"A STUDY OF ECG PATTERNS IN 1ST, 2ND AND 3RD TRIMESTERS OF PREGNANCY"

By

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DISSERTATION SUBMITTED TO THE BLDE UNIVERSITY,BIJAPUR

In partial fulfillment
of the requirements for the degree of

DOCTOR OF MEDICINE

IN

PHYSIOLOGY

Under the guidance of

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B.L.D.E UNIVERSITY, KARNATAKA, BIJAPUR.

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I **Dr.Nandini.B.N,** here by solemnly declare that this dissertation entitled "**A STUDY OF ECG PATTERNS IN 1ST,2ND AND 3RD TRIMESTERS OF PREGNANCY."** is a bonafide and genuine research work carried out by me under the guidance of **Dr. MANJUNATHA.AITHALA** MD Associate Professor, Department of Physiology, B.L.D.E.U'S Shri B. M. Patil Medical College, Hospital and Research Centre, Bijapur.

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Date: **Dr NANDINI.B.N.**

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LIST OF ABBREVIATIONS USED

(In alphabetical order)

AVN- Atrio- Ventricular Node

BP- Blood Pressure

BMI- Body Mass Index

BSA- Body Surface Area

bpm- beats per minute

DBP- Diastolic blood pressure

ECG- Electrocardiogram

HR- Heart rate

Ht- Height

LBBB- Left bundle branch block

MAP- Mean Arterial pressure

PP- Pulse Pressure

PR- Pulse Rate

RBBB- Right Bundle Branch Block

RR – Respiratory Rate

SAN-Sino Atrial Node

SBP- Systolic blood pressure

Wt- Weight

ABSTRACT

Background & Objectives: Pregnancy is considered to be the most beautiful and enriching experience in the life of a women. It is characterized by profound changes in the function of virtually every regulatory system in the human body. Pregnancy not only causes remarkable changes in pregnancy, in cardiovascular system, but also brings about various changes in ECG. Thus this study was designed to evaluate the ECG patterns in 1^{st}, 2^{nd} and 3^{rd} trimesters of pregnancy.

Methods: 150 healthy pregnant women in the age group of 20-35yrs who were attending the OPD of OBGy of Shri B.M. Patil Medical College 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 and compared with another apparently healthy age matched 50 non pregnant women (control group). Through physical & systemic examination of each subject was done and physical, physiological & ECG parameters were recorded in control and study groups.

Results: Statistically significant observations such as Heart rate was increased in 1^{st}, 2^{nd} and 3^{rd} 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^{nd} and 3^{rd} trimesters of pregnancy, ST segment depression was noticed in 4 subjects in 2^{nd} trimester and 6 in 3^{rd} trimester of pregnancy, QTc interval showed an increase in pregnant women in 1^{st}, 2^{nd} and 3^{rd} trimesters of pregnancy , T wave abnormalities in lead III showed an increase in both 2^{nd} and 3^{rd} trimesters of pregnancy and QRS frontal axis was decreased in 1^{st}, 2^{nd} and 3^{rd} trimesters of pregnancy.

Conclusion:

The appearance of electrocardiographic changes are attributed to major adaptations in the maternal cardiovascular system. These changes may also be influenced by gestational autonomic imbalance and sex hormone environment. This knowledge of electrocardiographic changes during pregnancy may be helpful in the prevention of gestational complications associated with an inadequate maternal hemodynamic adaptation.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Pregnancy is considered to be the most beautiful and enriching experience in the life of a woman. It is characterized by profound changes in the function of virtually every regulatory system in the human body.

In pregnant women, large number of local and systemic changes are known to occur. These changes will continue throughout pregnancy[1] especially cardiovascular changes such as increase in heart rate, cardiac output and intravascular volume[2]. 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 fetus[3]. In the absence of these adaptations, incidence of gestational complications such as fetal growth restriction and pregnancy induced hypertension are known to increase.[4]

In normal pregnancy, functional systolic murmurs are quite common. They are heard over the precordium. It is of great importance to document the presence or absence of the systolic murmur and to identify it as innocent or pathologic[6]. Respiratory effort is accentuated at times, dyspnoea may be experienced. Bilateral pedal edema usually develops after midpregnancy[5]. The cardiorespiratory adaptation during pregnancy is well tolerated by healthy women. However, these changes pose a threat to those who are with heart diseases.[7]

Heart diseases contribute significantly to maternal mortality throughout the world. Although heart diseases rarely occur during pregnancy, it is a fact that greater number of women with known or potential heart diseases are becoming pregnant[7].

Hemodynamic changes during pregnancy play a major role in the induction of arrhythmias. The increased incidence of arrhythmias during pregnancy is also reported. [8]

MATERNAL ADAPTATIONS TO PREGNANCY

Physiological Changes during pregnancy:

The anatomical, physiological and biochemical adaptations to pregnancy are profound. Many of these changes begin soon after fertilization and continue throughout gestation.[9]

Changes in the cardiovascular system:

During pregnancy and puerperium, there are remarkable changes in the heart and circulation. The most important changes in cardiac function occur in the first eight weeks of pregnancy.[9]

Blood volume: [10]

During pregnancy, a great deal of new maternal tissue is synthesized, especially in the uterus and the breasts. These areas show an increase in the size of the vascular bed. The blood volume increases during pregnancy to fill the enlarged vascular bed. There is also redistribution of blood. Both plasma volume and red cell mass increase in proportion to the duration of gestation to about 1000ml and 250ml respectively above non-pregnant values. The total increase in blood volume is about 25-30%. But, the percentage increase of plasma is almost 50% while that of red cells is only 18%. This shows that there is a much greater increase in plasma than in cells, which is a fundamental feature of pregnancy.

This hemodilution results in "Physiological Anemia". The hematocrit falls from 40-42% to about 34% in the pregnant women. The percentage of water in the blood rises from about 91% to 92% whereas electrolyte and protein levels fall. The relative dilution of blood in pregnancy serves a useful purpose in lowering the viscosity which in turn allows efficient perfusion of the placenta.

Cardiac Output:

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 liter per minute. At the eighth month of pregnancy, this rises to about 5.5 L.[10]

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.[11] 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.[9]

Heart rate: [10]

Cardiac output depends on the heart rate and on the output of the ventricles at each beat i.e, the stroke volume.

The resting pulse rate increases as the pregnancy advances.

Non-pregnancy	→	70 beats/min
Early pregnancy	→	78 beats/min
End of pregnancy	→	85 beats/min

The heart rate increases by 10-15 beats per minute more than the pre - pregnant state. There is an increase in both stroke volume and heart rate. The stroke volume increases to 10% more than the non-pregnant value, where as 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.

