RESEARCH PAPER | Medical Science | Volume : 6 | Issue : 6 | June 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

A Comparative Study on Effect of Obesity on Cardiovascular Reactivity in Young Female Individuals

KEYWORDS: Obesity,Cardiovascular,Body Mass Index, Female, Blood pressure

Jeewandeep Kaur	Arvinder Pal Singh Batra
Assistant Professor, Physiology (C R L), BPS GMC for Women, Sonepat.	Professor & Head, Anatomy, BPS GMC for Women, Sonepat.

ABSTRACT *Background-Studies have reported that both gender and adiposity influence cardiovascular reactivity amongst adolescents and young adults. However, not much is known about the effect of gender on the association of adiposity with cardiovascular reactivity.*

Aims and objective: to learn how adiposity affects cardiovascular reactivity amongst young female individuals so as to develop preventive strategies.

Material and methods: A cross-sectional study was conducted on 60 girls (normal weight n=30, over weight & Obese n= 30) of age group 18-22 years. Adiposity was assessed in terms of Body Mass Index (BMI), Waist Circumference (WC) and waist hip ratio. Rise in Pulse Rate and Rise in systolic and Diastolic Blood Pressure during Isometric Handgrip Test were used to assess the cardiovascular reactivity to acute sympathetic stress. Pearson's correlation coefficient was determined to find the association of adiposity with cardiovascular reactivity.

Results: Obese and overweight girls were found to have a significantly larger WC, lower physical fitness and greater rise in SBP& DBP in comparison to girls with normal weight. In both the groups, BMI and Waist Circumference showed significant positive association with DBP and SBP.

Conclusions: adiposity affects the cardiovascular reactivity in young female individuals such that girls with overweight and obese tend to have a larger correlation between adiposity and cardiovascular reactivity than girls with normal weight and thus preventive strategies need to be devised to prevent effect of obesity on cardiac vasculature in females

INTRODUCTION

Central (Visceral) adiposity is shown to have a larger influence on the cardiovascular risk factors for hypertension as compared to peripheral adiposity across all the age groups (1, 2). Pathophysiological imbalances in cardiovascular autonomic activity, cardiovascular reactivity to physical and mental stress, insulin sensitivity, leptin sensitivity, plasma lipid profile, vascular endothelial function and functioning of Renin-Angiotensin System (RAS) have been implicated in the obesity associated rise in blood pressure (3, 4). However, studies have also reported that differences exist in the etiopathogenesis of hypertension across varied ethnic populations, age groups and gender (5). Since exaggerated cardiovascular reactivity is associated with obesity and is considered as a risk factor for hypertension (1).

Obesity is associated with a greater prevalence of cardiovascular risk factors and a higher risk of cardiovascular events, and contributes to the rise in cardiovascular morbidity and mortality worldwide (7). Increased BMI is established as an independent risk factor for cardiovascular disease (CVD). Attention has recently been drawn to alternate measures of adiposity/obesity, such as waist circumference, waist-to-hip ratio and waist-to-height ratio, that provide information regarding body fat distribution. Although BMI is the established clinical measurement to estimate CVD risk associated with excess bodyweight, there is evidence suggesting that abdominal obesity could represent a better marker of CVD risk than BMI (7). Obesity is associated with several co-morbid conditions: dyslipidemia, hypertension, hyperglycemia, non-alcoholic fatty liver disease (NAFLD) and a conglomeration of conditions known as the metabolic syndrome (8).

It is essential to understand which of the adiposity indices has a predominant effect on the cardiovascular reactivity according to gender so as to help in framing guidelines for preventive measures. The biological mechanisms linking obesity and stress reactivity are poorly understood. Adipose tissue is now recognized as a major endocrine organ that secretes signalling molecules playing a central role in inflammation, weight regulation and metabolic function including cytokines (9). We set out to investigate the relationship between adiposity, and cardiovascular reactivity in a sample of healthy young female. We predicted that women with greater adiposity would have heightened or prolonged cardiovascular reactivity. Studies have shown that women with a larger waist circumference or waist–hip ratio have heightened cortisol responses to acute laboratory stress, as well as impaired dexamethasone suppression of cortisol (10-11).

