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# A Comparative Study of Electrocardiographic Changes during Different Trimesters of Pregnancy with Nonpregnant Controls

Nandini B.N.<sup>1</sup>, Manjunath M.L.<sup>2</sup>

## Abstract

**Introduction:** During normal gestation, a variety of hemodynamic changes are going to occur. They will influence the condition of heart, which in turn results in changes in electrocardiography. Hence, the present study was designed to study the electrocardiographic changes during different trimesters of pregnancy was compared with nonpregnant controls. **Materials and Methods:** It is a cross sectional study conducted in the Department of Physiology after institutional clearance and consent from all the participants, 150 pregnant women in the age group of 20-35yrs who were attending the OPD of OBG were recruited and divided into 3 subgroups comprising 50 women in first, second and third trimesters of pregnancy. The control group was comprising of another apparently healthy age matched 50 non-pregnant women. ECG was recorded in all 12 leads and was evaluated for different parameters such as heart rate, P wave, PR interval, QRS complex, Q wave, T wave, QTc interval, axis deviation, R and S amplitudes and ST segment. **Statistical Analysis:** The data were expressed as Mean $\pm$ SD. Z test was used for comparison between control and study groups and within the study group. 'p' value of 0.05 or less was considered as statistically significant. **Result:** There was statistically significant decrease in PR interval ( $p < 0.001$ ) in all trimesters of pregnancy when compared to control group. QT intervals did not show significant difference between the control and the study groups or within in the subgroup of study group ( $p > 0.05$ ). But, QTc interval showed a statistically significant increase 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimester of pregnancy when compared to control group ( $p < 0.001$ ). Similarly, a statistically significant increase in QTc interval was observed in 2<sup>nd</sup> and 3<sup>rd</sup> trimester ( $p < 0.001$ , Table-6) of pregnancy when compared to 1<sup>st</sup> trimester and also in 3<sup>rd</sup> trimester ( $p < 0.001$ ) of pregnancy when compared to 2<sup>nd</sup> trimester. **Discussion:** Heart rate was increased in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy, a decrease in PR interval was seen all the trimesters of pregnancy, Occurrence of Q wave in leads II and III showed an increase in 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy, ST segment depression was noticed in 4 subjects in 2<sup>nd</sup> trimester and 6 in 3<sup>rd</sup> trimester of pregnancy, QTc interval showed an increase in pregnant women in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy. **Conclusion:** This knowledge may be helpful in the prevention of gestational complications associated with an inadequate maternal hemodynamic adaptation.

**Keywords:** Pregnancy; Hemodynamic Changes; ECG Changes; Trimesters.

## Introduction

In pregnant women, large number of local and systemic changes are known to occur. These changes will continue throughout pregnancy especially cardiovascular changes such as increase in heart rate, cardiac output and intravascular volume [1]. The physiological changes during pregnancy facilitate the adaptation of the cardiovascular system to the increased metabolic needs of the mother enabling adequate delivery of oxygenated blood to the peripheral tissues and to the foetus.

The demands for an increased flow of blood during pregnancy are met mainly by increasing the cardiac

output. In an average non-pregnant woman, cardiac output is about 4.5 litre per minute. At the eighth month of pregnancy, this rises to about 5.5 L. The cardiac output rises to a peak in the middle of pregnancy and thereafter slowly declines thereafter though it still remains 1 L/min above the non-pregnant values. The decline in cardiac output in late pregnancy might be due to postural changes. In the supine position, the large uterus often impedes cardiac venous return. It can decrease to about 20% less in supine position as compared to the lateral recumbent position [2,3].

Cardiac output depends on the heart rate and stroke volume. The heart rate increases by 10-15 beats per minute more than the pre - pregnant state. There

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is an increase in both stroke volume and heart rate. The stroke volume increases to 10% more than the non-pregnant value, whereas the heart rate increases to 20% more than the non-pregnant value. In the early months of pregnancy, the stroke volume rises rapidly to a peak and then declines, while the pulse rate slowly increases. The mechanisms of increasing the cardiac output have varying importance at the extremes of pregnancy [4].

Heart diseases during pregnancy remain a serious problem. One of the important tools for the diagnosis of heart diseases is recording electrocardiogram. Electrocardiography is one of the simplest techniques used to detect ischemic heart diseases, hypertensive heart diseases & asymptomatic arrhythmias [5]. The effect of pregnancy on the electrocardiogram has been a subject of great interest since the early days of electrocardiography [6]. A major purpose of recording ECG in clinical practice is to help the clinician in the diagnosis and prognosis of heart diseases.

Halphen C et al., conducted electrocardiographic study of left ventricular performance in normal pregnancy and reported that the heart rate raised significantly from third to ninth month and then fell during the postpartum period [7]. 50% of the maximum increase in heart rate had already occurred by 8 weeks. The initial abrupt increase was followed by a more gradual progressive rise as pregnancy continued which plateaued after 32 weeks. The abrupt increase in heart rate in early pregnancy suggests a hormonal mechanism. This is linked to the production of chorionic gonadotropin with the later gradual increase being related to the vascular changes which accompany placental and foetal growth.

Lechmanova M and Parizet A et al., compared the measured parameters of the electrical field with hemodynamic parameters before & after delivery in the group of non-obese women with physiological pregnancy & in a group of healthy non-obese & non-pregnant women [8]. They observed several significant changes of the electrical field in the pregnant women such as, increase in the heart rate, shortening of AV conductance, prolongation of QT interval normalized for the heart rate (QTc), change in the ventricular depolarization & repolarisation pattern.

Philip J Podrid observed ECG changes during normal pregnancy and observed shortening of PR & QT interval, may accompany the increase in the heart rate, frontal lead axis changes were rare despite significant elevation of diaphragm when seen, slight right ward shift were more common than leftward

deviation, nonspecific abnormalities of ST segment & T wave appeared in 4 to 14% of the pregnancy [9]. These changes predominated in left precordial leads & resolved in the majority of subjects after delivery.

During normal gestation, a variety of hemodynamic changes are going to occur. They will influence the condition of heart, which in turn results in changes in electrocardiography. Hence, the present study was designed to study the electrocardiographic changes during different trimesters of pregnancy was compared with nonpregnant controls.

## Materials and Methods

It is a cross sectional study conducted in the Department of Physiology, Shimoga institute of Medical Sciences. Sagar Road, Shivamogga. 150 pregnant women in the age group of 20-35yrs who were attending the OPD of OBG were included in the study group. The study group was in turn divided into 3 subgroups. Each sub group was comprising of 50 women in first, second and third trimesters of pregnancy. The control group was comprising of another apparently healthy age matched 50 non-pregnant women.

The nature and purpose of the study were explained to the subjects who had volunteered for the study. From each participant an informed consent was obtained. A thorough physical & systemic examination of each subject was done (in particular, cardiovascular and respiratory system). Recordings were taken during morning hours between 9 am to 12 Noon.

Apparently healthy subjects of Indian origin were included in the study. The apparent health status of the subject was determined through thorough clinical examination and history taking. Subjects with history or clinical signs of cardiovascular diseases, acute respiratory infection in the previous three months, history of diabetes mellitus, hypertension, history of tobacco consumption in any form, history of alcohol intake, any endocrine disorders, obesity and with moderate to severe anaemia were excluded.

ECG was recorded after giving 5 minutes of rest to the subject to allay anxiety. ECG was recorded in all 12 leads such as 3 Standard Bipolar Limb Leads I, II & III, 3 Unipolar augmented limb leads: aVR, aVL, aVF and 6 Precordial leads: VI to V6, by connecting electrodes to left arm, right arm, left leg and right leg in supine position. Date of recording, name and age of the subject were written on ECG strip.

ECG recorded was evaluated for different

parameters such as heart rate, P wave, PR interval, QRS complex, Q wave, T wave, QTc interval, axis deviation, R and S amplitudes and ST segment.

### Statistical Analysis

The results were expressed as Mean±SD. Z test was used for comparison between control and study groups and within the group. A 'p' value of 0.05 or less was considered as statistically significant.

### Result

The heart rate showed a statistically significant increase in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnant women when compared to non-pregnant women (p <0.001) (Table 1). Similarly, there was a statistically significant increase in heart rate in 2<sup>nd</sup> and 3<sup>rd</sup> trimesters (p <0.001) when compared to 1<sup>st</sup> trimester of pregnant women. There was a statistically significant increase in heart rate in 3<sup>rd</sup> trimester (p <0.001) when compared to 1<sup>st</sup> trimester of pregnant women (Table 2).

The P wave duration among control and study group and within the subgroups of study group were not statistically significant (p>0.05). The P wave amplitude among control and study group and within the subgroup of study group were not statistically significant (p>0.05, Table 3).

There was statistically significant decrease in PR

interval (p<0.001) in all trimesters of pregnancy when compared to control group. Similarly, a statistically significant decrease in PR interval was observed in 2<sup>nd</sup> (p<0.05) and 3<sup>rd</sup> trimesters (p<0.001) when compared to 1<sup>st</sup> trimester of pregnancy. Duration of QRS Complex (seconds) did not show significant difference among the control and study groups or within the subgroups of study group (p>0.05, Table 3).

Occurrence of Q wave in lead I showed a slight increase in 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy when compared to 1<sup>st</sup> trimester of pregnancy and control group. But, there was no significant statistical difference between the groups (p>0.05, Table 4).

QT intervals (sec) also did not show significant difference between the control and the study groups or within in the subgroup of study group (p> 0.05, Table 5). But, QTc interval showed a statistically significant increase 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimester of pregnancy when compared to control group (p<0.001). Similarly, a statistically significant increase in QTc interval was observed in 2<sup>nd</sup> and 3<sup>rd</sup> trimester (p< 0.001, Table 6) of pregnancy when compared to 1<sup>st</sup> trimester and also in 3<sup>rd</sup> trimester (p< 0.001) of pregnancy when compared to 2<sup>nd</sup> trimester (Table 7).

The T wave duration among control and study groups and within groups was not statistically significant (p>0.05, Table 8). The T wave amplitude among control and study groups and within the groups was not statistically significant (p>0.05, Table 9).

**Table 1:** Mean ± SD of Heart rate, P wave and PR Interval of subjects in Control and Study groups

Parameters	Control Mean ± SD	1 <sup>ST</sup> Trimester Mean ± SD	2 <sup>ND</sup> Trimester Mean ± SD	3 <sup>RD</sup> Trimester Mean ± SD
HR (beats/min)	75.68 ± 3.99	82.28 ± 7.84	88.24 ± 9.10	95.52 ± 7.04
P wave				
Duration (sec)	0.08 ± 0.01	0.08 ± 0.01	0.08 ± 0.01	0.07 ± 0.01
Amplitude (mv)	1.00 ± 0.17	1.00 ± 0.23	1.02 ± 0.27	1.02 ± 0.28
PR Interval(sec)	0.15 ± 0.01	0.14 ± 0.02	0.14 ± 0.02	0.13 ± 0.02

**Table 2:** Test of Significance of Heart rate, P wave and PR Interval using Z Statistics b/n Control and Study groups

Parameters	Control & 1 <sup>ST</sup> Trimester P-Value	Control & 2 <sup>ND</sup> Trimester P-Value	Control & 3 <sup>RD</sup> Trimester P-Value
HR (beats/min)	0.0001***	0.0001***	0.0001***
P wave			
Duration (sec)	0.667	0.696	0.711
Amplitude(mv)	1	1	1
PR Interval (sec)	0.0003**	0.0003**	0.0003**

p>0.05: Not Significant, \*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant

**Table 3:** Test of Significance for Heart rate, P wave and PR Interval using Z Statistics within the subgroups of Study group

Parameters		1 <sup>ST</sup> & 2 <sup>ND</sup> Trimesters P-Value	1 <sup>ST</sup> & 3 <sup>RD</sup> Trimesters P-Value	2 <sup>ND</sup> & 3 <sup>RD</sup> Trimesters P-Value
P wave	HR (beats/min)	0.0001***	0.0001***	0.0001***
	Duration (sec)	0.696	0.218	0.624
	Amplitude(mv)	1	0.689	1
	PR Interval (sec)	0.012*	0.0001***	0.0034**

p>0.05: Not Significant, \*p<0.05: Significant, \*\*p <0.01: Highly significant, \*\*\*p<0.001: Very highly significant.

**Table 4:** Chi Square Test for association of occurrence of Q wave in Std Limb Leads between the study and the control groups

Limb Leads	Occurrence of Q Wave		
	Control&1 <sup>st</sup> Trimester	Control & 2 <sup>nd</sup> Trimester	Control & 3 <sup>rd</sup> Trimester
I	NS	NS	NS
II	p>0.05	P<0.05	P<0.001
III	p>0.05	P<0.05	P<0.001

p>0.05: Not Significant, \*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant.