Arterial Blood Pressure:

In normal pregnancy, the blood pressure in first few months is similar to that of non-pregnant woman. In the middle three months, however, the blood pressure tends to fall on an average by about 3-5 mm Hg. But sometimes the blood pressure drop may be of the order of 20-30 mm Hg, though the patients seem not to suffer at all from this. In the last three months of pregnancy, the blood pressure slowly rises again until it comes back to the normal non-pregnant level.[10]

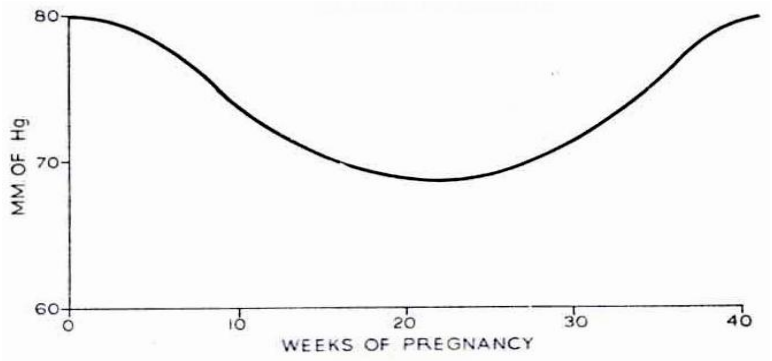


Fig. 1: Blood pressure changes during pregnancy

The antecubital venous pressure remains unchanged during pregnancy. But, in the supine position the femoral venous pressure rises steadily from 8 cm H_2O to 24 cm H_2O at term. These alterations contribute to dependent edema, varicose veins in the legs and hemorrhoids.[10]

Supine Hypotension Syndrome of Pregnancy

In late pregnancy with the woman in the supine position, the large pregnant uterus consistently compresses the venous system which returns blood from the lower half of the body. As a result, the cardiac filling will be reduced with a decrease in cardiac output. In about 10% of women, this causes significant arterial hypotension which sometimes referred to as "Supine Hypotensive Syndrome". These hemodymaic changes are associated with weakness, light headedness, nausea, dizziness and even syncope. When the supine position is abandoned, these hemodynamic effects and symptoms are usually promptly relieved.[11]

Cardiac Arrhythmias:

There is an increased susceptibility for cardiac arrhythmias during pregnancy. It is manifested by frequent sinus tachycardia, atrial or ventricular premature beats and paroxysmal supraventricular tachycardia.[11]

Position of Heart:

As the diaphragm becomes progressively elevated, the heart is displaced to the left and upward, while at the same time it is rotated on its long axis during pregnancy. As a result, the apex of the heart is moved somewhat laterally from its normal position in the normal non pregnant state. An increase in the size of cardiac silhouette is found in

radiographs. The extent of these changes is influenced by the size and position of the uterus, tone of the abdominal muscles and configurations of the abdomen and thorax[12]

Innocent Systolic Murmur:

These murmurs can be heard in most pregnant women due to hyperkinetic circulatory state. Murmurs are usually soft and midsystolic. They are best heard at the lower areolar edge and over the pulmonic area and radiate to suprasternal notch more to the left than to the right side of the neck. The benign murmur of pregnancy may be louder or longer.They may sound like those associated with atrial septal defect or stenosis of one of the semilunar valves. In such subjects, an echocardiographic and Doppler evaluation is warranted to rule out cardiac abnormalities.

Two benign continuous murmurs that may be heard during gestation are "Cervical Venous Hum" and "Mammary Soufflé". The venous hum is usually heard maximally over the right supraclavicular fossa, but can radiate to the contralateral area and sometimes to the area below the clavicle. The mammary soufflé may be either systolic or continuous, heard over the breast in late gestation or in the lactating period. It is caused by increased flow in the mammary arteries. Characteristically, the murmur decreases or vanishes when the patient moves to the upright position.

Diastolic murmur may be heard in normal pregnant women due to increased blood flow through the atrioventricular valve. Such a finding however, is infrequent in the healthy pregnant woman and therefore requires careful diagnostic work up to rule out underlying cardiac diseases.[11]

Heart diseases during pregnancy remain a serious problem. One of the important tools for the diagnosis of heart diseases is recording electrocardiogram. In order to make use of it accurately, knowledge about ECG changes during normal pregnancy is a must.[13] Electrocardiography is one of the simplest techniques used to detect ischemic heart diseases, hypertensive heart diseases & asymptomatic arrythmias.[14]

An appropriate knowledge of physiological aspects of ECG is required to understand and distinguish ECG changes that occur in various pathological conditions.

ELECTROCARDIOGRAM:

The history of electrocardiography dates from the end of eighteenth century.[15]

In 1787 – Aloysio Luigi Galvani demonstrated that the muscle of the hind limbs of a frog also manifested electromotive phenomena.[15] He accidentally observed that the muscles of a frog exhibited vigourous contractions whenever sparks were drawn from an electrical machine and the nerves of the preparation were touched with a knife simultaneously. He suspected that this phenomenon was related to the electrical discharge.[15]

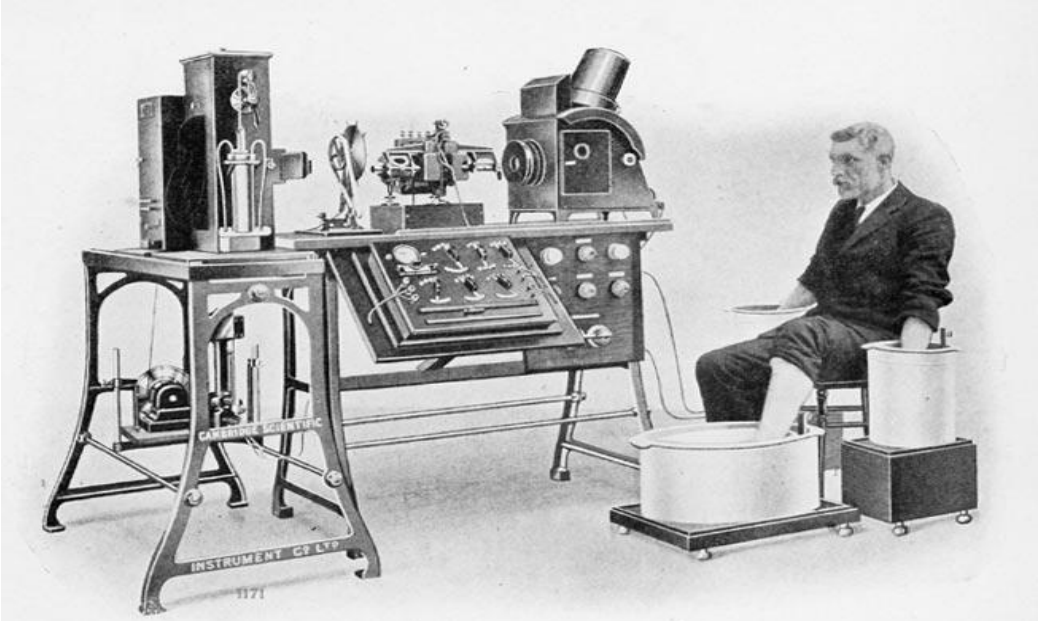
In 1856 – Rudolph Von Kolliker and Heinrich Muller demonstrated the presence of electrical currents associated with each heart beat by applying a galvanometer to the base and apex of exposed ventricle.[16]