Similarly, central obesity has been associated with elevated or prolonged cardiovascular responses to acute stress in some but not all studies and a recent report found larger cortisol and cardiovascular responses to public speaking stress in obese versus non-obese women (12).

AIMS AND OBJECTIVES

The current study is planned to learn how adiposity affects the cardiovascular reactivity in the young female individuals so as to develop preventive strategies for that group of population.

MATERIALS AND METHODS

Thirty (30) obese/ overweight and thirty (30) non-obese female volunteers aged between 18 and 22 years were participated in the study. The revised BMI cut-off for Asians as recommended by WHO were considered to classify overweight and obesity (6) Table-1. The purpose of the study

Volume : 6 | Issue : 6 | June 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

was explained to all the participants. Institutional ethical committee approval has already been taken. Pulse rate and blood pressure was measured prior to exercise at the brachial artery from the arm not involved in exercise (non-exercising arm) using the Omron T8 Automatic Blood Pressure instrument.

Inclusion criteria for both groups:

1) Healthy young adults aged between 18-22 years
2) Sedentary life style.

Exclusion criteria for both groups:

1) Subjects with history of cardiopulmonary disease
2) Chronically ill
3) Medicationfor long duration,
4) History of any major surgery (cardiac, pulmonary, abdominal) related to study and
5) Subjects undergoing any physical conditioning program.

BMI will be calculated using Quetlet's index as weight (kg) divided by (height in m) ^{2}.

BMI = Weight (Kg) / (height in meter) 2.

W/H Ratio (Waist-Hip Circumference ratio): The waist Circumference is the minimum Circumference between the costal margin and iliac crest, measured in horizontal plane, with the subject standing. Hip circumference is the maximum Circumference in the horizontal plane, measured over the buttocks. The ratio of the former to the latter provides an index of proportion of intra-abdominal fat.

TABLE1: Cut-offs of obesity and abdominal obesity for Asian Indians *vs.* international criteria.

Variable	Consensus guidelines for Asian Indians [a]	Prevalent International Criteria
Generalized obesity	Normal: 18.0–22.9	Normal: 18.5–24.9 [b]
(BMI cut-offs in kg/m^2)	Overweight: 23.0–24.9 Obesity: >25	Overweight: 25.0–29.9 [b] Obesity: >30 [b]
Abdominal obesity (Waist circumference cut-offs in cm)	Men: >90 [c] Women: >80 [c]	Men: >102 [d] Women: >88 [d]

Notes: [a] From Consensus guidelines for Asian Indians; [b] According to World Health Organization guidelines; [c] Both as per Consensus Guidelines for Asian Indians and International Diabetes Federation; [d] According to Modified National Cholesterol Education Program, Adult Treatment Panel III guidelines.

CARDIOVASCULAR REACTIVITY: The cardiovascular reactivity was assessed by measuring the changes in pulse rate and blood pressure during an Isometric Hand Grip Test by dynamic hand dynamometer. The participants were asked to do maximum voluntary contraction using the handgrip dynamometer with the dominant hand. Three attempts were made at intervals of 1 minute and the highest reading amongst the attempts was considered as the maximum voluntary contraction (MVC) for the participant. Pulse rate and blood pressure were measured prior to exercise at the brachial artery from the arm not involved in exercise (non-exercising arm) using the Omron T8 Automatic Blood Pressure instrument. The participants were then asked to perform isometric handgrip exercise at an intensity of 30% MVC for 1 minute in the sitting posture. Pulse rate and Blood Pressure was measured at 1 minute from the non-exercising arm. The percentage rise in pulse rate, systolic blood pressure and diastolic blood pressure due to isometric exercise was calculated from the pre-exercise and 1-minute exercise values. Mean and Standard Deviation was calculated for the independent and dependent variables. Pearson's correlation coefficient was determined to find the association of adiposity and gender with cardiovascular reactivity.