**Table 5:** Mean  $\pm$  SD of QRS Complex, QT Interval, QTc Interval and QRS frontal axis in subjects of Control and Study groups

Parameters	Control Mean $\pm$ SD	1 <sup>ST</sup> Trimester Mean $\pm$ SD	2 <sup>ND</sup> Trimester Mean $\pm$ SD	3 <sup>RD</sup> Trimester Mean $\pm$ SD
QRS Complex (sec)	0.08 $\pm$ 0.01	0.08 $\pm$ 0.01	0.08 $\pm$ 0.01	0.08 $\pm$ 0.01
QT Interval (sec)	0.35 $\pm$ 0.02	0.35 $\pm$ 0.02	0.35 $\pm$ 0.02	0.36 $\pm$ 0.01
QTc Interval (sec)	0.38 $\pm$ 0.01	0.39 $\pm$ 0.01	0.40 $\pm$ 0.01	0.41 $\pm$ 0.01
QRS frontal axis (degree)	64.56 $\pm$ 7.66	60.48 $\pm$ 11.05	55.70 $\pm$ 12.61	45.4 $\pm$ 22.54

**Table 6:** Test of Significance for QRS Complex, QT Interval, QTc Interval and QRS frontal axis using Z Statistics between Control and Study groups

Parameters	Control & 1 <sup>ST</sup> trimester P-Value	Control & 2 <sup>ND</sup> trimester P-Value	Control & 3 <sup>RD</sup> trimester P-Value
QRS Complex (sec)	0.067	0.105	0.091
QT Interval (sec)	0.401	0.384	0.293
QTc Interval (sec)	< 0.0001***	< 0.0001***	< 0.0001***
QRS frontal axis (degree)	0.032*	<0.0001***	<0.0001***

p>0.05: Not Significant, \*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant

**Table 7:** Test of Significance for QRS Complex, QT Interval, QTc Interval and QRS frontal axis using Z Statistics within the subgroups of Study group

Parameters	1 <sup>ST</sup> & 2 <sup>ND</sup> Trimesters P-Value	1 <sup>ST</sup> & 3 <sup>RD</sup> Trimesters P-Value	2 <sup>ND</sup> & 3 <sup>RD</sup> Trimesters P-Value
QRS Complex (sec)	0.105	0.833	0.849
QT Interval (sec)	0.384	0.027	0.030
QTc Interval (sec)	0.0001***	0.0001***	0.0001***
QRS frontal axis (degree)	0.051	0.0001***	0.0001***

p>0.05: Not Significant, \*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant

**Table 8:** Mean $\pm$ SD of T wave in subjects of Control and Study groups

Parameters		Control Mean $\pm$ SD	1 <sup>ST</sup> Trimester Mean $\pm$ SD	2 <sup>ND</sup> Trimester Mean $\pm$ SD	3 <sup>RD</sup> Trimester Mean $\pm$ SD
T wave	Duration(sec)	0.19 $\pm$ 0.04	0.19 $\pm$ 0.04	0.18 $\pm$ 0.03	0.19 $\pm$ 0.04
	Amplitude (mv)	2.72 $\pm$ 1.01	2.70 $\pm$ 1.11	2.78 $\pm$ 0.89	2.45 $\pm$ 0.93

**Table 9:** Test of significance for T wave using Z Statistics between Control and Study groups

Parameters		Control & 1 <sup>ST</sup> trimester P-Value	Control & 2 <sup>ND</sup> trimester P-Value	Control & 3 <sup>RD</sup> trimester P-Value
T wave	Duration (sec)	0.275	0.833	0.275
	Amplitude (mv)	0.928	0.920	0.920

p>0.05: Not Significant, \*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant

## Discussion

In our study there was a statistically significant increase in the heart rate in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy as compared to non-pregnant women.

A progressive increase in heart rate is observed as age of pregnancy advances. Heart rate increased by approximately 15% in the 5<sup>th</sup> week. It increased after 8<sup>th</sup> week to a maximum of approximately 85-90 beats per minute. In the last trimester of pregnancy, there is a chance of an increase of 10-20 beats per min. The heart rate of a pregnant woman steadily increased throughout pregnancy [10]. The increase in the heart rate is linked to autonomic nervous system changes that produce alterations in cardiac autonomic modulation [11]. Failure of these adaptations may result in pregnancy related complications.

The increase in heart rate may have been triggered to maintain the cardiac output in a state of relative hypovolemia [12]. The increase in heart rate was due to a decrease in vagal baroreflex as well as a decrease in parasympathetic tone. The increase in heart rate mainly during third trimester of pregnancy compensates for the fall in the stroke volume resulting from caval compression [13]. The observations made in our study are in agreement with the findings of other studies [14].

In our study, there was no statistically significant difference in measurements of P wave amplitude and duration when compared between the control and the study groups. PR interval was shown to be statistically significantly decreased in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy as compared to control group. There was also a statistically significant decrease in 2<sup>nd</sup> & 3<sup>rd</sup> trimesters of pregnancy compared to 1<sup>st</sup> trimester of pregnancy and in 3<sup>rd</sup> trimester of pregnancy when compared to 2<sup>nd</sup> trimester of pregnancy.

The decrease in PR interval during pregnancy could be due to shortening of A-V conductance with respect to tachycardia that accompanies during pregnancy. Similar report was made by Joseph E Carruth et al. In their study, they found that mean PR interval was shorter at 3<sup>rd</sup> trimester when compared

to 1<sup>st</sup> and 2<sup>nd</sup> trimesters of normal pregnancy & it was statistically significant [15].

In the present study, there was a statistically significant increase in occurrence of Q wave in the 2<sup>nd</sup> and 3<sup>rd</sup> trimesters when compared to 1<sup>st</sup> trimester of pregnancy and the control group. These changes may be either due to an increase in the circulating vasopressor agents or may reflect diaphragmatic changes that have been associated with pregnancy [16]. The frequent occurring of Q wave during pregnancy when compared to normal non-pregnant women may be due to altered position of the heart [17].

QRS Complex measurement had no statistically significant difference in duration either when compared between the study and the control groups, nor between the subgroups within study groups. In the present study, ST segment depression was noticed in 4 subjects in 2<sup>nd</sup> trimester and 6 subjects in the 3<sup>rd</sup> trimester of pregnancy. There was no change in ST segment in the subjects of control group and 1<sup>st</sup> trimester of pregnancy. There was no statistically significant difference between the control and the study groups or between subgroups within study group. One of the causes for ST segment depression during pregnancy may be due to electrolyte imbalance such as hypokalaemia as a result of persistent vomiting, but the prevalence of hypokalemia and hyperemesis was less. It has been suggested that transient ST segment depression is associated with anxiety which may be a provoking stimulus and that can be attributed to an endogenous hypersensitivity. One of the mechanisms by which adrenaline induces hypersensitivity is by increasing oxygen demand by the increased muscular action and coronary dilation. Our findings are in accordance with the observations made by others [18-20].

In our study, there was no statistically significant increase or decrease in the QT interval when compared between the control and study groups or within the study group. QTc Interval in electrocardiogram reflects the time taken for depolarization and repolarization in the ventricular myocardium. The QT interval when corrected for heart rate is QTc. It must be emphasized that the surface

electrocardiographic QTc interval reflects complex and interrelated aspects of cardiac electrophysiology, cardiac geometry, torso shape, tissue impedance and biological signal processing.

In the present study, it was found that there was a statistically significant increase in QTc interval in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy when compared to control group. There was also a statistically significant increase in QTc interval in 2<sup>nd</sup> and 3<sup>rd</sup> trimesters when compared to 1<sup>st</sup> trimester of pregnancy and also in the 3<sup>rd</sup> when compared to 2<sup>nd</sup> trimester of pregnancy. It is first necessary to determine the normal range of QTc interval in healthy pregnant women. It seemed possible that the altered circulatory dynamics during pregnancy might have some effect on its duration. It appears that the physical and emotional stress during 9 months of pregnancy may be a factor in triggering heart rhythm disorders in some vulnerable women. An increase in the QTc interval may be due to tachycardia. They must be considered as a complex consequence with changes in regulatory mechanisms during normal pregnancy [21].

In the present study, there was no statistically significant change in the T wave amplitude and duration when compared between the control and the study group, or between the subgroup within the study group. During pregnancy, there is an increase in blood volume, which in turn results in a temporary increase in workload on heart. Eventually temporary ischemia develops which is represented by T wave inversion.

It has been suggested that in normal pregnant women, flat or negative T waves may be observed during pregnancy and this fact should be kept in mind while interpreting electrocardiograms of pregnant women. With increase in gestational age, position of heart changes from vertical to intermediate indicating that heart shifted to left as gestation proceeds. The change in the electrical axis may be due to raise in the diaphragm during pregnancy. The changes in the left ventricular size and mass and associated increased volume may cause the apical impulse to be displaced to the left. Elevation and rotation of the heart resulting from the enlarging uterus and left axis shift in early pregnancy can be explained from the fact that there is an increased blood volume which in turn causes left ventricular load.

## Conclusion

Heart rate was increased in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy, a decrease in PR interval was seen all

the trimesters of pregnancy, Occurrence of Q wave in leads II and III showed an increase in 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy, ST segment depression was noticed in 4 subjects in 2<sup>nd</sup> trimester and 6 in 3<sup>rd</sup> trimester of pregnancy, QTc interval showed an increase in pregnant women in 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy. This knowledge may be helpful in the prevention of gestational complications associated with an inadequate maternal hemodynamic adaptation.

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Community and Public Health Nursing	Triannual	5500	5000	430	391
Dermatology International	Semiannual	5500	5000	430	391
Gastroenterology International	Semiannual	6000	5500	469	430
Indian Journal of Agriculture Business	Semiannual	5500	5000	413	375
Indian Journal of Anatomy	Bi-monthly	8500	8000	664	625
Indian Journal of Ancient Medicine and Yoga	Quarterly	8000	7500	625	586
Indian Journal of Anesthesia and Analgesia	Monthly	7500	7000	586	547
Indian Journal of Biology	Semiannual	5500	5000	430	391
Indian Journal of Cancer Education and Research	Semiannual	9000	8500	703	664
Indian Journal of Communicable Diseases	Semiannual	8500	8000	664	625
Indian Journal of Dental Education	Quarterly	5500	5000	430	391
Indian Journal of Emergency Medicine	Quarterly	12500	12000	977	938
Indian Journal of Forensic Medicine and Pathology	Quarterly	16000	15500	1250	1211
Indian Journal of Forensic Odontology	Semiannual	5500	5000	430	391
Indian Journal of Genetics and Molecular Research	Semiannual	7000	6500	547	508
Indian Journal of Hospital Administration	Semiannual	7000	6500	547	508
Indian Journal of Hospital Infection	Semiannual	12500	12000	938	901
Indian Journal of Law and Human Behavior	Semiannual	6000	5500	469	430
Indian Journal of Legal Medicine	Semiannual				
Indian Journal of Library and Information Science	Triannual	9500	9000	742	703
Indian Journal of Maternal-Fetal & Neonatal Medicine	Semiannual	9500	9000	742	703
Indian Journal of Medical & Health Sciences	Semiannual	7000	6500	547	508
Indian Journal of Obstetrics and Gynecology	Bi-monthly	9500	9000	742	703
Indian Journal of Pathology: Research and Practice	Monthly	12000	11500	938	898
Indian Journal of Plant and Soil	Semiannual	65500	65000	5117	5078
Indian Journal of Preventive Medicine	Semiannual	7000	6500	547	508
Indian Journal of Research in Anthropology	Semiannual	12500	12000	977	938
Indian Journal of Surgical Nursing	Triannual	5500	5000	430	391
Indian Journal of Trauma & Emergency Pediatrics	Quarterly	9500	9000	742	703
Indian Journal of Waste Management	Semiannual	9500	8500	742	664
International Journal of Food, Nutrition & Dietetics	Triannual	5500	5000	430	391
International Journal of Neurology and Neurosurgery	Quarterly	10500	10000	820	781
International Journal of Pediatric Nursing	Triannual	5500	5000	430	391
International Journal of Political Science	Semiannual	6000	5500	450	413
International Journal of Practical Nursing	Triannual	5500	5000	430	391
International Physiology	Triannual	7500	7000	586	547
Journal of Animal Feed Science and Technology	Semiannual	78500	78000	6133	6094
Journal of Cardiovascular Medicine and Surgery	Quarterly	10000	9500	781	742
Journal of Forensic Chemistry and Toxicology	Semiannual	9500	9000	742	703
Journal of Geriatric Nursing	Semiannual	5500	5000	430	391
Journal of Global Public Health	Semiannual				
Journal of Microbiology and Related Research	Semiannual	8500	8000	664	625
Journal of Nurse Midwifery and Maternal Health	Triannual	5500	5000	430	391
Journal of Organ Transplantation	Semiannual	26400	25900	2063	2023
Journal of Orthopaedic Education	Triannual	5500	5000	430	391
Journal of Pharmaceutical and Medicinal Chemistry	Semiannual	16500	16000	1289	1250
Journal of Practical Biochemistry and Biophysics	Semiannual	7000	6500	547	508
Journal of Psychiatric Nursing	Triannual	5500	5000	430	391
Journal of Social Welfare and Management	Triannual	7500	7000	586	547
New Indian Journal of Surgery	Bi-monthly	8000	7500	625	586
Ophthalmology and Allied Sciences	Triannual	6000	5500	469	430
Otolaryngology International	Semiannual	5500	5000	430	391
Pediatric Education and Research	Triannual	7500	7000	586	547
Physiotherapy and Occupational Therapy Journal	Quarterly	9000	8500	703	664
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# A Study of Autonomic Function Tests in Undergraduate Medical Students

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## Abstract

*Context:* Among undergraduate medical students, the relation between mood disturbances associated with the psychological or physical stressors induced by studying and the medical training and the modifications of ANS has been recently reported. *Aims:* To examine the impact of stress of medical college environment among the undergraduate medical students in terms of altered parasympathetic functions and altered sympathetic functions. *Settings and design:* A prospective study was carried out in the department of Physiology, Lt. BRKM Government Medical College. *Methods and Material:* Present study was conducted among randomly selected 90 first year medical students. The students underwent evaluation at the start of the course, just before the terminal examination and after the terminal examination. *Statistical Analysis:* The data was analyzed using mean values and  $\pm$ two standard deviations. Students't test was applied and p value was calculated which was considered significant if it is less than 0.05. *Results:* "Heart rate changes", "Heart rate response to Valsalva maneuver", "Heart rate response to deep breathing", "orthostatic test" were found to be significantly changed during the examination period. There was significantly lesser variation in the female group for all above mentioned parameters. Rise of Diastolic blood pressure in handgrip test was not found to be significantly changed during the examination period. *Conclusion:* We conclude that undergraduate students preparing for examination represents a widely employed model of a real life stressor, raising resting blood pressure and heart rate. There is a shift towards sympathetic activation and vagal withdrawal at rest and by enhanced vasomotor and reduced cardiac sympathetic standing.