In 1876 – Marey used the electrometer to record the electrical activity of an exposed frog's heart.[15]

In 1878 – Saunderson and Page recorded the cardiac events in laboratory animals by means of the capillary electrometer. [15]

In 1887 – Augustus D Waller was first to demonstrate a measurable amount of current in the human body associated with contraction of the heart by using the capillary electrometer.[16] He showed that the currents produced by the heart muscle could be recorded in intact animals by the use of surface electrodes. He then proceeded to apply this method to human beings. He discovered that the electrical activity of the human heart could be recorded by means of the capillary electrometer without opening the chest and exposing the heart. [15]

In 1902 - The electric current from the human heart was registered in an accurate quantitative manner by the application of a new instrument, String Galvanometer by William Einthoven. He was awarded Noble prize for his contributions in the field of cardiology in 1924.



PHOTOGRAPH OF A COMPLETE ELECTROCARDIOGRAPH, SHOWING THE MANNER IN WHICH THE ELECTRODES ARE ATTACHED TO THE PATIENT, IN THIS CASE THE HANDS AND ONE FOOT BEING IMMERSED IN JARS OF SALT SOLUTION

Fig. 2: Electrocradiograph constructed by William Einthoven

In 1932 - Direct writing electrocardiography was designed by Duchosal and Luthi.[18]

In 1933 – Frank N Wilson and his associates devised the unipolar lead system. The unipolar method of recording electrical activity of the heart was first used for experimental purposes. Later on, it was adopted for clinical medicine. Today, the conventional clinical ECG consists of 12 leads which constitutes so called "Scalar ECG".[19]

Physiological Basis of Electrocardiography

The electrocardiogram (ECG) is the graphical recording of the electrical activity of the heart recorded from the body surface by electrodes which are positioned to reflect the activity from variety of spatial perspectives.[20]

The following factors are involved in the genesis of the electrocardiogram

1) Initiation of impulse formation in the primary pacemaker (SAN).
2) Transmission of impulse through the specialized conducting system of the heart.
3) Depolarization of the atrial and ventricular myocardium (activation).
4) Repolarization of all the above areas (recovery).[15]

Intracellular Potentials:

Most of the cardiac cells maintain a resting membrane potential (RMP) of -90mv, with inside of the cell being negative with respect to outside. The major factor that determines the RMP is gradient of Potassium (K^+) across the cell membrane. Intracellular concentration of K^+ is 30 to 45 times higher than the extra cellular concentration of Na^+ (It is about 10-15 times higher than intracellular concentration). At the onset of

depolarization of the cardiac muscle cell, there is an abrupt change in permeability of cell membrane to Na^+ ions (and Calcium ions to a lesser degree) Na^+ ions enter the cell and result in sharp rise in intracellular potential to +20mv.This is designated as phase 0 and represent first inward current.[20]

Following the depolarization, there is a relatively slow and gradual return of intracellular potential to RMP (Phase 4). This is called repolarization. It is divided into three phases.

Phase – 1: An initial return of intracellular potential to 0mv. This results mainly due to abrupt closing of Sodium channels. Chloride ions entering the cell may also contribute to this phase.

Phase – 2: A plateau phase of repolarization owing to slow entrance of Calcium ions into the cell.

Phase – 3: This represents the slow gradual return of intracellular potential to RMP. It is due to extrusion of K^+ out of cells, which reestablishes normal negative resting potential.

However, the cell is left with an excess of Na^+ and deficit of K^+. To restore the original ionic concentration in the cell membrane, Sodium Potassium pump mechanism becomes effective. The energy required for this pump is derived from breakdown of ATP to ADP. This pump pumps $3Na^+$ ions out and $2K^+$ ions in.[20]

Physiological Basis of ECG.

Body fluid is a volume conductor. The fluctuations in potential that represent algebric sum of the action potentials of myocardial fibres can be recorded extracellularly. The record of these potential fluctuations during the cardiac cycle is called as

Electrocardiogram (ECG). Most of electrocardiographic machines record these fluctuations on moving strip of paper.[21]

The ECG may be recorded by using an active or exploring electrode connected to an indifferent electrode at zero potential (unipolar recording) or by using two electrodes (bipolar recording). In a volume conductor, the sum of potentials at the point of equilateral triangle with a current source in the centre is zero at all times. A triangle with the heart at it's centre (Einthoven's triangle) can be approximated by placing electrodes on both arms and on left leg. These are three Standard Limb Leads in electrocardiography. If these electrodes are connected to a common terminal, an indifferent electrode that stays near zero potential is obtained.[21]

Depolarization moving toward an active electrode in a volume conductor produces a positive deflection, whereas depolarization moving in the opposite direction produces negative deflection. [21] The names of various waves and segments of ECG in human shown in figure 3. Conventionally, upward deflection is written when the active electrode becomes positive relative to the indifferent electrode and a downward deflection is written when active electrode becomes negative.