OBSERVATIONS AND RESULTS

Table 1.1 & 1.2 depicts Mean and standard deviation of anthropometric profile of study population. The BMI, waist circumference and waist hip Ratio were significantly higher amongst Obese & Overweight individuals compared to Normal weight individuals (P< 0.005 one tailed & P<0.001 two tailed)

TABLE 1.1- GROUP STTISTICS AND INDEPENDENT SAMPLE TEST FOR ANTHROPOMETRIC PROFILE OF STUDY POPULATION

Group Statistics				
Variables	Group	N	Mean	Std. Deviation
BMI (Kg/m^2)	1	30	20.66	1.31
	2	30	25.53	3.21
Waist (Inches)	1	30	27.03	2.23
	2	30	30.63	3.45
Hip (Inches)	1	30	35.81	2.23
	2	30	40.46	2.93
Waist/Hip Ratio	1	30	0.75	0.055
	2	30	1.0000	0.058

Table 1.2- GROUP STTISTICS AND INDEPENDENT SAMPLE TEST FOR ANTHROPOMETRIC PROFILE OF STUDY POPULATION

Independent Samples Test							
		Levene's Test for Equality of Variances		t-test for Equality of Means			
		F	Sig.	t	Df	Sig. (2-tailed)	Mean Difference
BMI (Kg/m^2)	Equal variances assumed	7.51	0.008	-7.683	58	0.0001	-4.87
	Equal variances not assumed			-7.683	38.44	0.0001	-4.87
Waist	Equal variances assumed	9.23	0.004	-4.790	58	0.0001	-3.60
	Equal variances not assumed			-4.790	49.58	0.0001	-3.60
HIP	Equal variances assumed	2.71	0.105	-6.908	58	0.0001	-4.65
	Equal variances not assumed			-6.908	54.177	0.0001	-4.65
Waist / hip Ratio	Equal variances assumed	69.75	0.0001	-24.331	58	0.0001	-.24
	Equal variances not assumed			-24.331	29.000	0.0001	-.24

Group 1= Normal weight, Group 2= Over weight & Obese

Table 2.1 & 2.2 depicts Mean and standard deviation of Blood Pressure Pulse profile of study population. There was increase in SBP, DBP, & PR after exercise in both the group but it was significant only for changes in the pulse rate (P< 0.014 for 1 tailed & 0.031 fo4 2 tailed & for SBP, DBP P>0.05 one tailed & two tailed)

Volume : 6 | Issue : 6 | June 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

TABLE 2.1 GROUP STTISTICS AND INDEPENDENT SAMPLE TEST FOR BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Group Statistics				
	Group	N	Mean	Std. Deviation
SBP Before	1	30	114.30	8.91
	2	30	116.23	10.51
SBP After	1	30	119.07	9.69
	2	30	122.93	12.06
DBP Before	1	30	69.43	6.30
	2	30	70.40	7.03
DBP After	1	30	72.83	6.80
	2	30	77.37	10.45
PR Before	1	30	74.27	8.38
	2	30	79.20	8.87
PR After	1	30	83.73	12.11
	2	30	88.67	8.60

TABLE 2.2 GROUP STTISTICS AND INDEPENDENT SAMPLE TEST FOR BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Independent Samples Test							
		Levene's Test for Equality of Variances		t-test for Equality of Means			
		F	Sig.	t	Df	Sig. (2-tailed)	Mean Difference
SBP Before	Equal variances assumed	1.67	0.201	-.768	58	0.446	-1.93
	Equal variances not assumed			-.768	56.49	0.446	-1.93
SBP After	Equal variances assumed	1.624	0.21	-1.368	58	0.177	-3.86
	Equal variances not assumed			-1.368	55.44	0.177	-3.86
DBP Before	Equal variances assumed	1.038	0.31	-.561	58	0.577	-.967
	Equal variances not assumed			-.561	57.32	0.577	-.96
DBP After	Equal variances assumed	2.197	0.144	-1.990	58	0.051	-4.53
	Equal variances not assumed			-1.990	49.85	0.052	-4.53
PR Before	Equal variances assumed	0.025	0.874	-2.213	58	0.031	-4.93
	Equal variances not assumed			-2.213	57.81	0.031	-4.93
PR After	Equal variances assumed	6.458	0.014	-1.819	58	0.074	-4.93
	Equal variances not assumed			-1.819	52.32	0.075	-4.933