**Keywords:** Autonomic Function Tests; Medical Students; Sympathetic Activity.

## Introduction

The importance and nature of complex, multifarious mechanisms linking environmental stressors like psychological stress to arterial hypertension have extensively been studied and explored in simulated conditions. In models experimented in the laboratory, it was found that there was increase in heart rate and the arterial pressure when exposed to mental stress [1].

With the alteration in normal balance in functioning of sympathetic and parasympathetic nervous system can have many presentations in the affected persons.

The subject may become dizzy; lose his balance, faint, tachycardia, bradycardia, hypotension, poor exercise tolerance, dizziness, sleep disorders, sweating, blurred vision, numbness and tingling [2].

It has been found that autonomic nervous system (ANS) is affected by many factors which may be operating at patient level like diabetes, uremia, amyloidosis, various metabolic or toxic neuropathy, GB syndrome, leprosy, various hereditary neuropathies, hepatic porphyria, botulism, Lambert Eaton myasthenic syndrome, para neoplastic effects like cancer lung [3].

The ANS can also be affected environmental factors like stress, smoking, air pollution etc. medical students are exposed to stress during study period from academic things like achievement, peer pressure etc, physical factors like hostel facilities etc, emotional factors, and social factors [4].

The ANS modulates the electrical and contractile activity of the myocardium via the interplay of the sympathetic and parasympathetic activity. The regulatory system includes simple reflexes in the brainstem and spinal cord as well as complex long

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circuit pathways through the higher brain centers. Most of the higher centers of regulatory functions are located in the reticular formation of brainstem and nuclei of hypothalamus with a great influence from the limbic areas and the cerebellum. In actuality, every activity of the nervous system is associated with autonomic function and every activity of cortex has an effect upon the autonomic regulatory controls [5].

Medical education has been found to be associated with stress especially during the first year. If the episodes are repeated then it leads to increased blood pressure. Those with high blood pressure show late recovery to normal blood pressure compared to those who had normal blood pressure [6].

If the stress is repeated several times, then the subjects are exposed to the risk of cardiovascular diseases. Stress leads to ANS reactivity which in turn leads to high blood pressure and the high blood pressure leads to increased risk of cardiovascular diseases [7].

Over activity of sympathetic nervous system also plays an important role in pathogenesis of neurogenic hypertension in young individuals. Stress influences the hypothalamus via cerebral limbic system and causes changes in heart rate variability through autonomic nervous system [3].

Among undergraduate medical students, the relation between mood disturbances associated with the psychological or physical stressors induced by studying and the medical training and the modifications of ANS has been recently reported. Some studies done on medical students have found positive correlation with stress associated with medical training and the changes in ANS. Finding from studies that examined the effects of real life stressor like university examination on the cardiovascular indices using spectral analysis of Heart Rate Variability (HRV) in healthy medical students were suggestive of cardiac sympathetic activation [6].

Present study aims to examine the impact of stress of medical college environment among the undergraduate medical students in terms of altered parasympathetic functions and altered sympathetic functions.

## Material and Methods

A prospective study was carried out in the department of Physiology among randomly selected 90 first year medical students. The students underwent evaluation at the start of the course, just

before the terminal examination and after the terminal examination.

All healthy first year medical students who were mentally and physically fit were included in the study. Students found suffering from any systemic illness or under any treatment were excluded after general and systemic examination.

A clearance from Ethics Committee was taken for the present study. Participant was made aware about the tests to be carried out. An informed consent obtained from the participants. Baseline data was recorded.

Students were forbidden to have any energetic or exciting drinks like tea etc or tobacco related products at least 12 hours before initiating test. They were allowed to have two hours before the test the light breakfast. Before starting the test the students were asked to relax for half an hour in supine position. The resting heart rate was recorded on a standard ECG from lead II, at a paper speed of 25 mm/sec. BP was measured and recorded with sphygmomanometer as per standard guidelines.

All the students underwent tests like "heart rate response to Valsalva maneuver", "heart rate response during deep breathing", "heart rate response to standing", "BP response tests", "blood pressure response to standing" ("fall in systolic blood pressure"), "blood pressure response to sustained handgrip" ("rise in diastolic blood pressure") and "cold pressor test."

The abnormal values of "heart rate response to Valsalva maneuver" was 1.1 or less, "heart rate response during deep breathing" was 10 beats per minute or less, "heart rate response to standing" was 1 or less, "blood pressure response to standing" ("fall in systolic blood pressure") was 30 mmHg or more and "blood pressure response to sustained handgrip" ("rise in diastolic blood pressure") was 10 mmHg or less.

The data was entered in the statistical software. The data was analyzed using mean values and  $\pm$  two standard deviations. Students' t test was applied and p value was calculated which was considered significant if it is less than 0.05.

## Results

Table 1 shows comparison of "heart rate response to standing" 30:15 ratios. Heart rate changes were found to be significant during the examination period. There was significantly lesser variation in the female group as compared to the male subjects.

Table 2 shows comparison of “heart rate response to Valsalva maneuver”. Heart rate response to Valsalva maneuver was found to be significantly changed during the examination period. There was lesser change in ANS functions in female subjects as compared to the male group.

Table 3 shows comparison of “heart rate response to deep breathing”. Heart rate response to deep breathing was found to be significantly changed during the examination period. The results were pointing towards better ANS functions in female subjects as compared to the male group.

Table 4 shows comparison of “diastolic blood pressure in handgrip test”. Rise of Diastolic blood pressure in handgrip test was not found to be significantly changed during the examination period.

There was lesser increase in diastolic blood pressure in female subjects as compared to the male group.

Table 5 shows comparison of “blood pressure response” (“fall in systolic blood pressure”) in response to standing. “Blood pressure response to standing” was significant in male group due to “increased sympathetic discharge”. There was lesser blood pressure increase in the female subjects. There were significant changes in orthostatic test during the exams.

Table 6 shows “Blood pressure response to cold pressor test”. There was significant increase in diastolic blood pressure during exams. There was a significant change in diastolic blood pressure in males as compared to females.

**Table 1:** Comparison of “heart rate response to standing” 30:15 ratios

Group	Mean ± SD	T value	P value
Before exams	1.4062±0.3058	-1.989	0.0456
After exams	1.3022±0.2688		
Female	1.2717±0.2133	-1.746	0.001
Male	1.1942±0.02539		

**Table 2:** Comparison of “heart rate response to Valsalva maneuver”

Group	Mean±SD	T value	P value
Before exams	1.7006±0.4870	-2.435	0.0178
After exams	1.4434±0.3462		
Female	1.4632±0.3752	1.3610	0.0462
Male	1.4012±0.262		

**Table 3:** Comparison of “heart rate response to deep breathing”

Group	Mean±SD	T value	P value
Before exams	24.2466±7.1638	3.4725	0.0324
After exams	27.4331±9.0481		
Male	26.2466±7.1368	0.6358	0.0278
Female	28.5134±6.8152		

**Table 4:** Comparison of “diastolic blood pressure in handgrip test”

Group	Mean±SD	T value	P value
Before exams	30.3125±10.4370	3.4725	0.0324
After exams	30.5625±11.1787		
Male	30.3925±11.1787	7.36835	0.5683
Female	22.4782±12.3		

**Table 5:** Comparison of “blood pressure response” (“fall in systolic blood pressure”) in response to standing

Group	Mean±SD	T value	P value
Before exams	2.00±3.1315	2.791	0.0089
After exams	4.3125±4.3436		
Male	4.672±4.7536	2.4130	0.0137
Female	2.173±4.1635		

**Table 6:** "Blood pressure response to cold pressor test"

Group	Mean±SD	T value	P value
Before exams	4.4598±6.6312	3.2645	0.0111
After exams	8.6690±11.3073		
Male	8.8754±7.4785	3.7758	0.02
Female	4.2675±6.5378		

## Discussion

In healthy young students, a real life stressor in the form of adjusting to the demands of medical training in first year undergraduate medical students, significantly impacts the autonomic inputs of cardiovascular regulation.

Lucini D et al [3] examined the relationship between stress and cardiac autonomic function among medical students studied the effect of appearing in a university examination on cardiac autonomic indices and found results similar to the present study.

Goldstein IB et al [4] studied ambulatory blood pressure and heart rate in healthy paramedics during a workday and a non workday. They found that high levels of stress have characteristically been associated with elevated blood pressure. From studies conducted in the work place, occupational stress has been suggested as a major cause of hypertension and cardiovascular disease.

Present study showed that during the stress days, the findings of the various autonomic function tests are suggestive of altered autonomic homeostasis with a shift towards cardiac sympathetic activation and vagal withdrawal with reduced baroreflex gain. The findings of the present study are agreement with the previous studies that there is stress induced rise in the blood pressure [3,4].

A marked elevation in systolic and diastolic arterial pressure observed in majority of subjects as found during handgrip test, the pre-exam period result was 30.31±10.43 showed 30.56±11.17. So these findings are pointing towards decreased baroreflex discharges and increase in sympathetic system. On the stress days active standing induced lesser tachycardia.

The finding of the present study of higher sympathetic activity and possibly vagal withdrawal in the stress (examination) group is in agreement with an earlier study on the effects of acute stress in healthy young undergraduate medical students [7].

Khaliq F et al [8] concluded that medical training was highly stressful particularly for those who are beginning their medical education. The present study

found a rise in blood pressure during CPT though not increased to level to be classified as abnormal but there was significant change in the test group as compared to control group.

Srinivasan K et al [2] in their study concluded that in medical students, who obtained scores in upper quartile of a self report stress scale there was an impaired cardiac autonomic regulation. There was significant correlation between stress scores and changes in heart rate on spectral analysis. Their findings were suggestive of a shift in cardiac autonomic regulation towards sympathetic activation in response to real life stressors. Their findings were in conformity with result from the present study findings. The present study findings are in agreement with previous studies [9] done on cardiac autonomic regulation towards increased sympathetic discharges in the test group under stress.

A rise of 20/20 mmHg or more during CPT has been documented to be an abnormal response [10]. In present study though the increase in diastolic blood pressure was significant in test group, pointing to heightened sympathetic responses, values were within the normal range and is in agreement with the previous studies [3,4,6].

Separate data was collected from post examination period. The main aim was to determine if the changes in autonomic nervous system during stress period does persists after stress period in determining if there is any additive effects of post examination ANS results to changes observed in future studies on the same group during their clinical posting. Supe AN et al [11] demonstrated that stress in medical students is common and is process oriented. It is more in second and third year. So, results from post examination period help in future analyses of same study group or other and determining if these changes have any additive effects on stress perceived and changes occurring in ANS as they enter from first year to second year and intense clinical training during the final year.

Lucini D et al [3] have found significant correlation between cortisol levels and indices of autonomic modulation of sino-atrial node suggesting that common mechanisms underlay gradual

individualized neural and humoral responses to stressors. Thus at the periphery autonomic adrenal innervations could be implicated, so further studies of autonomic function along with measurement of plasma or salivary cortisol are recommended. Present study did not include biochemical parameters of stress such as plasma or salivary cortisol.

The results of present study may help understand the pattern of response to the examination stress and enable development of strategies that will assist the students to handle the stress in a more efficient manner. There are many strategies among them one could be which enable the students to face the examination or to increase the number of periodic weekly, monthly tests that will give feedback to the students and guide them to improve deficiencies in learning. The findings of the present study have implications for studies that evaluate the role of stress on psychology, endocrine system and impaired neuronal activity in humans. To confirm whatever pattern of response found needs to be evaluated on larger scale and one longer period of time.

### Conclusion

We conclude that undergraduate students preparing for examination represents a widely employed model of a real life stressor, raising resting blood pressure and heart rate. There is a shift towards sympathetic activation and vagal withdrawal at rest and by enhanced vasomotor and reduced cardiac sympathetic standing. These alterations of autonomic homeostasis suggests a future link between real life stressor and various manifestations of cardiovascular disorders such as increased arterial blood pressure and reduction in protective baroreflex.

### Key Messages

The findings of the present study have implications for studies that evaluate the role of stress on psychology, endocrine system and impaired neuronal activity in humans.

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# A Study to Evaluate Body Composition and Lipid Profile in Postmenopausal Women

Shireen Swaliha Quadri<sup>1</sup>, Manjunath M.L.<sup>2</sup>

## Abstract

**Introduction:** Menopause is physiological event in women's life and is associated with unfavourable changes in body composition and abdominal fat distribution as well more atherogenic lipid profile. Therefore, the present study was undertaken to study the body composition and lipid profile in postmenopausal women. **Materials and Methods:** Sixty-nine postmenopausal women were selected after their consent and divided into two groups. Group-I consists of 32 postmenopausal women within 5 years of menopause and Group-II consists of 37 postmenopausal women after 5 years of menopause. The body composition parameters were measured by classical anthropometry and skin fold callipers and lipid profile parameters were analysed by enzymatic method. Institutional ethical clearance was obtained. **Statistical Analysis:** Data was expressed as mean $\pm$ SD. Student's unpaired 't' test was used for analysis. p value less than 0.01 was considered statistically significant. **Results:** The mean values of body Weight, BMI, BSA, WC, HC, FM, FFM, FMI and MM were more in Group-I compared to Group-II and WHR and BF% were more in Group-II compared to Group-I. These variations were not statistically significant. The lipid profile parameters i.e. TC, TG, HDL-C, LDL-C, VLDL-C, TC/HDL in Group-II subjects were found to be nonsignificantly higher when compared to Group-I subjects. **Discussion and Conclusion:** The increase in the lipid profile in Group-II subjects may be due to increased intra-abdominal adipose tissue. These changes may be due to hormonal changes and depend on physical activity, life style, diet, smoking, alcohol consumption, ethnicity and genetic make-up of individual rather than on duration after menopause.

**Keywords:** Menopause; Body Composition; Lipid Profile.

## Introduction

Menopause is a natural event in the ageing process and signifies the end of reproductive years with cessation of cyclic ovarian functions as manifested by cyclic menstruation. It is heralded by menopausal transition, a period when the endocrine, biological and clinical features of approaching menopause begins [1]. The hormonal changes associated with menopause i.e. low plasma levels of oestrogen and marked increase in LH and FSH levels exerts a significant effect on plasma lipids and lipoproteins [2].