The P wave is produced by atrial depolarization, QRS complex by ventricular depolarization and ST segment and T wave by ventricular repolarization. The manifestations of atrial repolarization are not normally seen as they are obscured by QRS complex. The U wave, an inconstant finding believed to be due to slow repolarization of papillary muscles.[21]

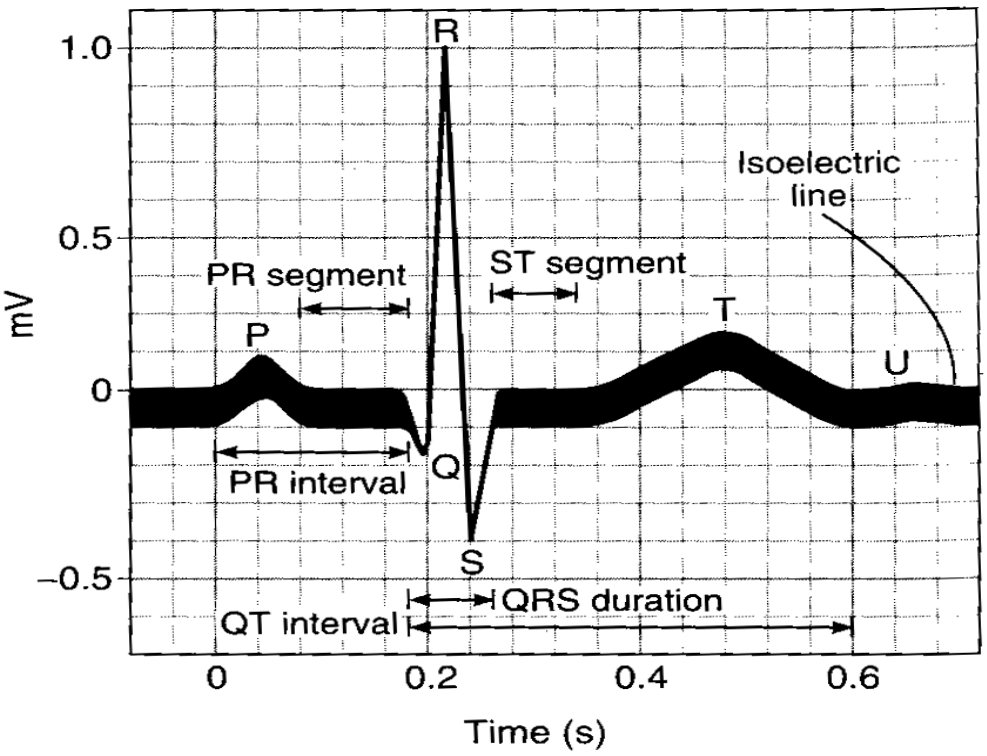


Figure 3: Waves & Segments of a Normal ECG

ELECTROCARDIOGRAPHIC LEADS[22] :

Electrocardiographic leads may be divided into two groups depending upon their orientation to the heart.

1. Frontal plane leads: These are oriented in the forntal plane or coronal plane of the body and consist of standard limb leads I, II, III and aVR, aVL and aVF.
2. The horizontal plane leads: These are oriented in the transverse or horizontal plane of the body and consist of precordial leads V1 to V6.[16]

Bipolar limb leads[21]**:**

Bipolar limb leads were used before unipolar leads were developed. Leads I, II, and III are regarded as Standard Limb Leads, each of which will record the differences in potential between two limbs. Since current flows only in the body fluids, the records obtained are those that would be obtained if the electrodes were at the points of attachment of the limbs, no matter where on the limbs the electrodes are placed.

In lead I: The electrodes are connected so that upward deflection is inscribed when negative electrode is placed on the right arm and positive electrode on the left arm.

In lead II: The electrodes are connected so that upward deflection is inscribed when negative electrode is placed on the right arm and positive electrode on the left foot.

In lead III: The electrodes are connected so that upward deflection is inscribed when negative electrode is placed on the left arm and positive electrode on the left foot.

Unipolar leads

An additional nine unipolar leads are also used in clinical electrocardiography. There are 3 unipolar limb leads : VR (right arm), VL (left arm) and VF (left foot) and six unipolar chest leads (precordial leads) which are designated as V1, V2, V3, V4, V5 and V6 [21]

Augmented Limb Leads:

The three augmented limb leads were devised by Emmanual Goldberger.[16]

They are named as aVR, aVL & aVF. The augmented limb leads are the recordings between one limb and other two limbs. This arrangement increases the size of potential by 50% without any change in configuration from non-augmented record.[21]

aVR: It is the augmented unipolar right arm lead. This lead is considered to be oriented to or faces the heart from the right shoulder. This lead is usually oriented to the cavity of the heart. Thus, all deflections – P wave, QRS complex and T wave are normally negative in this lead.[22]

aVL: It is the augmented unipolar left arm lead. This lead is considered to be oriented to or faces the heart from the left shoulder. This lead is usually oriented to the anterolateral or superior surface of the left ventricle.[22]

aVF: It is the augmented unipolar left leg lead. This lead is considered to be oriented to the inferior surface of the heart.[22]

Unipolar leads can also be placed at the tips of the catheters and inserted into esophagus and heart.[21]

Precordial Leads or Chest Leads:

The chest leads V1- V6 record electrical activity from the chest.

- V1 - Fourth intercostal space at the right sternal border.
- V2 - Fourth intercostal space at the left sternal border.
- V3 – mid way between lead V2 and lead V4 electrode position.
- V4 – fifth intercostal space in the left midclavicular line
- V5 - fifth intercostal space in the left anterior axillary line
- V6 - fifth intercostal space in the left mid axillary line.[23]