Group 1= Normal weight, Group 2= Over weight & Obese, SBP Before= Systolic Blood Pressure Before start of exercise, SBP After = Systolic Blood Pressure after of exercise, DBP Before = Diastolic Blood Pressure Before start of exercise, DBP After = Diastolic Blood Pressure after exercise, PR Before = Pulse rate Before start of exercise, PR After = Pulse rate After exercise

Table 3.1-3.2 depicts Mean and standard deviation of Blood Pressure Pulse profile before and after exercise in the of study population when both the groups taken together. When paired t test was applied it was observed that there was significant increase in SBP, DBP, & PR after exercise when we considered both the groups as a single group (P= 0.0001 two tailed)

TABLE 3.1 PAIRED SAMPLE STATISTICS AND CORRELATIONS OF BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Paired Samples Statistics				
		Mean	N	Std. Deviation
Pair 1	SBP Before	115.27	60	9.716
	SBP After	121.00	60	11.025
Pair 2	DBP Before	69.92	60	6.639
	DBP After	75.10	60	9.040
Pair 3	PR Before	76.73	60	8.914
	PR After	86.20	60	10.708

TABLE 3.2 PAIRED SAMPLE STATISTICS AND CORRELATIONS OF BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Paired Samples Correlations				
		N	Correlation	Sig.
Pair 1	SBP Before & SBP After	60	.574	0.0001
Pair 2	DBP Before & DBP After	60	.595	0.0001
Pair 3	PR Before & PR After	60	.676	0.0001

TABLE 3.3 PAIRED SAMPLE STATISTICS AND CORRELATIONS OF BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Paired Samples Test						
		Paired Differences		t	df	Sig. (2-tailed)
		Mean	Std. Deviation			
Pair 1	SBP Before – SBP After	-5.733	9.644	-4.605	59	0.0001
Pair 2	DBP Before – DBP After	-5.183	7.373	-5.446	59	0.0001
Pair 3	PR Before – PR After	-9.467	8.066	-9.091	59	0.0001

SBP Before= Systolic Blood Pressure Before start of exercise, SBP After = Systolic Blood Pressure after of exercise, DBP Before = Diastolic Blood Pressure Before start of exercise, DBP After = Diastolic Blood Pressure after exercise, PR Before = Pulse rate before start of exercise, PR After = Pulse rate after exercise

Table 4.1-4.2 depicts descriptive statistics and auto correlations of mean and standard deviation of Blood Pressure Pulse profile before and after exercise in the of study population based on BMI when both the groups taken together. There was significant increase in SBP, DBP, & PR after exercise when we considered both the groups as a single group and when we correlated the data before and after exercise (P= 0.0001 two tailed)

Volume : 6 | Issue : 6 | June 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

TABLE 4.1 DESCRIPTIVE STATISTICS AND CORRELATION OF BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Descriptive Statistics			
Variables	Mean	Std. Deviation	N
SBP Before	115.2667	9.71608	60
SBP After	121.0000	11.02539	60
DBP Before	69.9167	6.63910	60
DBP After	75.1000	9.03984	60
PR Before	76.7333	8.91365	60
PR After	86.2000	10.70846	60
BMI(Kg/m^2)	23.0981	3.45776	60

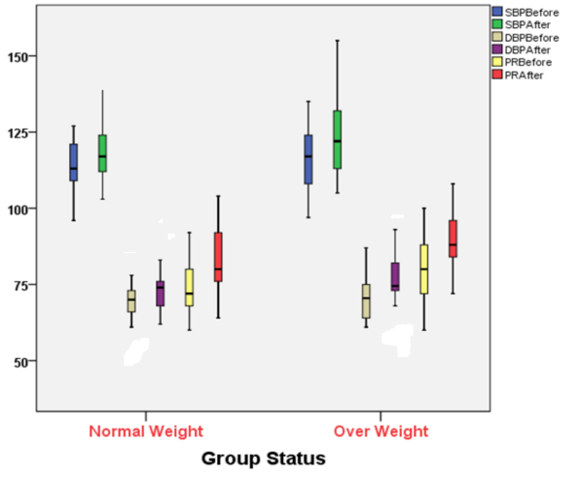
TABLE 4.2 DESCRIPTIVE STATISTICS AND CORRELATION OF BLOOD PRESSURE PULSE PROFILE OF STUDY POPULATION