It was reported that, the body mass index (BMI) matched obese postmenopausal women have significantly higher waist circumference (WC), waist hip ratio (WHR) and intra-abdominal fat volume compared with premenopausal women. Total Cholesterol (TC) and Triglyceride (TG) were

significantly higher and HDL-C was significantly lower in postmenopausal women. When age matched pre- and postmenopausal women were compared only TC was significantly higher in postmenopausal group [3]. However, older postmenopausal women (>50years) had significantly higher WC and WHR compared with younger postmenopausal women (<50years).

There was no difference in total body fat-free or appendicular skeletal muscle mass in healthy premenopausal women and early postmenopausal women. In contrast, total body fat mass was 28% higher and percentage fat 17% higher in postmenopausal women compared with premenopausal women. Postmenopausal women had a 49% greater intra-abdominal and a 22% greater abdominal subcutaneous fat area compared to premenopausal women. The menopause related difference in intra-abdominal fat persisted after statistical adjustment for age and total body fat mass,

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whereas no difference in abdominal subcutaneous fat was noted. A similar pattern of differences in total and abdominal adiposity was noted in sub samples of pre- and postmenopausal women matched for age or fat mass [4].

In healthy women, during the time from premenopausal to first year Postmenopausal examinations, the changes in LDL-C, TG, and BMI were larger than those between first and fifth year Postmenopausal examinations [5]. There have been studies regarding the effect of menopause on body weight, fat distribution, total fat%, and also on lipid profile, but most of the studies are conducted on western population. As Indian population differs in body composition and lifestyle, this study was undertaken to evaluate the effect of duration of menopause on body composition parameters and lipid profile parameters in postmenopausal women.

## Materials and Methods

The present study was conducted in the department of physiology, Shimoga institute of Medical Sciences, Sagar Road, Shivamogga. Sixty-nine postmenopausal were selected after their consent. The institutional ethical clearance was obtained. The participants were divided into two groups as, Group-I consists of 32 postmenopausal women within 5 years of menopause and Group-II consists of 37 postmenopausal women after 5 years of menopause. All healthy postmenopausal women, who attained menopause by natural means were included. Postmenopausal women who have undergone hysterectomy, diabetic, hypertensive, on hormone replacement therapy, lipid lowering drugs & with H/O Gynaecological & hormonal disorders were excluded.

### *Measurement of Physiological Parameters*

Height was measured on a wooden stadiometer, bearing a flat stand and a vertical surface with marking in centimetres. A sliding head piece was used for accurate work. Height was recorded and expressed in centimetres. Body weight (Wt) of all the subjects was measured by using weighing scale and expressed in kilogram. Body surface area (BSA) in Sq. Mts ( $m^2$ ) was calculated by Duboi's nomogram. The body mass index was derived by Quetelet's index from body weight (kg)/height ( $m^2$ ). Waist circumference was measured at narrowest part of torso with plastic tape. Hip circumference was measured at maximal extension of buttocks with

plastic tape. Waist-Hip Ratio was calculated by dividing waist circumference by hip circumference.

Body fat percentage was calculated by using skin fold callipers. A skin fold callipers is a device which measures the thickness of a fold of skin with its underlying layer of fat. By measuring at key locations it is possible to estimate the total percent of body fat of a person. The callipers used is scientifically developed and calibrated. The instrument has springs which exerts certain pressure on skin fold ( $10g/mm^2$ ) and measures the thickness in millimetres.

The fold of skin with its underlying layer of fat was pulled out and grasped in the fingers of left hand, while holding the callipers in the right hand, the jaws of callipers were held about one fourth inch from the fingers of the left hand, which continues to hold the fold of the skin. The trigger of the callipers was released so that the entire force of the jaws was on the skin folds.

The skin fold thickness was measured on the left side at four sites such as Back of the mid-arm (Triceps), Front of the mid-arm (Biceps), Below the shoulder blade on the back (sub scapular) and Waist area above hip bone (suprailiac). The measurement was taken in all four areas & added together, and then body fat percentage was determined from the chart in the instruction manual.

The fat mass was calculated as  $FM = Wt / 100 \times B\%F$  and expressed in kilograms. Fat free mass was calculated and expressed in kilograms as  $FFM = \text{Weight} - \text{fat mass}$ . Fat mass index was calculated from fat mass in (kg)/Height in ( $m^2$ ). Muscle Mass was calculated by knowing fat free mass as  $MM = 50\%$  of FFM.

### *Measurement of Lipid Profile Parameters*

After overnight fasting 2ml of venous blood was collected from each subject and centrifuged at 3000rpm for 15min to obtain the serum which was used for analysis.

The level of Triglyceride, Total-cholesterol, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol were measured using semi-automated analyser (Erba star 21 plus) using commercially available kits.

### *Statistical Analysis*

Data was expressed as mean  $\pm$  SD. The student's unpaired 't' test was used to analyse the variations in body composition and lipid profile between two groups of postmenopausal women. P value less than 0.05 was the level of significance.

**Results**

The mean ± SD of age of subject was found to be 48±3.5yrs in Group-I and 58±7.8yrs in Group-II, the mean duration after menopause was 2.81±1.43yrs in Group-I and 13.73±6.34yrs in Group-II.

The weight of the subject was 50.91±11.35kg in Group-I and 49±8.2kg in Group-II. The BMI of the subject was 23.3±4.86kg/m<sup>2</sup> in Group-I and 22.42±3.60 kg/m<sup>2</sup> in Group-II. The BSA of the subject was found to be 1.4±0.10m<sup>2</sup> in Group-I and 1.4±0.1m<sup>2</sup> in Group-II. WC of the subject was found to be 75.31±12.20cms in Group-I and 75±9.1cms in Group-II. The difference in all these parameters were statistically insignificant (p>0.05) when compared between two groups (Table 1).

The HC of the subject was 95.6±11.30cms in Group-I and 94±8.6cms in Group-II. The WHR of the subject was 0.79±0.07 in Group-I and 0.80±0.1 in Group-II. The BF% of the subject was 29.5±5.82% in Group-I and 30.1±3.77% in Group-II. The FM of the subject was 15.6±6.46 kg in Group-I and 15±3.91kg in Group-II. The difference in all these parameters were also statistically insignificant (p>0.05) when compared between two groups (Table 1).

The FFM of the subject was 35.4±5.28 kg in Group-I and 34.2±4.91kg in Group-II. The FMI of the subject was 7.12±2.86 kg/m<sup>2</sup> in Group-I and 6.85±1.81kg/

m<sup>2</sup> in Group-II. The MM of the subject was 17.7±2.66kg in Group-I and 17.1±2.46kg in Group-II. MM was more in Group-I compared to Group-II. But these parameters differ nonsignificantly (p>0.05) when compared between two groups (Table 1).

*Lipid Profile Parameters*

The TC of the subject was 194±28.44 mg/dl in Group-I and 205.3±38.59 mg/dl in Group-II (Table 2). The TG of the subject was 189±55.07mg/dl in Group-I and 194±51.1mg/dl in Group-II. The HDL-C of the subject was 44.7±6.78mg/dl in Group-I and 46.13±6.05mg/dl in Group-II. HDL-C was more in Group-II compared to Group-I (Table 2). The LDL-C of the subject was 112±26.76mg/dl in Group-I and 120.17±35.86mg/dl in Group-II. LDL-C was more in Group-II compared to Group-I. The VLDL-C of the subject was 37.6±10.81mg/dl in Group-I and 38.88±10.23mg/dl in Group-II. VLDL-C was more in Group-II compared to Group-I (Table 2). The TC/HDL of the subject was 4.49±0.93 in Group-I and 4.56±1.10 in Group-II. TC/HDL was more in Group-II compared to Group-I. The HDL/LDL of the subject was 0.42±0.12 in Group-I and 0.42±0.14 in Group-II which is same in both groups. There was no statistical difference (t=0.0404, p>0.05). These parameters also did not show any significant difference (p>0.05) when compared between two groups (Table 2).

**Table 1:** Comparison of bodycomposition parameters of subjects between Group-I and Group-II. Values are mean±S.D. N=69

Parameters	Group-I	Group-II	t-value	p-value
Weight (kg)	50.91±11.35	49±8.2	0.8086	>0.05, NS
Height (cms)	148±6.84	148±9.01	0.0124	>0.05, NS
Body Mass Index (kg/m <sup>2</sup> )	23.3±4.86	22.42±3.60	0.8618	>0.05, NS
Body Surface Area (m <sup>2</sup> )	1.4±0.10	1.4±0.1	0.000	>0.05, NS
Waist Confence (cm)	75.31±12.20	75±9.1	0.1206	>0.05, NS
Hip Conference (cm)	95.6±11.30	94±8.6	0.6667	>0.05, NS
Waist Hip Ratio	0.79±0.07	0.8±0.1	0.4739	>0.05, NS
Body Fat (%)	29.5±5.82	30.1±3.77	0.5148	>0.05, NS
Fat Mass (kg)	15.6±6.46	15±3.91	0.4738	>0.05, NS
Free Fat Mass (kg)	35.4±5.28	34.2±4.91	0.9776	>0.05, NS
Fat Mass Index (kg/m <sup>2</sup> )	7.12±2.86	6.85±1.81	0.4926	>0.05, NS
Muscle Mass (kg)	17.7±2.66	17.1±2.46	0.9730	>0.05, NS

**Table 2:** Comparison of lipid profile parameters of subjects between Group-I and Group-II. Values are mean± S.D. N=69

Parameters	Group-I	Group-II	t-value	p-value
Total Cholesterol (mg/dl)	194±28.44	205.3±38.59	1.3659	>0.05, NS
Triglyceride (mg/dl)	189±55.07	194±51.1	0.3910	>0.05, NS
HDL (mg/dl)	44.7±6.78	46.13±6.05	0.9258	>0.05, NS
LDL (mg/dl)	112±26.76	120.17±35.86	1.0585	>0.05, NS
VLDL (mg/dl)	37.6±10.81	38.88±10.23	0.5049	>0.05, NS
TC/HDL	4.49±0.93	4.56±1.10	0.0404	>0.05, NS
LDL(mg/dl)	0.42±0.12	0.42±0.14	0.0000	>0.05, NS

## Discussion

In the present study, the mean values of body weight, BMI, BSA, WC, HC, FM, FFM, FMI and MM were more in Group-I compared to Group-II and WHR and BF% were more in Group-II compared to Group-I but not statistically significant. All the body composition parameters were within physiological limits in both the groups except BF% and FM which were found to be above the normal range. Insignificant difference in body composition parameters between Group-I and Group-II is similar to the study by Quinglong wang et al [6].

Increase in BF% and FM above physiological limit in both groups is similar to the study as reported by Douchi T et al [7]. Other body composition parameters were within physiological limit is supported by Edith T, Kevin PD, and Douglas RS [8]. Body fat and fat distribution are more dependent on age than on menopause, changes in fat free mass, including a postmenopausal decline in both soft lean tissue mass and bone mass are mainly menopause related [6]. So, in our study increase in BF%, FM above normal range in both the groups may be age related rather than duration of menopause and menopause itself. The previous studies have proved that lean body mass does not change in premenopausal women and decreases after the menopause and correlates with years since the onset of menopause. However, studies proving these data used modern sophisticated methods such as dual-energy X-ray absorptiometry or computed tomography for lean mass, bone and muscle mass assessment and not the classical anthropometry [9]. Body composition parameters also depend on physical activity, life style, dietary habits [10], ethnic group and genetic makeup of individual. This may be the reason for most of body composition parameters to be within physiological limits in our study.

In Group-I subjects TC, HDL-C, VLDL-C, TC/HDL were normal, LDL-C was above normal and TG was borderline high whereas in Group-II subjects HDL-C, VLDL-C were normal LDL-C was above normal and TC, TG were borderline high and TC/HDL was also high. However, it was found that lipid profile parameters in Group-II subjects were found to be statistically non-significant higher when compared to Group-I subjects. In our study no relation between lipid profile parameters and time since menopause i.e. duration after menopause could be established. This may indicate that the determining factors of lipid profile in postmenopausal women are physical activity, life style [11], diet, smoking, alcohol

consumption [12], ethnicity and genetic make-up of individual rather than duration after menopause. The changes in lipid profile occur at menopausal transition itself under the influence of hormonal change.

## Conclusion

The increase in the lipid profile in Group-II subjects may be due to increased intra-abdominal adipose tissue. These changes may be due to hormonal changes and depend on physical activity, life style, diet, smoking, alcohol consumption, ethnicity and genetic make-up of individual rather than on duration after menopause.

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# A Comparative Study of Visual Reaction Time for Red and Green Colors in Medical Students

Siva Kumar Uppara<sup>1</sup>, Kiran Buge<sup>2</sup>, Sunita Nighute<sup>3</sup>

## Abstract

**Background:** RT (Reaction time) is the interval time between the presentation of a stimulus and the initiation of the muscular response to that stimulus. Aim of the study was to compare visual reaction time for Red and Green colors in the first year medical students. **Material & Methods:** Total 120 first year M.B.B.S students in age group of 17-24 yrs both male and females were selected. Visual Reaction time was recorded by using audiovisual Reaction time apparatus designed by Anand agencies Pune. The study was carried out in the research lab, Department Of Physiology, Vikhe Patil Medical College, between 3 to 5 pm. **Result:** Data was analyzed by unpaired "t" test we found that visual Reaction time for Red and Green colors were statistically significant both in male and female ( $p < 0.05$ ) visual reaction time for red color light is faster than green color light both in males and females. **Conclusion:** Thus our study results showed visual reaction time for green color was significantly more than red color. This could be because individual color mental processing time for green color is more than red color

**Keywords:** Green Color; Red Color; Reaction Time; Visual Reaction Time; RT: Reaction Time; VRT: Visual Reaction Time.

## Introduction

RT (Reaction time) is the interval time between the presentation of a stimulus and the initiation of the muscular response to that stimulus [1,2]. Human body has a tendency to produce responses for number of external environmental stimuli of different modalities. There is definite time period between application of stimulus and proper motor response. human body gives different responses to different sensory modalities at different speed. This plays essential role in day to day life as well as in emergency survival as while driving it is required to put break as fast as possible to vehicle when required. RT in response to a condition can remarkably effect our lives due to its practical implications. Fast RT can produce rewards (e.g. in sports) whereas slow RT can produce grave consequences (e.g. driving and road safety matters) [3,4].