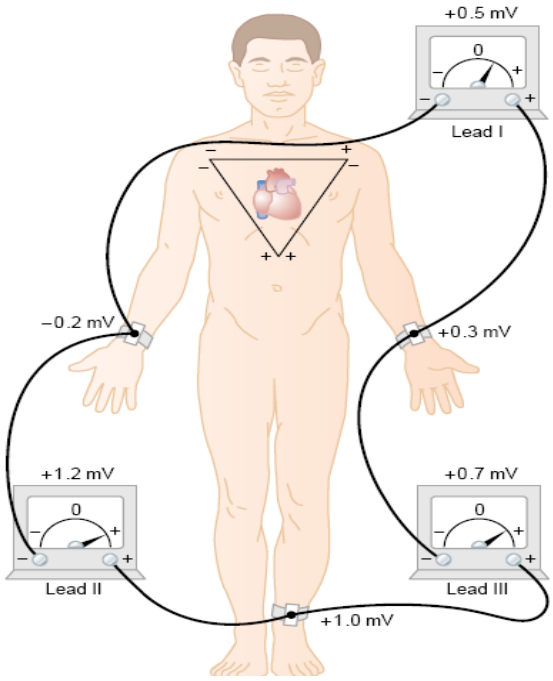


Fig 4: Standard Bipolar Limb Lead

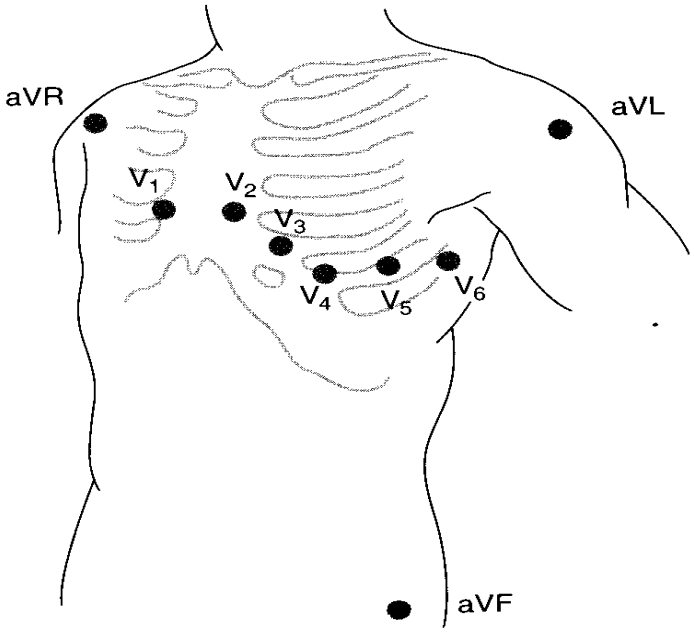


Fig 5: Unipolar electrocardiographic leads

ECG Interpretation[21]:

Before recording ECG, the electrocardiograph has to be properly calibrated so that standardization mark is 10mm tall. One has to check also for limb lead reversal and ECG artifacts.

P wave:

Normally, P wave does not exceed 2.5 mm in amplitude and less than 3mm wide in all leads.

It does not exceed 0.11sec in duration. Tall peaked P waves may be a sign of right atrial overload (P pulmonale). Wide P waves (P mitrale) are seen with left atrial abnormality.

PR Interval:

The normal PR interval (measured from the beginning of P wave to beginning of QRS complex) is 0.12 to 0.2 seconds. A uniformly prolonged PR interval is often referred to as first degree AV block. A short PR interval with retrograde P waves generally indicates an ectopic.

Width of QRS complex:

Normally QRS width is 0.04 to 0.1 second

Following are the causes for wide QRS complex

1. Bundle branch blocks including classical RBBB, LBBB patterns.
2. Toxic conduction delays due to some extrinsic factors such as hyperkalemia or drugs.
3. Beats arising in the ventricles which may be ventricular premature beats or ventricular ectopic beats.
4. WPW type pre excitation

Intervals	Normal Duration (s)		Events in the Heart During Interval
	Average	Range	
PR interval[a]	0.18[b]	0.12–0.20	Atrial depolarization and conduction through AV node
QRS duration	0.08	to 0.10	Ventricular depolarization and atrial repolarization
QT interval	0.40	to 0.43	Ventricular depolarization plus ventricular repolarization
ST interval (QT minus QRS)	0.32	...	Ventricular repolarization

[a]Measured from the beginning of the P wave to the beginning of the QRS complex.
[b]Shortens as heart rate increases from average of 0.18 s at a rate of 70 beats/min to 0.14 s at a rate of 130 beats/min.

QRS voltage:

One has to look for signs of right or left ventricular hypertrophy. Low voltage may be due to pericardial effusion or pleural effusion.

T wave:

Normally, they are positive with positive QRS complex in Lead II and LeadsV3 to V6 in adults. They are negative in Lead aVR.

QT interval:

A prolonged QT interval may be due to electrolyte disturbances (hypocalcemia or hypokalemia), drug effects or myocardial infarction. Shortened QT intervals are seen with hyperkalemia and digitalis effect.

QTc interval:

It is basically the QT interval corrected for Heart rate**.**

It is calculated by using Bezzett's formula

$$QTc = \frac{\text{QT Interval}}{\sqrt{\text{RR interval}}}$$

The cut off point for QTc is 390 ms.

Mean QRS axis:

The mean QRS axis is determined in frontal plane. By inspection, it is possible to decide whether the axis is normal or not (it is normally between -30 to +100, or whether left or right axis deviation is present).

Abnormal Q waves:

Prominent Q waves in leads II, III and aVF may indicate inferior wall infarction. Prominent Q waves in leads I, aVL, and V1 to V6 may indicate anterior wall infarction.

ST segment:

One has to look for ST elevation or depression. Horizontal ST segment depression over 1.0mm, down sloping ST segment depression over 1 mm with J point depressed by 2 mm or more beyond 0.8 sec from the J point is considered to be significant and indicates myocardial hypoxia.

U wave:

One has to look for prominent U waves. These waves may be a sign of hypokalemia or drug effect or toxicity.

Heart rate:

The following methods can be used to measure the heart rate.

1. When heart rate is regular, the number of large boxes between two successive QRS complexes are counted. Then, this is divided by a constant.
2. If heart rate is irregular, an average rate is determined by counting the number of cardiac cycles every 6 seconds and multiplying this number by 10.

If the heart rate is faster than 100beats per minute, tachycardia is present. Heart rate slower than 60 beats per minute means bradycardia is present.

Rhythm:

The rhythm is usually of

1. Normal sinus rhythm
2. Sinus rhythm with extra ectopic beat such as atrial/ventricular premature beats.
3. Extreme ectopic rhythms such as atrial fibrillation or flutter, ventricular tachycardia or an AV junctional escape rhythm.