Correlations								
Control Variables			SBP Before	SBP After	DBP Before	DBP After	PR Before	PR After
BMI Kg/m^2	SBP Before	Correlation	1.000	.517	0.635	0.342	-.001	-.110
		Significance (2-tailed)	.	.0001	0.0001	0.008	0.995	0.409
		Df	0	57	57	57	57	57
	SBP After	Correlation	.517	1.000	0.57	0.505	-.127	-.059
		Significance (2-tailed)	.0001	.	0.0001	.0001	0.340	0.655
		Df	57	0	57	57	57	57
	DBP Before	Correlation	.635	.577	1.00	0.582	0.140	0.061
		Significance (2-tailed)	.0001	.0001	.	0.0001	0.289	0.646
		Df	57	57	0	57	57	57
	DBP After	Correlation	.342	.505	0.58	1.000	0.041	-.005
		Significance (2-tailed)	.008	.0001	0.0001	.	0.760	0.972
		Df	57	57	57	0	57	57
	PR Before	Correlation	-.001	-.127	0.140	0.041	1.000	0.668
		Significance (2-tailed)	.995	.340	0.289	0.760	.	0.0001
		Df	57	57	57	57	0	57
	PR After	Correlation	-.110	-.059	0.061	-.005	0.668	1.000
		Significance (2-tailed)	.409	.655	0.646	.972	0.0001	.
		Df	57	57	57	57	57	0

SBP Before= Systolic Blood Pressure Before start of exercise, SBP After = Systolic Blood Pressure after of exercise, DBP Before = Diastolic Blood Pressure Before start of exercise, DBP After = Diastolic Blood Pressure after exercise, PR Before = Pulse rate before start of exercise, PR After = Pulse rate after exercise

From-Figure 1 depicts graphical representation of mean and standard error of mean of Blood Pressure Pulse profile before and after exercise in the of study population (Normal weight & Overweight & Obese group) based on BMI

FIGURE 1: BLOOD PRESSURE PULSE PROFILE IN THE STUDY GROUPS BEFORE AND AFTER EXERCISE



DISCUSSION

We investigated the association between adiposity measures and cardiovascular reactivity responses using a multiple linear regression approach, descriptive statistical approach and independent sample test and paired test. Three aspects of physiological response were analyzed: baseline anthropometrics profiles, cardiovascular reactivity (computed as the change in levels of Blood pressure pulse profiles between baseline and isometric hand grip test). This time adiposity measures (BMI, waist circumference and waist Hip ratio) were also included as covariates, since adiposity is a major determinant of stress reactivity. Results are presented as mean and standard deviation with standard errors of mean. Associations between adiposity measures and cardiovascular reactivity are illustrated by displaying the mean cardiovascular response to isometric hand grip test.

The findings of our study show that obese & overweight girls have a stronger correlation between adiposity and cardiovascular reactivity as compared to normal weight girls. This finding may be explained by the fact that obese girls have a significantly lower physical fitness in comparison to normal weight girls and since physical fitness shows a significant negative correlation with cardiovascular reactivity amongst normal weight girls, it is possible that the low physical fitness in obese & overweight girls predisposes them to a greater cardiovascular reactivity to acute sympathetic stress. This view is supported by the observations that, fit individuals show significantly attenuated heart rate and systolic blood pressure reactivity and a trend towards attenuated diastolic blood pressure reactivity (13-14). Our findings also suggest that visceral adiposity as indicated by waist circumference though has lower correlations with vascular reactivity in comparison to total adiposity it seems to be more influential in increasing cardiovascular responsiveness to sympathetic stress than the BMI. Similar findings have also been reported by other studies which were conducted to learn the pathogenesis of high blood pressure