Many factors have been exhibited to affect reaction time including gender, age, physical fitness, level of fatigue, distraction, alcohol, different colors, personality type, limb used for test, biological rhythm, and health and whether the stimulus is auditory or

visual. Prolonged reaction time denotes decreased performance [4].

Reaction time is having mainly two components : (1) Premotor time - which is time required for receiver to perceive stimulus, identifying and analyzing of stimulus, and decide the proper motor response (2). Motor time - it is time required to perform movement after selection of response [3].

There are three types of reaction time [3]. (1) Simple reaction time - reaction time from a task in which a single known response is produced when a single stimulus is presented (2) Recognition reaction time - here there are some stimulus that should be responded to and other that should not get response (3) Choice reaction time - reaction time for a task in which each response to be made is associated with a different stimulus.

The wave length and energy of each color varies with the intensity and this in turn affects human body in different ways. Several studies have been assumed to examine the influence of color on the simple reaction time. In few studies, reaction time has been revealed to be independent of wavelength while others have found that reaction time to red

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stimuli was less than that to green or blue stimuli [4]. The present study was under taken to confirm these findings.

Thus, this study was carried out to scientifically grant to the field of RT.

#### *Aims and Objectives*

To compare visual reaction time for red and green colors in the first year medical students.

### **Materials and Methods**

After obtaining approval from research and ethical committees, DVVPF's medical college a total 120 first year medical students of 2016 batch were selected and written informed consent were taken from all the participants.

The study was conducted in the research lab, Department Of Physiology, vikhe Patil Medical College, between 3 to 5 pm.

#### *Inclusion Criteria*

120 healthy medical students in age group of 17-24 yrs both male and females.

#### *Exclusion Criteria*

1. History of smoking, alcoholism
2. Those having any history of visual disorders,
3. History of any medications affecting cognitive performance will be excluded from study.
4. Those having any major illness in the present or past,
5. History of color blindness

Visual Reaction time was recorded by using audiovisual Reaction time apparatus designed by Anand agencies Pune. It works on 230 volts AC. The instrument is specially designed to measure reaction time in seconds [5].

All the participants were completely familiarized with the apparatus. All tests were done in quite room at normal room temperature.

Visual reaction time-The visual stimulus was provided in the form of green and red color light. Red and green color light visual stimulus was given separately. Subject was asked to press response switch as soon as the red or green color light blinks. 3 readings were taken; an average of 3 readings was taken as subject's best reading.

The data was statistically analyzed by using student unpaired t test.

**Table 1:** Comparison of visual reaction time for red and green colours in all participants in seconds

Colour	Number	Mean $\pm$ SD	'p' value
Red	120	0.186 $\pm$ 0.033	0.000**
Green		0.198 $\pm$ 0.034	

\*p<0.05 statistically significant \*\*p<0.001 statistically highly significant

Table 1 show that visual reaction time for red color light (0.186 $\pm$ 0.033) was significantly faster than in green color (0.198 $\pm$ 0.034) in all participants

**Table 2:** Comparison of visual reaction time for red and green colours in seconds in females

Sex	Number	Colour	Mean $\pm$ SD	'p' value
Females	60	Red	0.190 $\pm$ 0.028	0.003*
		Green	0.202 $\pm$ 0.032	

\*p<0.05 statistically significant \*\*p<0.001 statistically highly significant

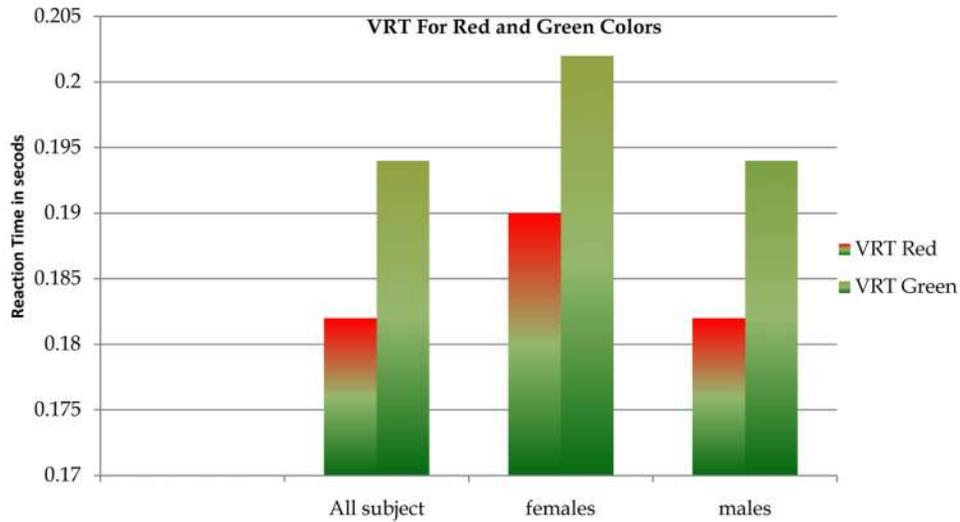
Table 2 show that visual reaction time for red color light was significantly faster (0.190 $\pm$ 0.028) than in green color light (0.202 $\pm$ 0.032) in females

**Table 3:** Comparison of visual reaction time for red and green colors in seconds in males

Sex	Number	Colour	Mean $\pm$ SD	'p' value
Males	60	Red	0.182 $\pm$ 0.037	0.002*
		Green	0.194 $\pm$ 0.036	

\*p<0.05 statistically significant \*\*p<0.001 statistically highly significant

Table 3 show that visual reaction time for red color light was significantly faster (0.182 $\pm$ 0.037) than in green color light (0.194 $\pm$ 0.036) in males



**Graph 1:** Comparison of VRT for Red and Green colour lights in all participants, females and males

Graph 1: showing faster VRT for Red colour light as compared to Green colour light in all participants, females and males

## Discussion

Reaction time is based on several factors like approach of the stimulus at the sensory organ, conversion of the stimulus by the sensory organ to a neural signal, neural transmissions and processing, muscular activation, soft tissue compliance, and the selection of an external measurement parameter [6]. The present study was aimed to compare visual reaction time for red and green colors in 1st year M.B.B.S medical students.

We compared visual reaction time for red and green color stimuli in all participants, 60 male and 60 female medical students. In our study we observed that Visual reaction time for Red and Green colors were statistically significant both in male and female. Table 1, Table 2 and Table 3, Graph 1, shows visual reaction time for red color light is faster than green color light both in males and females.

Few other studies also shown same finding that is Visual Reaction time to red light is faster was compared to the green light in both males and females [7,8].

This can be described on the basis of the Trichromatic Theory of color vision. when Tomita and Co-workers illuminated the retina with micro-electrode penetration of a single cones, They found that 74% of units peaked in the red spectrum, 10% in the green spectrum and 16% in blue spectrum [9].

## Conclusion

Thus our study results showed visual reaction time for green color was significantly more than red color. This could be because individual color mental processing time for green color is more than red color.

## Acknowledgement

Authors are thankful to the first M.B.B.S students involved in the study.

## Abbreviations

RT- Reaction Time

VRT- Visual Reaction Time

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## Association of Circadian Variation of Blood Pressure with Obesity in Healthy young Adult Males

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### Abstract

Blood pressure follows a circadian variation i.e. it falls at night during sleep (nocturnal dipping) and rises in the morning. Lack of nocturnal dipping predisposes to various cardiovascular diseases. Obesity is increasing day by day especially in younger age. It may be considered as a risk factor for various cardio metabolic diseases like hypertension, diabetes. The present study was designed to investigate the association of various obesity parameters with circadian variation of blood pressure as assessed by dipper state. 60 healthy young adults of age between 20 to 35 years were enrolled. Subjects with any H/o hypertension, cardiovascular, renal disorders were excluded. BMI was calculated. Waist circumference (WC) was measured in cms. Skinfold thickness was measured using Harpendent skinfold calipers and body fat percentage was calculated using Durnin-Womersley formula. 24 hours Ambulatory Blood Pressure was measured using Contec Ambulatory Blood Pressure Monitor (ABPM). ABPM was set to measure BP every 15 min during daytime and every 30-min in night time while sleeping. Subjects were divided into two groups according to their dipper profile, as defined: dippers (nocturnal decrease in systolic BP was  $\geq 10\%$  of daytime BP) and non-dippers (nocturnal decrease in systolic BP was  $<10\%$  of daytime BP). The average BMI, waist circumference and body fat percentage were significantly more in non-dippers than dippers. Non-dippers even if normotensives are at more risk of cardiovascular complications. Hence, ABPM should be performed in obese individuals to know their dipper state and assess the risk of cardiovascular complications associated with non-dipping.

**Keywords:** BMI; Waist Circumference; Body Fat Percentage; Ambulatory Blood Pressure Monitoring; Non Dipper.

### Introduction

Circadian rhythm may be defined as the intrinsic, regular fluctuation of a physiologic process. Like many other physiological processes in the body, BP also generally varies according to a circadian rhythm characterized by a reduction during sleep and an increase during wakefulness [1,2].

The decrease in BP during sleep is referred to as “nocturnal dipping” and is partly attributable to decrease in sympathetic output. Although arbitrary, a decrease of 10% to 20% in mean nocturnal BP (both systolic and diastolic) compared with mean daytime BP is considered normal. Conversely, an absence of nocturnal dipping, or non-dipping, is designated as a less than 10% decrease in nocturnal BP. Lack or diminished nocturnal dipping of BP is a strong, independent predictor of cardiovascular risk. The Ohasama study noted that on average, each 5%

deficiency in the normal decline in nocturnal BP was associated with an approximately 20% greater risk in cardiovascular mortality [3]. Many diseases are associated with diminished or absence of nocturnal dipping, including most secondary causes of hypertension, chronic kidney disease, diabetes, older age, resistant hypertension, and obstructive sleep apnea (OSA) [4].

Obesity is increasing at an alarming rate in the modern world. Nowadays, due to sedentary lifestyle, the problem of obesity starts from a younger age. Epidemiological studies clearly demonstrate a correlation between body weight and blood pressure in obese populations. In the Framingham Study, 70% of the new cases of essential hypertension were related to excess body fat [5,6]. Obesity is also a risk factor of non-dipping state not only in hypertensives but also in normotensives. Study done by Kotsis et al showed that in subjects more than 40 years of age more than 70% of obese were non-dippers [2].

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Ambulatory blood pressure monitoring has gradually become a widely used clinical tool for diagnoses of hypertension. It is a simple and novel method to measure BP even at night when the subject is sleeping and can easily give information about the dipping state.

The present study was designed to investigate the effect of obesity on dipper state in young adult males.

#### *Objectives*

1. To compare BMI in dippers and non-dippers in healthy young adult males
2. To compare Waist Circumference in dippers and non-dippers in healthy young adult males
3. To compare body fat percentage in dippers and non-dippers in healthy young adult males

#### **Subjects and Methods**

##### *Study Sample*

The present study was conducted in the Department of Physiology of Saraswathi Institute of Medical Sciences, Hapur from the month March 2017 to July 2017. A convenient sample of 60 healthy young adult males who volunteered for the study were enrolled after taking written informed consent from all the subjects. Ethical clearance was obtained from Institutional Ethical Committee. Subjects with any H/o hypertension, cardiovascular, respiratory or renal disorders, smokers and alcoholics were excluded from the study.

The subjects were supposed to report to the Department of Physiology at 10.30 am. Their Weight, Height, WC and Skin fold thickness were measured and measurement of Ambulatory BP recording was started by 11 am. The subjects were supposed to tie the cuff of Ambulatory BP monitor for 24 hours even during their sleep. As disturbed sleep may not decrease the sympathetic activity in the body and hence may not result in decrease in BP during sleep. the subjects who complained of disturbed sleep at night were also excluded from the study.

##### *Obesity Parameters*

Body weight was measured in kg by a mechanical scale to the nearest kg. Height was measured to the nearest one cm using stadiometer. BMI ( $\text{kg}/\text{m}^2$ ) was calculated using Quetelet's index i.e. weight in kgs divided by square of height in meters.

Waist circumference (WC) was measured midway between the lowest rib and the iliac crest using a non-stretchable measuring tape by average of three measurements nearest to 0.5 cm. Skinfold thickness was measured using Harpendent skinfold calipers at four sites viz triceps, biceps, subscapular and suprailiac region. Body fat percentage was calculated using Durnin-Womersley formula [7].

##### *Ambulatory BP Monitoring*

Subjects were allowed to sit quietly for 15 min prior to assessment of BP; three consecutive measurements were made 5 min apart, and baseline BP was determined as the mean of the three readings. 24 hours Ambulatory Blood Pressure was measured using Contec Ambulatory Blood Pressure Monitor. The cuff of the BP apparatus was tied on the non-dominant arm. Subjects were enquired about daily morning wake up time and night bed time. AMBP was set to measure BP every 15 min during daytime and every 30-min in night time while sleeping. All the subjects were instructed to perform their normal daily routine but refrain from heavy physical activity. Subjects were divided into two groups according to their dipper profile, as defined: dippers (nocturnal decrease in systolic BP was  $\geq 10\%$  of daytime BP) and non-dippers (nocturnal decrease in systolic BP was  $< 10\%$  of daytime BP).

##### *Statistical Analysis*

The data thus obtained was arranged by groups and analysed using standard descriptive statistics and the association between obesity parameters and Dipper state was analysed by independent t test. SPSS version 20 software was used to perform all the statistical analysis.