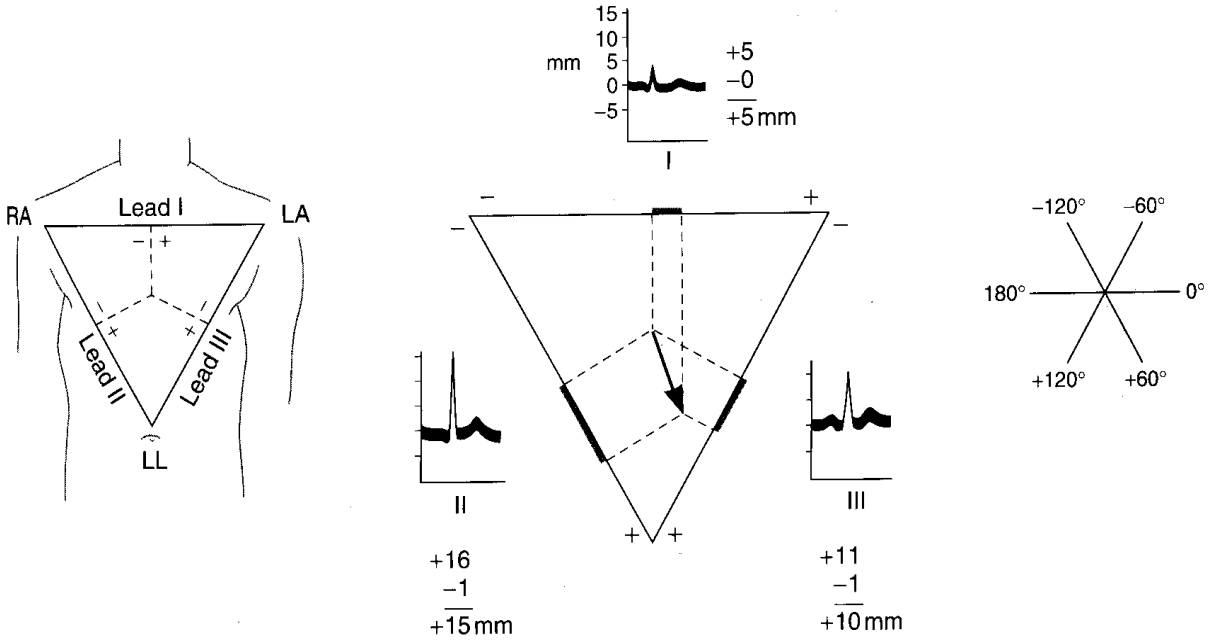
Bipolar limb leads & cardiac vector[21]:

As the standard limb leads are the records of the potential differences between two points, the deflection in each lead at any instance indicates the magnitude and direction in the axis of the lead of the electromotive force generated in the heart (cardiac vector or axis).

The vector at any given moment in two dimensions of the frontal plane can be calculated from any two standard limb leads if it is assumed that the three electrode locations from the points of an equilateral triangle (Einthoven's triangle) and that the heart lies in the center of the triangle. These assumptions are not completely warranted but calculated vectors are useful approximations.

An approximate mean QRS vector is often plotted by using the average QRS deflection in each lead. This is a mean vector as appose to an instantaneous vector. The average QRS deflection should be measured by integrating the QRS complexes. However, they can be approximated by measuring the net differences between positive and negative peaks of QRS.

The normal direction of the mean QRS vector is generally said to be -30 to +110 on the coordinate system shown in figure 6. The left or right axis deviation is said to be present if the calculated axis falls to the left of -30 or to the right of +110 respectively. Right axis deviation suggests right ventricular hypertrophy and left axis deviation may be due to left ventricular hypertrophy. But, there are better more reliable ECG criteria for determining ventricular hypertrophy.



A- Einthoven's triangle **B-Calculation: QRS vector** **C- Reference axis**

FIG 6: Cardiac Vector

Einthoven's triangle[21] [Fig-6A]:

Perpendiculars dropped from mid points from the sides, of the equilateral triangle intersect at the center of electrical activity. (RA – Right arm, LA Left arm,LL – Left leg).

Calculation of mean QRS vector: **[Fig-6B]**

In each lead, distances equal to the height of the R Wave minus the height of the largest negative defection in the QRS complex are measured from the mid point of the side of the triangle representing that lead. An arrow drawn from the center of electrical activity to the point of intersection of perpendiculars extended from the distances measured off on the sides represents the magnitude and direction of the mean QRS vector.

Reference axis for determining the direction of the vector: [Fig-6C]

A major purpose of recording ECG in clinical practice is to help the clinician in the diagnosis and prognosis of heart diseases.

The effect of pregnancy on the electrocardiogram has been a subject of great interest since the early days of electrocardiography.[24]

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 is designed with an objective to determine electrocardiographic changes in normal pregnancy.[25]

This knowledge may be helpful in the prevention of gestational complications associated with an inadequate maternal hemodynamic adaptation.[26]

OBJECTIVES

1. To determine ECG patterns in 1st, 2nd and 3rd trimester of pregnancy and to compare with that of non-pregnant women.
2. To compare ECG changes between 1st, 2ndand 3rd trimester of pregnancy in different women.

REVIEW OF LITERATURE

ECG CHANGES DURING PREGNANCY

Halphen C, Leuludec D, Valet R, Haiat R conducted Electrocardiographic study of left ventricular performance in normal pregnancy. In the study they stated that the heart rate raised significantly from third to ninth month and then fell during the postpartum period. 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 fetal growth.[27]

Joseph J. Rovinsk & Herbert conducted a study on cardiovascular hemodynamics in pregnancy. They studied certain significant parameters of cardiovascular hemodynamics in 6 normal non-gravid control subjects, 34 women at different stages of singleton pregnancy & 20 women with twin pregnancy. They observed 15% increase in mean cardiac rate, 30% increase in mean cardiac stroke volume & 16% increase is mean circulation time changed in the same direction & approximately to the same degree in both single & twin pregnancies.[28]

M. Lechmanova, A. Parizet et al studied changes of the electrical field resulting from the changed spatial position of the heart during last trimester of pregnancy in healthy women. They 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.

They observed several significant changes of the electrical field in the pregnant women. They were as follows:

1. Increase in the heart rate
2. Shortening of A V conductance
3. Prolongation of QT interval normalized for the heart rate ie, QTc
4. Change in the ventricular depolarization & repolarisation pattern.[29]

Clapp III JF & Capless E. conducted a study on cardiovascular function before, during and after the first and subsequent pregnancies. It had been stated that the heart rate increased early during pregnancy, peaked at term by about 15 per min above prepregnancy levels, returned to base line by 12 weeks of postpartum and remained at that level throughout the first year of postpartum.[30]

Carla A conducted a longitudinal study of maternal hemodynamics during normal pregnancy. They had concluded from the study that mean heart rate increased significantly from 10-18 to 34-42 weeks of gestations by 5.5 $\pm$ 1.5 beats per minute. This increase was not purely linear but slightly curved.[31]

Carruth JE & Shierly studied the electrocardiographic changes in normal pregnancy in 157 women without clinical evidence of heart diseases. They recorded & analysed 12 lead ECG during each trimester of pregnancy. They recorded the following observations:

1. Increase in the heart rate with greater increase in the third trimester.
2. No clinically significant change in cardiac rhythm or ECG intervals.
3. The mean PR interval was slightly shorter at third trimester as compared to first and second trimesters.[32]

Stein PK & Hagley MT studied the changes in 24 hours heart rate variability during normal pregnancy. They observed the mean heart rate increased in early pregnancy. There was only slight increase in mean heart rate in the beginning of 2nd trimester and midpregnancy. Heart rate was increased significantly in late pregnancy when compared to the non pregnant state.[33]