Volume : 6 | Issue : 6 | June 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

associated with an increase in adiposity. A study conducted on 46 White and 49 Black normotensive adolescents with family histories of essential hypertension found that after controlling for peripheral (that is, triceps skinfold) and overall (that is, BMI) adiposity, the group with higher Waist to Hip Ratio exhibited greater SBP (that is, peak response minus mean pre-stressor level) to all three stressors and greater DBP reactivity to postural change and cold pressor (all P<0.05). The study concluded that Central adiposity appears to adversely influence hemodynamic functioning during adolescence (15).

Another study examined the association between central adiposity, measured by waist circumference, and cardiovascular reactivity to stress among 106 White and 105 Black adolescents, approximately 50% of whom were girls. Participants engaged in 4 laboratory tasks while cardiovascular reactivity measures were taken. Independent of body mass index, race, and gender, participants with a greater waist circumference exhibited greater systolic blood pressure reactivity and diastolic blood pressure reactivity. Results from the study suggested that central adiposity is associated with blood pressure reactivity early in life (16). Prolonged sympathetic reactivity to acute stress has been shown to predict future hypertension in initially normotensive samples as well as an increased risk of cardiac events in patients with documented cardiovascular disease (17).

Although few studies have examined the prospective relationship between acute cytokine responses and cardiovascular risk, and one previous study showed that IL-6 responses to laboratory stress were predictive of 3-year elevations in BP in men and women from the Whitehall II cohort (18, 19).

A major limitation in the current study is that it is a cross-sectional study which is a useful but a weak tool in assessing the relationship between two variables. Another major limitation is the lack of the study of sex hormones and their influence on the study variables.

CONCLUSION

It can thus be concluded from our study that though overweight & obese individuals tend to have a higher cardiovascular reactivity to acute sympathetic stress than normal weight girls, they tend to show a greater correlation between adiposity and vascular reactivity due to low physical fitness and therefore low physical fitness predisposes girls to the adverse effects of adiposity on the vasculature. It is also concluded from the study that visceral adiposity seems to be the type of adiposity which influences cardiovascular reactivity to greater extent than total adiposity amongst young female individuals. Further study may be conducted to search for further evidence in this line by studying the influence of sex hormones on the relationship and studying the relationship during the two phases of menstrual cycle.

SUMMARY

Studies have reported that both gender and adiposity influence cardiovascular reactivity amongst adolescents and young adults. However, not much is known about the effect of gender on the association of adiposity with cardiovascular reactivity. The current study was conducted to learn how adiposity affects cardiovascular reactivity in the amongst young female individuals so as to develop preventive strategies. A cross-sectional study was conducted on 60 girls (normal weight n=30, over weight & Obese n= 30) of age group 18-22 years. Adiposity was assessed in terms of Body Mass Index (BMI), Waist Circumference (WC) and waist hip ratio. Rise in Pulse Rate and Rise in systolic and Diastolic Blood Pressure during Isometric Handgrip Test were used to assess the cardiovascular reactivity to acute sympathetic stress. Pearson's correlation coefficient was determined to find the association of adiposity with cardiovascular reactivity. Obese and overweight girls were found to have a significantly larger WC, lower physical fitness and greater rise in SBP& DBP in comparison to girls with normal weight. In both the groups, BMI and Waist Circumference showed significant positive association with DBP and SBP. It could thus be concluded that adiposity affects the cardiovascular reactivity in young female individuals such that girls with overweight and obese tend to have a larger correlation between adiposity and cardiovascular reactivity than girls with normal weight. Nevertheless, our findings throw some light on the potential mechanisms linking obesity, stress and cardiovascular reactivity in young females. BMI, waist circumference and waist-to-hip ratio measurements are all useful tools for assessing adiposity/obesity in clinical practice, and should be evaluated with other cardio-metabolic risk factors to refine cardiovascular reactivity stratification.