#### **Results**

The mean age of the subjects was  $27.76 \pm 6.46$  years. There were 9 subjects who complained of disturbed sleep at night, so, they were excluded from the study. So overall 51 subjects were included in the study. Out of the 51 subjects left, 17 subjects were non - dippers i.e. their BP decreased less than 10% of the average day systolic BP. The mean value of all the obesity parameters which are considered in the study i.e. BMI, WC and Body Fat% were significantly more in non-dipper subjects as compared to dippers as shown in table 1 suggesting that obesity may be a contributing factor for non-dipper state.

Table 1: Comparison of BMI, Waist Circumference and Body Fat % in dipper & non-dipper

Obesity Parameter	Mean $\pm$ Standard deviation		P value
	Dipper (n= 34)	Non Dipper (n = 17)	
BMI (kg/ m <sup>2</sup> )	24.55 $\pm$ 3.87	29.81 $\pm$ 3.96	0.001
WC (cm)	84.68 $\pm$ 10.95	97.18 $\pm$ 13.14	0.009
Fat percentage (%)	12.96 $\pm$ 2.71	17.43 $\pm$ 1.74	<0.01

## Discussion

The present study was a retrospective study which intended to investigate the association of dipper state with obesity parameters.

Many studies have shown that an increased risk of developing hypertension in overweight & obese. Kumanyika et al have shown body mass index to be even more strongly related than race to blood pressure and that its effect is similar across surveys in the United State and within sex and racial groups [8]. Mean systolic and diastolic BP levels were higher among subjects with elevated BMI was the result of another study [9].

WC reflects abdominal fat, which contains higher amounts of visceral fat. Visceral fat is made by liver, turned into cholesterol, and released into the bloodstream where it forms plaque on the artery walls, resulting in high blood pressure and cardiovascular disease [10].

Studies have shown that BF% had a strong association with the prevalence of dyslipidemia. As dyslipidemia has been reported to precede the appearance of blood pressure elevation, so increased BF% can be a good indicator of elevation in blood pressure [11,12].

Our study showed that all the obesity parameters like BMI, WC and Fat % were significantly increased in non-dippers. So, we interpret that obesity can result in non-dipping of blood pressure during sleep. There can be multiple factors responsible for this. Obesity may play an important role in impaired nocturnal BP drop include Sympathetic nervous system (SNS) activation which is suggested as most crucial, altered kidney functions, obesity-driven hormones elevation, endothelial dysfunction and vascular structural changes [13,14].

Several studies provide evidence of SNS activation in obese patients and those with high-caloric intake. High muscle SNS activity were detected by microneurographic methods in obese. Plasma norepinephrine concentration and rise of norepinephrine turnover in peripheral tissues correlated positively with level of caloric intake [13,15,16].

Obesity may lead to increased renin-angiotensin-aldosterone system (RAAS) activity, hyperinsulinaemia, impaired baroreceptor sensitivity and increased free fatty acids circulation and adipokines level. These factors are considered as those which result in increased sympathetic activity [15,17]. Animal models with high fat and carbohydrate intake developed significant rise in BP due to intensified peripheral alpha-1- and beta-adrenergic receptors sensitivity [18]. Corresponding results were reported in human studies, where BP of obese patients after one month of alpha- and beta-adrenergic receptors pharmacological blockade was noticeably more reduced than BP of lean ones [19].

Leptin is considered as a mediator of SNS activation. Its amount of secretion from adipose tissue stays in direct proportion with adipose tissue mass [20,21]. After passing blood-brain barrier leptin binds to hypothalamus and brainstem, where it suppresses hunger drive and stimulates SNS [13,22]. Also leptin levels in blood are higher between midnight & early morning. So, this may be another important reason of sympathetic overdrive during sleep and hence non-dipping state in obese [23].

Thus, our study has shown that obese even if normotensives may be non-dippers. Studies have shown that non-dipping state may result in increased risk of cardiovascular morbidities and mortality. Several cross-sectional studies have revealed that cardiac hypertrophy, silent cerebral infarction and microalbuminuria in normotensive or hypertensive populations were more common in non-dippers than dippers [24,25]. Furthermore, certain prospective studies have shown that each 5% attenuation in the nocturnal BP decline conferred a 20% increase in the risk of cardiovascular mortality in the normotensive or hypertensive population [26].

## Conclusion

Our study revealed that obese young adult subjects even if normotensives were more likely to become non-dippers. Non-dippers have a great risk of cardiovascular complications. Ambulatory Blood pressure monitoring should be done in obese

individuals to assess their dipping status and hence diagnose the risk of cardiovascular complications associated with non-dipping early.

#### Key Messages

As young obese individuals even if normotensive were non-dippers, Ambulatory Blood pressure monitoring should be done in obese individuals to assess their dipping status and hence diagnose the risk of cardiovascular complications associated with non-dipping early.

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# A Study of BMI and Parasympathetic Functions in Healthy Offsprings of Type 2 Diabetes Mellitus

Sangeeta Dattatreya Tuppada

## Abstract

**Objective:** Diabetes Mellitus is a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of Diabetes Mellitus exist and are caused by interaction of genetics and environmental factors. Individuals with Diabetes Mellitus may develop signs of autonomic dysfunction involving cholinergic, noradrenergic and peptidergic systems. Type 2 Diabetes Mellitus has a strong genetic component. Individuals with a parent with Type 2 Diabetes Mellitus have an increased risk of Diabetes. If both parents have Type 2 Diabetes Mellitus, the risk approaches to 40%. Thus this study is designed to evaluate BMI and autonomic functions in nondiabetic offsprings of Type 2 diabetic parents. **Method:** The cross-sectional study of BMI and Parasympathetic Functions was carried out in 30 healthy offsprings of Type 2 Diabetic Parents (Study group) and 30 healthy offsprings of Nondiabetic Parents (Control group) in the age range of 18 - 21 years randomly selected among 1<sup>st</sup> MBBS students. Statistical Analysis is done by 'Z' test. **Results:** There is insignificant decrease in parasympathetic functions and insignificant increase in BMI in Study Group compared to Control Group. **Conclusion:** Our observations indicate that subclinical autonomic dysfunction may develop without the presence of long-term hyperglycemia in family members of Type 2 diabetic subjects having higher BMI; Thus, it is not simply a complication of the hyperglycemia in these patients. An explanation could be that it is possible to inherit susceptibility genes for autonomic neuropathy, and that these genes could be expressed before or maybe even without the subjects developing diabetes. Different factors (including hyperglycemia) could subsequently affect the expression of the genes and influence the progression of neuropathy.

**Keywords:** Parasympathetic Functions; Healthy Offsprings.

## Introduction

Diabetes Mellitus is a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of Diabetes Mellitus exist and are caused by a interaction of genetics and environmental factors [1]. Individuals with Diabetes Mellitus may develop signs of autonomic dysfunction involving cholinergic, noradrenergic and peptidergic systems. Autonomic neuropathies affecting cardiovascular system cause a resting tachycardia and orthostatic hypotension. Reports of sudden death have also been attributed to autonomic neuropathy [2]. Type 2 Diabetes Mellitus has a strong genetic component. The concordance of Type 2 Diabetes Mellitus in identical twins is between 70% to 90%. Individuals with a parent with Type 2 Diabetes Mellitus have an increased risk of Diabetes. If both

parents have Type 2 Diabetes Mellitus, the risk approaches to 40%. Now a day Type 2 Diabetes Mellitus is being diagnosed more frequently in children and young adults, particularly in obese adolescents [1]. The possibility of early alterations in neural cardiovascular regulation in healthy offspring of diabetic patients has been addressed recently [2-5].

Aim of this study was to assess relation between BMI and Parasympathetic Functions in the offsprings of Type 2 Diabetic Parents & compare them with that offsprings of Nondiabetic parents.

## Materials and Methods

The cross-sectional study was carried out in 30 healthy offsprings of Type 2 Diabetic Parents (Study

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group) and 30 healthy offsprings of Nondiabetic Parents (Control group) in the age range of 18 - 21 years randomly selected among 1<sup>st</sup> MBBS students of BLDEU's Shri B M Patil Medical College, Bijapur. The ethical clearance for the study was obtained from ethical committee.

### *Method of Collection of Data*

*Study Group:* This group consists of 30 normal healthy male medical students (Offsprings of Type 2 Diabetic Parents) of BLDEU'S Shri B.M. Patil Medical College, Bijapur.

*Control Group:* This group consists of 30 age matched normal healthy male medical students (Offsprings of Nondiabetic Parents) of BLDEU'S Shri B.M. Patil Medical College, Bijapur. Each subject taking part was explained about the procedure to be adapted in the research.

All the subjects after thoroughly understanding the procedures to be adopted signed an informed consent form provided to them. All subjects underwent thorough clinical examination.

### *Inclusion Criteria*

Only healthy subjects of Indian origin were included in the study. The subjects without signs of cardiovascular, endocrinological, neurological, hematological & inflammatory diseases were selected for the study. The apparent health status of the subject was determined through clinical examination and history taking.

### *Exclusion Criteria*

The subjects with any of following findings were excluded from study.

1. Evidence of hypertension (systolic blood pressure more than 150 and diastolic blood pressure more than 90 mm Hg).
2. Subjects having diabetes mellitus, bronchial asthma, giddiness on standing, syncopal spells, visual disturbances, nocturnal diarrhea.
3. Subjects receiving drugs that are known to interfere with cardiac function or respiratory functions such as beta blockers, sympathomimetic drugs, vasodilators and diuretics.
4. Associated disease or conditions known to affect autonomic function like Guillain Barre syndrome,

Poliomyelitis, Diphtheria, Tuberculosis, Syphilis, Amyloidosis, Chronic renal failure.

5. Subjects with history of alcohol intake/ tobacco consumption in any form.
6. Any disease condition affecting the autonomic nervous system.

### *Following Parameters were Recorded in Each Subject*

- A. *Record of Physical Anthropometry of subjects:* Height (in centimeters), Weight (in kilograms), & Body Mass Index were measured.
- B. *Record of Physiological parameters:* Pulse rate (beats per minute), Blood Pressure (SBP and DBP in mmHg), Respiratory Rate (Cycles/Minute) were recorded.
- C. *Record of Autonomic Function Parameters:* The subject was informed about the procedure. Consent from each subject was taken before recording. For each parameter, three readings were taken. Mean of three readings was taken for calculation.

The following parasympathetic function parameters were recorded using Computerised 4 channel Physiopac performed as per methods described by Sir Roger Bannister [10].

- I. Heart Rate response to Valsalva Maneuver
- II. Heart rate response to deep breathing.
- III. Immediate heart rate response to standing

### *Statistical Analysis*

All values are presented as Mean + Standard Deviation (Mean + SD). Comparison of mean values of parameters between Control and Study group is done by Z test. Correlation between various autonomic function parameters & glycemic status is done by correlation. A p value of 0.05 or less was considered as statistically significant.

## **Results**

### *Heart Rate Response to Valsalva Maneuver*

Mean VR±SD of Control Group- 1.33±0.20  
Mean VR±SD of Study Group - 1.29±0.24.

There is insignificant (p=0.222) decrease in the Valsalva ratio (VR) of subjects in Study Group compared to Control Group.

*Heart rate variation (HRV) during Deep Breathing*  
 Mean HRV±SD of Control Group-28.56±7.44  
 Mean HRV±SD of Study Group-26.25±8.47  
 There is insignificant (p=0.132) decrease in the heart rate variation during deep breathing in Study Group compared to Control Group.

*Immediate Heart Rate Response to Standing (30:15 ratio)*  
 Mean Ratio±SD of Control Group-1.34±0.20  
 Mean Ratio±SD of Study Group-1.31±0.20  
 There is insignificant (p=0.335) decrease in the immediate heart rate response to standing in Study Group compared to Control Group.

**Table 1:** Anthropometric & Physiological Parameters (Mean ± SD) of Control and Study Group

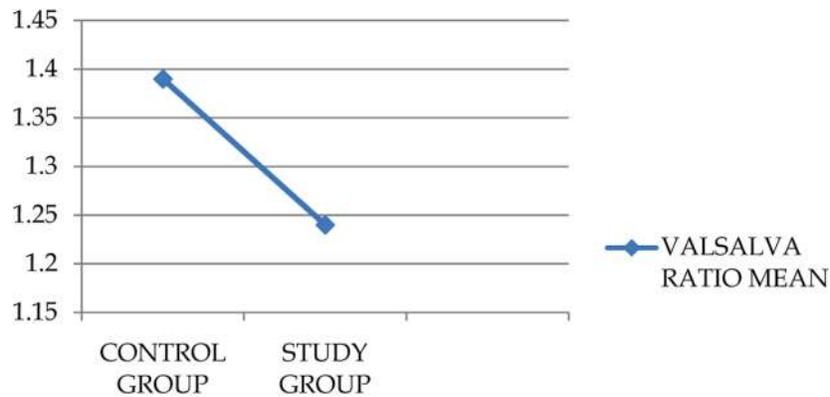
Parameters	Control Group	Study Group	Level of significance
Age (Years)	18.86 ± 1.04	19.0 ± 0.78	0.293
Height (cms)	170.16± 5.14	172.73±6.51	0.04*
Weight (Kg)	65.2 ± 10.82	70.16 ± 11.01	0.04*
BMI (kg/m <sup>2</sup> )	22.52 ± 3.55	23.51 ± 3.33	0.13
Resting SBP (mm of Hg)	119.53±11.9	124.13±12.80	0.07
Resting DBP (mm of Hg)	77.06±5.29	77.66±6.74	0.35

\*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant.

**Table 2:** Parasympathetic function parameters of subjects in Study and Control Groups

Parasympathetic function parameters	Control Group	Study Group	Level of significance
Valsalva Ratio	1.33 ± 0.20	1.29 ± 0.24	0.222
HR variation to deep breathing	28.56 ± 7.44	26.25 ± 8.47	0.132
Immediate HR response to standing (30:15)	1.34 ± 0.20	1.31 ± 0.20	0.335

\*p<0.05: Significant, \*\*p<0.01: Highly significant, \*\*\*p<0.001: Very highly significant.