Curis Lester Mendelson discussed various etiologic types of heart diseases encountered during pregnancy in terms of medical care, cardiovascular surgery & obstetric management. He also studied the electrocardiographic changes in normal pregnancy. He concluded that as pregnancy progresses a large Q wave may develop in lead III & T – wave may become negative in lead III. The mean electrical axis shifts to left with advancing pregnancy until the latter part of the third trimester, when it shifts back to the right.[34]

Patricia L, Peter P, Robert E and Jeffery N conducted a study on changes in 24-hour heart rate variability during normal pregnancy. By 24 hour Holter monitoring, recordings were obtained from 8 healthy pregnant volunteers during 6, 10, 18 &34 weeks of gestation. They concluded that changes in heart rate variability and respiratory rates between subsequent stages during pregnancy varied in both magnitude and direction.[35]

Boyle D M & Jones RLL conducted a study on electrocardiographic ST segment in normal pregnancy. The study group consisted of 54 subjects attending the antenatal clinic at the Middle sex Hospital. In their study, 4% of subjects showed ST segment depression in excess of 0.5mm and in none of these, the depression was 1mm. In each subject, the ST segment depression occurred in one lead only i.e in I, II, aVF or V6. The ST segment depression occurred in the first trimester in 3 subjects and in the second trimester in 1

subject. In each case, ST segment sloped upwards from J point did not resemble "ischaemic" depression and would not normally have been considered pathological.[24]

Ville Jean- Claude et al studied the effects of pregnancy on electrocardiogram in healthy subjects during strenuous exercise. 12 lead electrocardiograms were obtained during rest, exercise, and recovery. They found T – wave inversion in V_2 which was more frequent in the pregnant than the non-pregnant subjects. 2 patients had marked T wave peaking and 1 had a biphasic T wave in V2. The subjects "late" in pregnancy had significantly fewer Q waves in Leads II, III & aVF than in the non-pregnant subjects.[36]

Edemka DB, Ekong MN et al studied the electrocardiogram of pregnant Nigerian women. The QRS axis showed significant leftward deviation in pregnant subjects as compared to non-pregnant subjects. The magnitude of deviation apparently increased as pregnancy progressed. The amplitude of different waves were lesser in the pregnant women when compared to non pregnant women.[37]

Wilkinson R. Gyaneshwar, C Mcwsker and Ryan H published a case report on the management of normal pregnancy with idiopathic long QT syndrome. The syndrome was defined as QT interval corrected for Heart rate (QTc) of > 0.44 sec.[38]

Christopher FC & Gerlic FM in their review article commented that there were both size & position changes of the heart which could lead to ECG appearance. The heart is enlarged by both chamber dilation & hypertrophy. Dilatation across the tricuspid valve could initiate mild regurgitant flow causing a normal grade I & II systolic murmurs. Upward displacement of the diaphragm by the enlarging uterus caused the heart to shift to left & anteriorly so that apex beat was moved upward & outward. These changes led to

common ECG finding of left axis deviation, sagging ST segments & frequently inversion or flattening of T-wave in lead III. [39]

Mark W. Comlinson in his book of "High risk pregnancy" had commented that normal physiologic changes which occur during gestation could aggravate underlying cardiac disease & lead to associated morbidity & mortality. [40]

Grospietsch G. studied normal physiological changes in cardiovascular system during pregnancy. They had observed increase in cardiac output, vasodilatation & hypervolumia. They were of clinical relevance as they were able to aggravate, mask or even imitate cardiovascular diseases. There was an increase in the cardiac size & volume during pregnancy. Furthermore, enlargement of the uterus may lead to diaphragmatic elevation & barrel shaped thorax followed by rotation of the cardiac axis to the left to 15° to 30°. Hemodynamic changes led to auscultatory and ECG changes (i.e, S1- Q3 type, ST depression, T – wave flattening). In addition, there was high incidence of functional systolic & diastolic sounds during pregnancy which were also able to imitate the cardiovascular diseases. He concluded that the physiological changes in pregnancy are similar to those changes which can be seen during heavy exercise. This will in turn result in continuous cardiac stress during whole pregnancy.[41]

R. Thorne, A Varnava et al conducted a study to see whether the pregnancy was well tolerated in subjects with hypertrophic cardiomyopathy. They concluded that most women with hypertrophic cardiomyopathy tolerated pregnancy well. However, sometimes rare complications may occur. Therefore, planned delivery & fetal monitoring were required for some patients. [42]

Philip J Podrid observed ECG changes during normal pregnancy. They were as follows:

1. Shortening of PR & QT interval, may accompany the increase in the heart rate.
2. Frontal lead axis changes were rare despite significant elevation of diaphragm when seen, slight right ward shift were more common than leftward deviation.
3. Non specific abnormalities of ST segment & T wave appeared in 4 to 14% of the pregnancy. These changes predominated in left precordial leads & resolved in the majority of subjects after delivery.[43]

Ozmen N conducted a study on incidence of P-wave dispersion during pregnancy. The P-wave and QT dispersion during pregnancy were investigated in healthy pregnant women (n=162) and healthy non pregnant women (n=150). It was concluded that shortening of the minimum P-wave duration resulted in an increased P-wave dispersion during pregnancy and QTc interval was prolonged in pregnant women than in control group.[2]

Nakagawa M et al conducted a study on new ventricular arrhythmias in pregnancy. The study group comprised of 11 pregnant women aged 35.4 $\pm$ 3.1yrs. It was concluded from the study that the onset of their first episode of ventricular arrhythmias were distributed equally over 3 trimesters. Ventricular premature contractions and Ventricular tachycardia exhibited a monographic configuration in all subjects.[8]

Kittnar O & Mleck M conducted a study on dispersion of QT interval. QT interval dispersion is conventionally interpreted as a result of repolarization heterogeneity in ventricular myocardium. The observations from healthy pregnant women were compared with those who were treated with dosulepine. QT interval was measured from unipolar

chest leads. It was concluded that the QTd was significantly increased in many physiological and pathological states.[44]

Thomson K J, Cohen E M and Hamilton B E conducted a study on electrocardiographic changes due to effects of circulation in pregnancy. The Study group comprised of 288 pregnant women. It was noted that slurring and notching of QRS axis was quite common. T wave variations, flat and biphasic T waves occurred in 51 patients.[45]

