensive offspring of normotensive parents.

Reduced parasympathetic tone may have role in the etiology of hypertension. This is supported by G M Schneider, they found delayed recovery to repeated mental stress in HR, DBP and pre-ejection period in positive family group compared to negative family group.[21]

In contrast Visser et al. found no difference in vagal activity between positive and negative family history group after giving mental stress but they used active coping tasks (shock avoidance and a memory search task).[22]

The studies by Borghi et al. and Falkner et al. suggest that reactivity in young borderline hypertensive subjects can be used to predict stable hypertension several years later.[35,51] Frank A. conducted examining prospective studies and concluded that enhanced cardiovascular reactivity can predict the development of preclinical states & even new events in some patients of hypertension.[23]Evidence of elevated sympathetic activation at such an early stage of disease progression emphasizes the necessity of targeting reduction in sympathetic activation as primary goal in prevention in the treatment of hypertension.

The limitations of the study were invasive cardiovascular measures were not used and different group of offspring of one &/or two hypertensive parents was not considered.

Future research may be on response can be checked by isometric exercise and mental stress. Gender affection can be checked. Predicted age can be checked.

CONCLUSION

There is statistically significant difference in the reactivity and cardiovascular functions in normotensive offsprings of hypertensive parents compared to normotensive offsprings of normotensive parents. So clinically, early identification of these individuals with a prehypertensive profile is necessary. Life style modification such as weight reduction and moderate intensity aerobic exercise programme which may delay or prevent the onset of hypertension should be done.

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Table 1: Mean of Hr Reactivity

	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
HR(bpm)	23.27	16.87	8.03	5.45	10.00	10.00	40.00	28.00

Table 2: Mean Of Sbp Reactivity

	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
SBP(mmHg)	16.77	11.68	4.91	2.92	12.00	8.00	26.00	20.00

Table 3: Mean Of Dbp Reactivity

	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
DBP(mmHg)	8.93	4.13	2.95	2.72	4.00	2.00	16.00	10.00

Table 4: Results Of Tests For Reactivity

	U-value	p-value	Comment
HR(bpm)	246	0.0025	Significant
SBP(mmHg)	163	<o.ooo1	Significant
DBP(mmHg)	108	<o.ooo1	Significant

Table 5: Mean Of Hr Response After Exercise

HR(bpm)	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
Immediately after	116.5	111.4	6.76	4.92	103	105	126	125
After 3min	97.6	89.8	3.83	4.55	90	83	104	96
After cool down	80.07	79.53	4.05	4.04	75	72	90	87

Table 6: Mean Difference Of Hr Response After Exercise

HR(bpm)	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
Immediately after	34.93	31.87	7.1	5.36	21	25	47	45
After 3min	16	10.27	5.7	2.79	5	7	27	15
After cool down	1.5	0.13	5.8	0.89	-19	-2	7	1

Table 7: Results of Tests for Hr Response after Exercise

HR(bpm)	U-value	p-value	Comment
Immediately after	409.2	0.0392	Significant
After 3min	258	o.oo11	Significant
After cool down	442	0.9	Non-significant

Table 8: Mean of Sbp Response after Exercise

SBP(mmHg)	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
Immediately after	130.7	123.1	3.03	45.03	126	116	136	132
After 3min	121.1	113.9	4.63	4.78	114	108	128	124
After cool down	106.5	105.2	5.06	7.15	96	92	120	120

Table 9: Mean Difference Of Sbp Response After Exercise

SBP(mmHg)	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
Immediately after	24.8	19.6	5.39	5.26	16	10	36	30
After 3min	15.2	10.4	5.74	5.26	6	0	28	20
After cool down	1.66	1.73	6.17	3.22	-10	-6	18	8

Table 10: Results of Tests For Sbp Response After Exercise

HR(bpm)	U-value	p-value	Comment
Immediately after	318	0.014	Significant
After 3min	248	0.002	Significant
After cool down	354	0.15	Non-significant

Table 11: Mean Of Dbp Response After Exercise

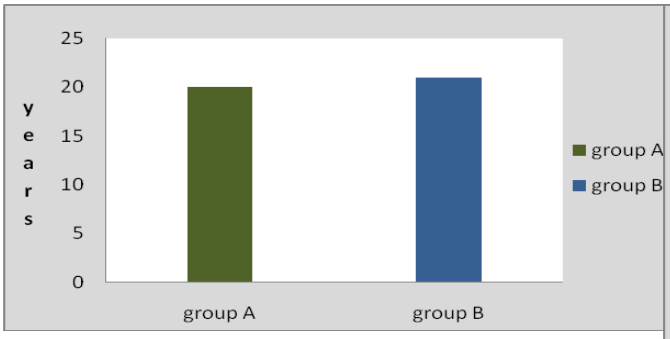
DBP(mmHg)	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
Immediately after	88.8	75.33	6.59	5.07	72	68	98	82
After 3min	79.73	66.53	5.55	4.14	70	60	88	74
After cool down	66.87	64.4	3.70	4.21	62	60	74	72

Table 12: Mean Difference Of Dbp Response After Exercise

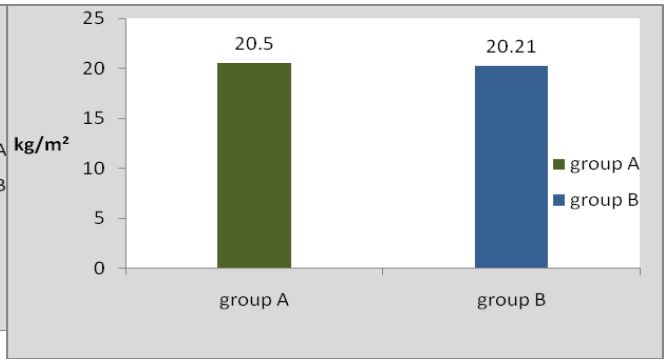
DBP(mmHg)	MEAN		SD		MINIMUM VALUE		MAXIMUM VALUE	
	Group A	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
Immediately after	18.53	11.07	6.27	4.38	8	6	32	20
After 3min	9.46	2.26	4.1	1.79	0	-2	18	4
After cool down	3.4	1.13	9.3	1.16	-24	-2	10	2

Table 13: Results Of Tests For Dbp Response After Exercise

HR(bpm)	U value	p value	Comment
Immediately after	242	0.0004	Significant
After 3min	52	<0.0001	Significant
After cool down	370	0.22	Non-significant



Graph 1: Age Distribution Of Subjects



Graph 2: Bmi Of Subjects

Photograph 1: Upperlimb & Trunk Exercise

Photograph 2: Treadmill Walking