**Graph 1:** Valsalva Ratio Mean

**Discussion**

The cross-sectional study is carried in 60 normal healthy medical students (Offsprings of Type 2 Diabetic Parents n=30; and Nondiabetic Parents n=30) in the age group of 18-21years. In our study we have recorded various physical & physiological parameters in both Control and Study groups. Parasympathetic function is assessed by heart rate response to Valsalva maneuver, heart rate response to deep breathing, immediate heart rate response to standing. Glycemic status was assessed by Oral Glucose Tolerance Test.

*Heart Rate Response to Valsalva Maneuver*

Our study showed insignificant decrease (p=0.222) in mean valsalva ratio in study group compared to control group. Our findings are in accordance with earlier studies done by C. Hauerslev Foss et al [8], Frontoni S et al [5]. Heart rate response to Valsalva maneuver appear to be more sensitive parameters to detect autonomic dysfunction amongst the three Parasympathetic function tests.

*Heart Rate Response to Deep Breathing*

There is insignificant (p=0.132) decrease in the heart rate variation during deep breathing in Study

Group compared to Control Group. Our study is in accordance with studies done by C. Hauerlev Foss et al [8], Frontoni S et al [5].

#### *Heart Rate Response to Standing (30: 15 Ratio)*

Heart rate response to standing in normal subjects consists of tachycardia maximum around 15<sup>th</sup> beat followed by relative bradycardia around 30<sup>th</sup> beat after standing. These hemodynamic responses are mediated by baroreceptors. In our study we found a insignificant ( $p=0.335$ ) decrease in the immediate heart rate response to standing in Study Group compared to Control Group. Our study is in accordance with studies done by C. Hauerlev Foss et al [8].

#### **Conclusion**

This cross sectional study shows that offsprings of Type 2 diabetic parents have increased prevalence of cardiac autonomic neuropathy compared with offsprings of nondiabetic parents. The results indicate that early autonomic neuropathy may be present in obese healthy offsprings of Type 2 Diabetic Parents and we suggest that autonomic neuropathy may be part of a genetic syndrome rather than a secondary complication of diabetes. In future studies on family members of Type 2 diabetic subjects, we will presumably gain important knowledge by following the trait of autonomic neuropathy as well as the diabetes trait itself.

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## Study of Lipid Profile in Hypothyroidism Patients

Sudhir Modala<sup>1</sup>, Deepak Das<sup>2</sup>, Usha Dhar<sup>3</sup>, K.V. Thimmaraju<sup>4</sup>, Manisha Baghel<sup>5</sup>

### Abstract

Hypothyroidism is a clinical case due to structural or functional deterioration of production of thyroid hormone and affects most the organ systems. Major clinical findings are fatigue, coarseness and dryness of the skin, intolerance to cold, poor concentration. The present study aim was to assess the association of lipid profile in hypothyroidism. 30 cases of hypothyroidism were chosen, 30 age group subjects' controls were chosen. Blood samples were collected from patient and T3, T4 and TSH levels were measured. Also, triglyceride, Cholesterol, HDL Cholesterol, LDL cholesterol levels in blood was measured. It was found that triglyceride, cholesterol, HDL, LDL levels were significantly increased in hypothyroidism cases than the controls. It is concluded that increasing total cholesterol, Triglycerides and LDL cholesterol Levels are at may enhance risk for developing cardiovascular disorders and Atherosclerosis

**Keywords:** Hypothyroidism; Dyslipidemia; Total Cholesterol; Triglycerides.

### Introduction

Hypothyroidism is a common metabolic disorder in the general population. Around 42 million people are suffering from thyroid diseases in India, Hypothyroidism is commonest disorder in India [1]. Hypothyroidism is characterized by as a decreased thyroid activity, lack of secretion of T3 and T4 hormones and leads to hyper secretion of pituitary TSH level [2]. Hypothyroidism is more common in women and increased levels of LDLC and TC [3]. Hypothyroidism is a secondary cause of dyslipidemia [4]. Thyroid hormones perform a large arrangement of metabolic functions including regulation of carbohydrate, protein and lipid metabolisms. The main significant effect on lipid metabolism consists of mobilization of triglycerides from the adipose tissue causing concentration of free fatty acids levels raised in plasma.

In patients with overt hypothyroidism there is increased in), Low Density Lipoprotein cholesterol (LDL-C), serum total cholesterol (TC). Apolipoprotein B, Lipoprotein (a) levels and possibly triglyceride

(TG) levels [5]. The present study aims to assess the association of hypothyroidism with lipid abnormalities.

### Materials and Methods

This study was carried out in the Department of Physiology collaboration with department of bio chemistry and general medicine in our college. 60 cases (mostly females) in the age group of 20 to 60 yrs were included. Patients with Low T3, Low T4 and TSH level above 6 $\mu$ U/ml were considered to be having hypothyroidism. Patients suffering from overt hypothyroidism who were undergoing treatment with anti thyroid drugs/ thyroxin, congestive cardiac failure, end stage renal disease, type II diabetes mellitus, patients on anti lipidemic drugs, post myocardial infarction and women on oral contraceptive pills were excluded from the study. The study included total 60 cases in that 30 cases of hypothyroidism and 30 cases of normal healthy euthyroids as controls. This data was statistically analyzed using the SPSS software (version 12.0) and by applying Student's t-test.

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## Results

A total of 60 subjects of age group 20 to 60 years, 30 hypothyroid cases and 30 (Euthyroid) controls were included in this study. Table 1 shows that there was

significant difference in the values between two groups. Table 2 shows that all the lipids measured, namely total cholesterol, triglycerides, HDL and LDL were found to be significantly increased in hypothyroid patients when compared to the euthyroid group ( $p < 0.001$ ).

**Table 1:** Levels of T3, T4, TSH in different groups

Parameters	Group- I CASES(n=30)	Group -II Controls(n=30)	P value
TSH( $\mu$ IU/ml)	8.04 $\pm$ 1.10	3.56 $\pm$ 0.82	0.000
T3(ng/ml)	0.93 $\pm$ 0.38	2.95 $\pm$ 0.55	0.000
T4( $\mu$ g/dl)	0.43 $\pm$ 0.15	1.36 $\pm$ 0.30	0.000

**Table 2:** Levels of lipid profile in different groups

Parameter	Group- I Cases (n=30)	Group - II Controls (n=30)	P value
Total Cholestrol(mg/dl)	227.57 $\pm$ 17.60	191.92 $\pm$ 7.49	0.000
Serum Triglycerides(mg/dl)	167.22 $\pm$ 16.37	144.63 $\pm$ 3.44	0.000
HDL Cholestrol(mg/dl)	43.76 $\pm$ 5.02	49.51 $\pm$ 1.73	0.000
LDL Cholestrol(mg/dl)	150.37 $\pm$ 20.32	113.60 $\pm$ 8.12	0.000
VLDL Cholesterol (mg/dl)	33.44 $\pm$ 3.27	28.81 $\pm$ 0.89	0.000

## Discussion

Hypothyroidism can bring unfavorable effects on the lipid metabolism. However, hyperthyroidism can be associated with acquired hypercholesterolemia or improvement of lipid profile [9]. The present cross sectional study was carried out in 30 Hypothyroid cases and 30 Healthy euthyroid subjects. Thyroid hormones main function is metabolism of lipids. Lack of thyroid hormones leads to form a hyperlipidemia. Some authors suggested that atherosclerosis might be caused due to chronic autoimmune thyroiditis independent of thyroid function. [10] This study suggests that lack of thyroid function is followed by decreased activity of HMG-CoA reductase, and increasing TC and LDL-C levels in hypothyroidism patients [6]. The higher prevalence of hypothyroidism among middle aged women, associated with an elevation in total plasma cholesterol level [7]. Hypothyroidism is followed by diastolic hypertension, in association with dyslipidemia may produced atherosclerosis and may effected to coronary artery disease [8]

In this study we aimed to find out the association of thyroid function with the lipid profile of hypothyroid patients by comparing with the euthyroid cases. Serum T3, T4, TSH, serum total cholesterol, serum triglycerides, HDL and LDL levels were measured by standard analytical methods. We

observed a positive association between hypothyroidism and serum total cholesterol, serum triglycerides, HDL and LDL levels. There was significant increase in lipid profile with the increase in serum TSH levels. It indicates that this interference of thyroid function with the lipid profile may increase the risk of developing CVDs in hypothyroid patients. The machainism for this association is that the thyroid hormones are the catabolic hormones and regulates various metabolic processes including synthesis, mobilization, and breakdown of lipids. The association between serum TSH and serum total cholesterol and LDL cholesterol might be both being a consequence of autoimmune activation involving lipoprotein (a) [11].

Altered levels of thyroid hormones can also influence the metabolism of HDL by showing the enhance activity of CETP (cholesterly ester transfer protein) which in turn transfers the CE (cholesterly esters) from HDL2 to VLDL in exchange for TG. [12]. Conversion of HDL2 to HDL3 and IDL to LDL is mediated by the action of hepatic lipase enzyme which is released and activated by HDL itself. Thyroid hormones also stimulates the activity of LPL (lipoprotein lipase enzyme) that breaks down the TG in chylomicrons and other lipoproteins into fatty acid and glycerol [13,14].

Changes in HL activity seem to be an important mechanism for the disturbance of cholesterol

metabolism in thyroid dysfunction while the thyroid hormone influence on LPL seems to be of importance mainly for the disturbance in triglyceride metabolism [15].

### Conclusion

From this study, it is concluded that Hypothyroidism is one of the most prevalent endocrine disease. Hypothyroid patients increasing total cholesterol, Triglycerides and LDL cholesterol Levels are at may enhance risk for developing cardiovascular disorders and Atherosclerosis.

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# A Correlative Study of Body Composition and Lipid Profile in Postmenopausal Women

Shireen Swaliha Quadri<sup>1</sup>, Manjunath M.L.<sup>2</sup>

## Abstract

**Introduction:** Cardiovascular disease is a leading cause of mortality in men and women in industrialized world. As Indian population differs in body composition and lifestyle, this study was undertaken to correlate the body composition parameters and lipid profile parameters as well as correlation of these parameters in postmenopausal women. **Materials and Methods:** Sixty-nine postmenopausal women were selected after their consent and divided into two groups. Group-I consists of 32 postmenopausal women within 5 years of menopause and Group-II consists of 37 postmenopausal women after 5 years of menopause. The body composition parameters were measured by classical anthropometry and skin fold callipers and lipid profile parameters were analysed by enzymatic method. Institutional ethical clearance was obtained. **Statistical Analysis:** Data was expressed as mean±SD. Pearson correlation test was used for analysis. p value less than 0.01 was considered statistically significant. **Results:** No statistically significant correlation was found between most of the body composition parameters in both the groups except for the weight ( $r = -0.3600$ ,  $p < 0.05$ ) BSA ( $r = -0.3555$ ,  $p < 0.05$ ), and FM ( $r = -0.3946$ ,  $p < 0.05$ ) which were significantly correlated with HDL-C in Group-II subjects. **Discussion and Conclusion:** This correlation may be due to increased intra-abdominal adipose tissue, as it has high sensitivity to catecholamine induced lipolysis. Non-esterified fatty acids mobilized from Intra-abdominal adipose tissue into the portal circulation, may increase hepatic production of TG and Apo-B, and increase subsequent export of VLDL particles.

**Keywords:** Menopause; Correlation; Body Composition; Lipid Profile.

## Introduction

Cardiovascular disease is a leading cause of mortality in men and women in industrialized world. The interactions between the various physiological risk factors for cardiovascular disease are complex; the incidence of cardiovascular disease increases with age in both sexes, but in women the risk increases markedly after menopause and eventually becomes equivalent to that of men. This observation has suggested, but has not proven, that estrogens have a protective effect against cardiovascular disease [1-3]. Menopause is a natural event in the ageing process and signifies the end of reproductive years with cessation of cyclic ovarian functions as manifested by cyclic menstruation. It is heralded by menopausal transition, a period when the endocrine, biological and clinical features of approaching menopause begins. The hormonal changes associated with menopause i.e. low plasma levels of estrogen and

marked increase in LH and FSH levels exerts a significant effect on plasma lipids and lipoproteins [4].

The BMI matched obese postmenopausal women, had significantly higher, WC, WHR and intra-abdominal fat volume compared with premenopausal women. TC and TG were significantly higher and HDL-C was significantly lower in postmenopausal women. When age matched pre- and postmenopausal women were compared only TC was significantly higher in postmenopausal group [5,6]. However, older postmenopausal women (>50years) had significantly higher WC and WHR compared with younger postmenopausal women (<50years).

There was no difference in total body fat-free or appendicular skeletal muscle mass in healthy premenopausal women and early postmenopausal women. In contrast, total body fat mass was 28% higher and percentage fat 17% higher in postmenopausal women compared with

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premenopausal women. Postmenopausal women had a 49% greater intra-abdominal and a 22% greater abdominal subcutaneous fat area compared to premenopausal women. The menopause related difference in intra-abdominal fat persisted after statistical adjustment for age and total body fat mass, whereas no difference in abdominal subcutaneous fat was noted. A similar pattern of differences in total and abdominal adiposity was noted in sub samples of pre- and postmenopausal women matched for age or fat mass [7,8]. In healthy women, during the time from premenopausal to first year Postmenopausal examinations, the changes in LDL-C, TG, and BMI were larger than those between first and fifth year Postmenopausal examinations [9,10].

There have been studies regarding the effect of menopause on body weight, fat distribution, total fat %, and also on lipid profile, but most of the studies are conducted on western population. As Indian population differs in body composition and lifestyle, this study was undertaken to correlate the body composition parameters and lipid profile parameters as well as correlation of these parameters in postmenopausal women.