REFERENCES

1. Rahmouni K, Correia ML, Haynes WG, Mark AL. Obesity-associated hypertension: new insights into mechanisms. Hypertension 2005;45: 9–14.
2. Aneja A, El-Atat F, McFarlane SI, Sowers JR. Hypertension and obesity. Recent Prog HormRes 2004; 59: 169-205.
3. Barnes VA, Treiber FA, Davis H, Kelley TR, Strong WB. Central adiposity and hemodynamic functioning at rest and during stress in adolescents. Int J Obes Relat Metab Disord 1998; 22: 1079–1083.
4. Schwandt P, Bertsch T, Haas GM. Anthropometric screening for silent cardiovascular risk factors in adolescents: The PEP Family Heart study. Atherosclerosis 2010; 211: 667–671.
5. Bell AC, Adair LS and Popkin BM. Ethnic Differences in the Association between Body Mass Index and Hypertension. Am J Epidemiol 2002; 155: 346–353.
6. WHO Expert Consultation: Appropriate body mass index for Asian population and its implications for policy and intervention strategies. Lancet 2004; 363: 157-163.
7. Rhéaume C[1], Leblanc MÈ, Poirier P. Adiposity assessment: explaining the association between obesity, hypertension and stroke. Expert Rev Cardiovasc Ther. 2011 Dec;9(12):1557-64.
8. Anoop Misra and Usha Shrivastava. Obesity and Dyslipidemia in South Asians. Nutrients. 2013; 5(7): 2708–2733.
9. Grundy S.M., Brewer H.B., Jr., Cleeman J.I., Smith S.C., Jr., Lenfant C. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Circulation. 2004;109:433–438. 24. Grundy S.M., Cleeman J.I., and Daniels S.R., et al. Diagnosis and management of the metabolic syndrome. An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Executive Summary. Circulation. 2005;112:2735–2752.
10. Epel E.S., McEwen B., and Seeman T., et al. Stress and body shape: stress-induced cortisol secretion is consistently greater among women with central fat. Psychosomatic Medicine. 2000;62:623–632.
11. Pasquali R., Ambrosi B.,and Armanini D., et al. Cortisol and ACTH response to oral dexamethasone in obesity and effects of sex, body fat distribution, and dexamethasone concentrations: a dose–response study. Journal of Clinical Endocrinology and Metabolism. 2002;87:166–175.
12. Benson S., Arck P.C., and Tan S., et al. Effects of obesity on neuroendocrine, cardiovascular, and immune cell responses to acute psychosocial stress in premenopausal women. Psychoneuroendocrinology. 2009; 34:181–189.
13. Sharma RK, Deepak KK, Bijlani RL, Rao PS. Short-term physical training alters cardiovascular autonomic response amplitude and latencies. Ind J Physiol Pharmacol 2004;48: 165–173.
14. Forcier K, Stroud LR, Papandonatos GD, Hitsman B, Reiches M, Krishna-

Volume : 6 | Issue : 6 | June 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

moorthy J et al. Links between physical fitness and cardiovascular reactivity and recovery to psychological stressors: A meta-analysis. Health Psychol 2006; 25:723–739.

15. Barnes VA, Treiber FA, Davis H, Kelley TR, Strong WB. Central adiposity and hemodynamic functioning at rest and during stress in adolescents. Int J Obes Relat Metab Disord 1998;22: 1079–1083.
16. Goldbacher EM, Matthews KA, Salomon K. Central adiposity is associated with cardiovascular reactivity to stress in adolescents. Health Psychol 2005; 24: 375–384.
17. Treiber F.A., Kamarck T., Schneiderman N., Sheffield D., Kapuku G., Taylor T. Cardiovascular reactivity and development of preclinical and clinical disease states. Psychosomatic Medicine. 2003;65:46–62.
18. Flaa A., Eide I.K., Kjeldsen S.E., Rostrup M. Sympathoadrenal stress reactivity is a predictor of future blood pressure: an 18-year follow-up study. Hypertension. 2008;52:336–341.
19. Brydon L., Steptoe A. Stress-induced increases in interleukin-6 and fibrinogen predict ambulatory blood pressure at 3-year follow-up. Journal of Hypertension. 2005;23:1001–1007.