## Materials and Methods

The present study was conducted in the department of physiology, Shimoga institute of Medical Sciences, Sagar Road, Shivamogga. Sixty-nine postmenopausal women were selected after their consent. The institutional ethical clearance was obtained. The participants were divided into two groups as, Group-I consists of 32 postmenopausal women within 5 years of menopause and Group-II consists of 37 postmenopausal women after 5 years of menopause. All healthy postmenopausal women, who attained menopause by natural means were included. Postmenopausal women who have undergone hysterectomy, diabetic, hypertensive, on hormone replacement therapy, lipid lowering drugs & with H/O Gynaecological & hormonal disorders were excluded.

### *Measurement of Physiological Parameters*

Height was measured on a wooden stadiometer, bearing a flat stand and a vertical surface with marking in centimetres. A sliding head piece was used for accurate work. Height was recorded and expressed in centimetres. Body weight (Wt) of all the subjects was measured by using weighing scale and expressed in kilogram. Body surface area (BSA) in Sq.

Mts ( $m^2$ ) was calculated by Duboi's nomogram. The body mass index was derived by Quetelet's index from body weight (kg)/height ( $m^2$ ). Waist circumference was measured at narrowest part of torso with plastic tape.

Hip circumference was measured at maximal extension of buttocks with plastic tape. Waist-Hip Ratio was calculated by dividing waist circumference by hip circumference.

Body fat percentage was calculated by using skin fold callipers. A skin fold callipers is a device which measures the thickness of a fold of skin with its underlying layer of fat. By measuring at key locations it is possible to estimate the total percent of body fat of a person. The callipers used is scientifically developed and calibrated. The instrument has springs which exerts certain pressure on skin fold ( $10g/mm^2$ ) and measures the thickness in millimetres.

The fold of skin with its underlying layer of fat was pulled out and grasped in the fingers of left hand, while holding the callipers in the right hand, the jaws of callipers were held about one fourth inch from the fingers of the left hand, which continues to hold the fold of the skin. The trigger of the callipers was released so that the entire force of the jaws was on the skin folds.

The skin fold thickness was measured on the left side at four sites such as Back of the mid-arm (Triceps), Front of the mid-arm (Biceps), Below the shoulder blade on the back (sub scapular) and Waist area above hip bone (suprailiac). The measurement was taken in all four areas & added together, and then body fat percentage was determined from the chart in the instruction manual.

The fat mass was calculated as  $FM = Wt / 100 \times BF\%$  and expressed in kilograms. Fat free mass was calculated and expressed in kilograms as  $FFM = \text{Weight} - \text{fat mass}$ . Fat mass index was calculated from fat mass in (kg)/Height in ( $m^2$ ). Muscle Mass was calculated by knowing fat free mass as  $MM = 50\%$  of FFM.

### *Measurement of Lipid Profile Parameters*

After overnight fasting 2ml of venous blood was collected from each subject and centrifuged at 3000rpm for 15min to obtain the serum which was used for analysis.

The level of Triglyceride, Total-cholesterol, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol were measured using semi-automated analyser (Erba star 21 plus) using commercially available kits.

*Statistical Analysis*

Data was expressed as mean±SD. The Pearson correlation test was used to correlate the body composition and lipid profile between two groups of postmenopausal women. P value less than 0.05 was the level of significance.

**Results**

In the present study, we evaluated and correlated all the body composition parameters and lipid profile between post menopause of five years with post

menopause of ten years. The correlation between body weight, and total cholesterol, triglyceride, HDL, LDL, VLDL, total cholesterol to HDL ratio and HDL to LDL ratio was found to be nonsignificant ( $p > 0.05$ ) in group I whereas, body weight was significantly negatively correlated with HDL in group-II subjects ( $r = -0.3600$ ,  $p < 0.05$ , Table 1).

The correlation of body mass index, waist circumference, hip circumference, waist hip ratio, fat percentage, free fat mass, muscle mass with total cholesterol, triglyceride, HDL, LDL, VLDL, total cholesterol to HDL ratio and HDL to LDL ratio was found to be nonsignificant ( $p > 0.05$ , Table 1,2,3,4).

**Table 1:** Correlation of body weight, body mass index and body surface area with the lipid profile in two different groups

Sl. No	Parameter	Group-I(N=32)			Group-II(N=37)		
		R	P	S/NS	R	P	S/NS
1	Wt vs TC	-0.1539	>0.05	NS	-0.008	>0.05	NS
2	Wt vs TG	0.2179	>0.05	NS	0.1682	>0.05	NS
3	Wt vs HDL	-0.3148	>0.05	NS	-0.3600	<0.05	S
4	Wt vs LDL	-0.1726	>0.05	NS	0.0141	>0.05	NS
5	Wt vsVLDL	0.2261	>0.05	NS	0.1660	>0.05	NS
6	Wt vs TC/HDL	0.1594	>0.05	NS	0.2659	>0.05	NS
7	Wt vs HDL/LDL	0.0260	>0.05	NS	-0.1245	>0.05	NS
8	BMI vs TC	-0.0200	>0.05	NS	-0.0787	>0.05	NS
9	BMI vs TG	0.3089	>0.05	NS	-0.0030	>0.05	NS
10	BMI vs HDL	-0.3193	>0.05	NS	-0.1105	>0.05	NS
11	BMI vs LDL	-0.0728	>0.05	NS	-0.0557	>0.05	NS
12	BMI vsVLDL	0.3353	>0.05	NS	-0.0040	>0.05	NS
13	BMI vs TC/HDL	0.2771	>0.05	NS	0.0787	>0.05	NS
14	BMI vsHDL/LDL	-0.0608	>0.05	NS	0.0843	>0.05	NS
15	BSA vs TC	-0.2780	>0.05	NS	0.0583	>0.05	NS
16	BSA vs TG	0.0656	>0.05	NS	0.2291	>0.05	NS
17	BSA vs HDL	-0.2878	>0.05	NS	-0.3555	<0.05	S
18	BSA vs LDL	-0.2354	>0.05	NS	0.0616	>0.05	NS
19	BSA vsVLDL	0.0374	>0.05	NS	0.2269	>0.05	NS
20	BSA vs TC/HDL	0.0224	>0.05	NS	0.2625	>0.05	NS
21	BSA vsHDL/LDL	0.0735	>0.05	NS	-0.2175	>0.05	NS

**Table 2:** Correlation of waist circumference, hip circumference and waist-hip ratio with the lipid profile in two different groups

1	WC vs TC	0.0173	>0.05	NS	0.0794	>0.05	NS
2	WC vs TG	0.2538	>0.05	NS	0.0938	>0.05	NS
3	WC vs HDL	-0.2433	>0.05	NS	-0.2218	>0.05	NS
4	WC vs LDL	-0.2450	>0.05	NS	0.0990	>0.05	NS
5	WCvsVLDL	0.2668	>0.05	NS	0.0920	>0.05	NS
6	WCvsTC/HDL	0.2546	>0.05	NS	0.2579	>0.05	NS
7	WC vs HDL/LDL	-0.0655	>0.05	NS	0.0800	>0.05	NS
8	HC vs TC	-0.1192	>0.05	NS	0.0447	>0.05	NS
9	HC vs TG	0.3025	>0.05	NS	0.1517	>0.05	NS
10	HC vs HDL	-0.2998	>0.05	NS	-0.2668	>0.05	NS
11	HC vs LDL	-0.1732	>0.05	NS	0.0566	>0.05	NS
12	HC vsVLDL	0.3203	>0.05	NS	0.1503	>0.05	NS
13	HC vs TC/HDL	0.2256	>0.05	NS	0.2454	>0.05	NS
14	HC vsHDL/LDL	0.0224	>0.05	NS	-0.0648	>0.05	NS
15	WHR vs TC	0.2720	>0.05	NS	0.0480	>0.05	NS
16	WHR vs TG	0.0007	>0.05	NS	0.0529	>0.05	NS
17	WHR vs HDL	0.1015	>0.05	NS	-0.0283	>0.05	NS
18	WHR vs LDL	0.2604	>0.05	NS	0.0678	>0.05	NS
19	WHR vsVLDL	-0.0014	>0.05	NS	-0.0539	>0.05	NS
20	WHRvsTC/HDL	0.1170	>0.05	NS	0.0883	>0.05	NS
21	WHRvsHDL/LDL	-0.1439	>0.05	NS	-0.0346	>0.05	NS

**Table 3:** Correlation of body fat percentage, fat mass and free fat mass with the lipid profile in two different groups

1	BF% vs TC	-0.0458	>0.05	NS	-0.1493	>0.05	NS
2	BF% vs TG	0.2500	>0.05	NS	0.1510	>0.05	NS
3	BF% vs HDL	-0.2746	>0.05	NS	-0.2457	>0.05	NS
4	BF% vs LDL	-0.0849	>0.05	NS	-0.1503	>0.05	NS
5	BF% vsVLDL	0.2691	>0.05	NS	0.1500	>0.05	NS
6	BF%vsTC/HDL	0.2532	>0.05	NS	0.0894	>0.05	NS
7	BF%vsHDL/LDL	-0.0447	>0.05	NS	0.0714	>0.05	NS
8	FM vs TC	-0.1382	>0.05	NS	-0.0883	>0.05	NS
9	FM vs TG	0.2202	>0.05	NS	0.1865	>0.05	NS
10	FM vs HDL	-0.2883	>0.05	NS	-0.3946	<0.05	S
11	FM vs LDL	-0.1667	>0.05	NS	-0.0678	>0.05	NS
12	FM vsVLDL	0.2343	>0.05	NS	0.1849	>0.05	NS
13	FM vsTC/HDL	0.1700	>0.05	NS	0.2415	>0.05	NS
14	FM vsHDL/LDL	0.0316	>0.05	NS	-0.0678	>0.05	NS
15	FFM vs TC	-0.1661	>0.05	NS	0.0566	>0.05	NS
16	FFM vs TG	0.1955	>0.05	NS	0.1323	>0.05	NS
17	FFM vs HDL	-0.3247	>0.05	NS	-0.2912	>0.05	NS
18	FFM vs LDL	-0.1703	>0.05	NS	0.0775	>0.05	NS
19	FFM vsVLDL	0.1957	>0.05	NS	0.1304	>0.05	NS
20	FFM vsTC/HDL	0.1315	>0.05	NS	0.2538	>0.05	NS
21	FFM vsHDL/LDL	0.0200	>0.05	NS	-0.1572	>0.05	NS

**Table 4:** Correlation of fat mass index and muscle mass with the lipid profile in two different groups.

1	FMI vs TC	-0.0583	>0.05	NS	-0.1245	>0.05	NS
2	FMI vs TG	0.2731	>0.05	NS	0.0671	>0.05	NS
3	FMI vs HDL	-0.2961	>0.05	NS	-0.1944	>0.05	NS
4	FMI vs LDL	-0.1044	>0.05	NS	-0.1086	>0.05	NS
5	FMI vsVLDL	0.2955	>0.05	NS	0.0663	>0.05	NS
6	FMI vsTC/HDL	0.2460	>0.05	NS	0.1005	>0.05	NS
7	FMI vsHDL/LDL	-0.0265	>0.05	NS	0.0781	>0.05	NS
8	MM vs TC	-0.1606	>0.05	NS	0.0557	>0.05	NS
9	MM vs TG	0.1924	>0.05	NS	0.1330	>0.05	NS
10	MM vs HDL	-0.3217	>0.05	NS	-0.2918	>0.05	NS
11	MM vs LDL	-0.1637	>0.05	NS	0.0775	>0.05	NS
12	MM vsVLDL	0.1924	>0.05	NS	0.1312	>0.05	NS
13	MM vsTC/HDL	0.1319	>0.05	NS	0.2539	>0.05	NS
14	MM vsHDL/LDL	0.0173	>0.05	NS	-0.1568	>0.05	NS

The correlation between body surface area and total cholesterol, triglyceride, HDL, LDL, VLDL, total cholesterol to HDL ratio and HDL to LDL ratio was found to be nonsignificant ( $p>0.05$ ) in group I whereas, body surface area was significantly negatively correlated with HDL in group-II subjects ( $r=-0.3555$ ,  $p<0.05$ , Table 2). The correlation between fat mass and total cholesterol, triglyceride, HDL, LDL, VLDL, total cholesterol to HDL ratio and HDL to LDL ratio was found to be nonsignificant ( $p>0.05$ ) in group I whereas, body surface area was significantly negatively correlated with HDL in group-II subjects ( $r=-0.3946$ ,  $p<0.05$ , Table 3).

## Discussion

In the present study, the mean values of body weight, BMI, BSA, WC, HC, FM, FFM, FMI and MM

were more in Group-I compared to Group-II and WHR and BF% were more in Group-II compared to Group-I but not statistically significant. All the body composition parameters were within physiological limits in both the groups except BF% and FM which were found to be above the normal range.

The body composition parameters and lipid profile parameters were correlated in both Group-I and Group-II subjects. No statistically significant correlation was found between most of the parameters in both the groups except for the weight ( $r = -0.3600$ ,  $p<0.05$ ) BSA ( $r = -0.3555$ ,  $p<0.05$ ), and FM ( $r = -0.3946$ ,  $p<0.05$ ) which were significantly correlated with HDL-C in Group-II subjects. This correlation may be due to increased intra-abdominal adipose tissue, as it has high sensitivity to catecholamine induced lipolysis. Non-esterified fatty acids mobilized from Intra-abdominal adipose tissue into the portal circulation, may increase hepatic production of TG

and Apo-B, and increase subsequent export of VLDL particles. Increased VLDL-TG in turn depress circulating concentrations of HDL-C due to the action of cholesterol-ester transfer protein [11,12]. Further study with more sophisticated methods like dual x-ray absorptiometry, computerized tomography scan and magnetic resonance imaging to measure body composition parameters may throw a better light in correlating body composition parameters with lipid profile parameters.

### Conclusion

There was no statistically significant correlation between most of the body composition parameters and lipid profile parameters in both the groups except for the weight, BSA and FM which were significantly negatively correlated with HDL-C in Group-II subjects. This correlation may be due to an increased intra-abdominal adipose tissue, as it has high sensitivity to catecholamine induced lipolysis.

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