Iwobi NN and Dapper DV studied the effect of normal pregnancy on the heart rate, respiratory rate, QRS axis and QRS complex duration of the ECG in Nigerian women. The study group consisted of 41 pregnant women and control group comprised of 39 non pregnant age and height matched Nigerian women. The heart rate increased in early pregnancy, peaked at term by about 15 per minute above prepregnancy levels & returned to base line by 12 weeks of postpartum. The QRS axis showed significant left ward shift of the electrical axis of ventricular depolarization, which was attributed to the upward movement of the diaphragm. There was an increased an ventricular voltage due to increased ventricular mass.[46]

Lechmanova M et al. conducted a study on QT dispersion and T-loop morphology in late pregnancy and after delivery. The electrocardiographic changes were obtained from 37 healthy women in the 36^{th} to 40^{th} weeks of pregnancy and 2 to 6 days after delivery. Electrocardiographic changes due to an altered thoracic geometry was evaluated to study the possible physiological determinants of QT prolongation. There was an increase in QT interval as well as prolongation of QTc interval during late pregnancy. Increase in QT interval as well as prolongation of QTc interval may be due to changed spatial

arrangement of chest organs & changed electrical properties of the myocardium during pregnancy. This in turn may be due to changed sympathetic and hormonal modulation of cardiac electrical activity during pregnancy.[47]

Bruch GE conducted a study on the spatial vectrocardiogram and mean ventricular gradient in normal pregnant women. They observed that Q wave was present in lead III of the electrocardiogram in about 23% subjects during pregnancy. This may be due to changes in spatial orientation and contour of the heart which in turn resulted in part from the changes in cardiac position and haemodynamic alterations associated with pregnancy.[18]

Misra J conducted an electrocardiographic study in pregnant women in normal and toxemia of pregnancy. Negative T wave in lead III was observed in 70% subjects of normal pregnancy of which in 23% of this negative T wave became positive in postpartum period. This may be explained by temporary increase in blood volume during pregnancy. This may result in temporary ischaemia leading to T wave inversion.[25]

Singh AD conducted a study on electrocardiographic findings at term, labour and immediate postpartum. He observed that electrical axis of +60 degree corresponding to semivertical heart position was commonest except in two subjects who showed horizontal heart position. Left axis deviation had been described during pregnancy as early as first two trimesters. This change in electrical axis was attributed to the elevation of diaphragm as pregnancy advances.[48]

MATERIALS AND METHODS

A cross sectional study was conducted in the Department of Physiology, Shri B.M.Patil Medical College,Hospital and Research Centre, Bijapur. Duration of the study was one year from December 2008 to November 2009.

The study was undertaken to determine the ECG changes in 1^{st}, 2^{nd} & 3^{rd} trimesters of pregnancy. The observations were compared with age matched healthy non pregnant women.

Method of Collection of data:

Study Group: 150 pregnant women in the age group of 20-35yrs who were attending the OPD of OBGy of Shri B.M. Patil Medical College 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.

Control Group: It 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 proforma was used to record the relevant information from each selected individual who had fulfilled inclusion criteria. The subjects who had exclusion criteria were dropped from the study. 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.

Inclusion Criteria

Apparently healthy subjects of Indian origin were included in the study. The apparent health status of the subject were determined through thorough clinical examination and history taking.

Exclusion Criteria

The Following Subjects were excluded from the study.

1. Subjects with history or clinical signs of cardiovascular diseases.
2. Subjects with acute respiratory infection in the previous three months.
3. Subject with history of diabetes mellitus, hypertension.
4. Subjects with history of tobacco consumption in any form.
5. Subjects with history of alcohol intake.
6. Subjects with any endocrine disorders.
7. Subjects with obesity.
8. Subjects with moderate to severe anemia.

Following parameters were recorded in each subject:

1) Height (in centimeters): this was measured with subject without footwear nearest to 0.1 cm.
2) Weight (in kilograms): subjects were measured in standardized machine with minimum clothing nearest to 0.1 Kg
3) Body surface area in square meters using Dubois nomogram.
4) Body Mass Index in kilograms/ meter^2 using Quetelet Index.
5) Resting pulse rate was expressed as bpm. It was examined by compressing radial artery in semi pronated forearm and slightly flexed wrist of subject.

6) Blood pressure (in mm Hg). It was measured by mercurial sphygmomanometer (diamond make) by palpatory and auscultatory method.

Recording of Electrocardiogram [22]

Instrument

Electrocardiograph is a sophisticated galvanometer. It is a sensitive electromagnet, which can detect and record changes in electromagnetic potential. It has positive and negative poles. The wire extension from these poles have electrodes at each end, a positive electrode at the end of extension from the positive pole and a negative at the end of extension from the negative pole. The paired electrodes together constitute an "Electrocardiographic Lead".

When paired electrodes are oriented in any particular direction, the theoretical straight line joining the electrodes is known as "Axis" of that lead or "Lead Axis". A Lead so placed will detect and transmit any changes in electrical potential which occurs between its electrodes. (Make: Maestros Magic R)

Electrocardiographic paper

The paper used is thermosensitive. The electrocardiographic recording paper has ruled lines. They divided in to large and small squares. The large squares are of width of 5mm. Each small square is 1mm in width.The squares from grid facilitate the measurement of:

1) Timed parameters (horizontal measurement) &

2) Deflection amplitude (vertical measurement)

Electrocardiogram is conventionally recorded at a paper speed of 25 mm per second. At this paper speed, five large squares represent one second, one large square represents 0.2 second (1/5 of second) and one small square represents 0.04 second (1/25 of second). In one minute, ECG paper moves a length of 1500mm. Each 1mm vertically represents 0.1mv.

Recording of ECG:

ECG was recorded after giving 5 minutes of rest to the subject to allay anxiety. ECG was recorded in all 12 leads i.e, 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.

Analysis of ECG recording:

ECG recorded was evaluated for different parameters such as heart rate, P wave, PR interval, QRScomplex, Q wave, T wave, QTc interval, axis deviation, R and S amplitudes and ST segment.

STATISTICAL ANALYSIS

The results were expressed as Mean $\pm$ SD for continuous data and number and percentages for categorial data. Z test was used for comparison between control and study groups and Z test was used for comparison within the study group. Categorial data was analysed by Chi- square test.

A ‘p’ value of 0.05 or less was considered as statistically significant.

FIG 7: Electrocardiograph (Make: Maestros Magic R)

FIG 8: Recording of ECG in a Pregnant women (Study Group)

FIG 9: Recording of ECG in a Non-Pregnant women (control group)

FIG 10: Showing ECG of Pregnant women (Study Group)

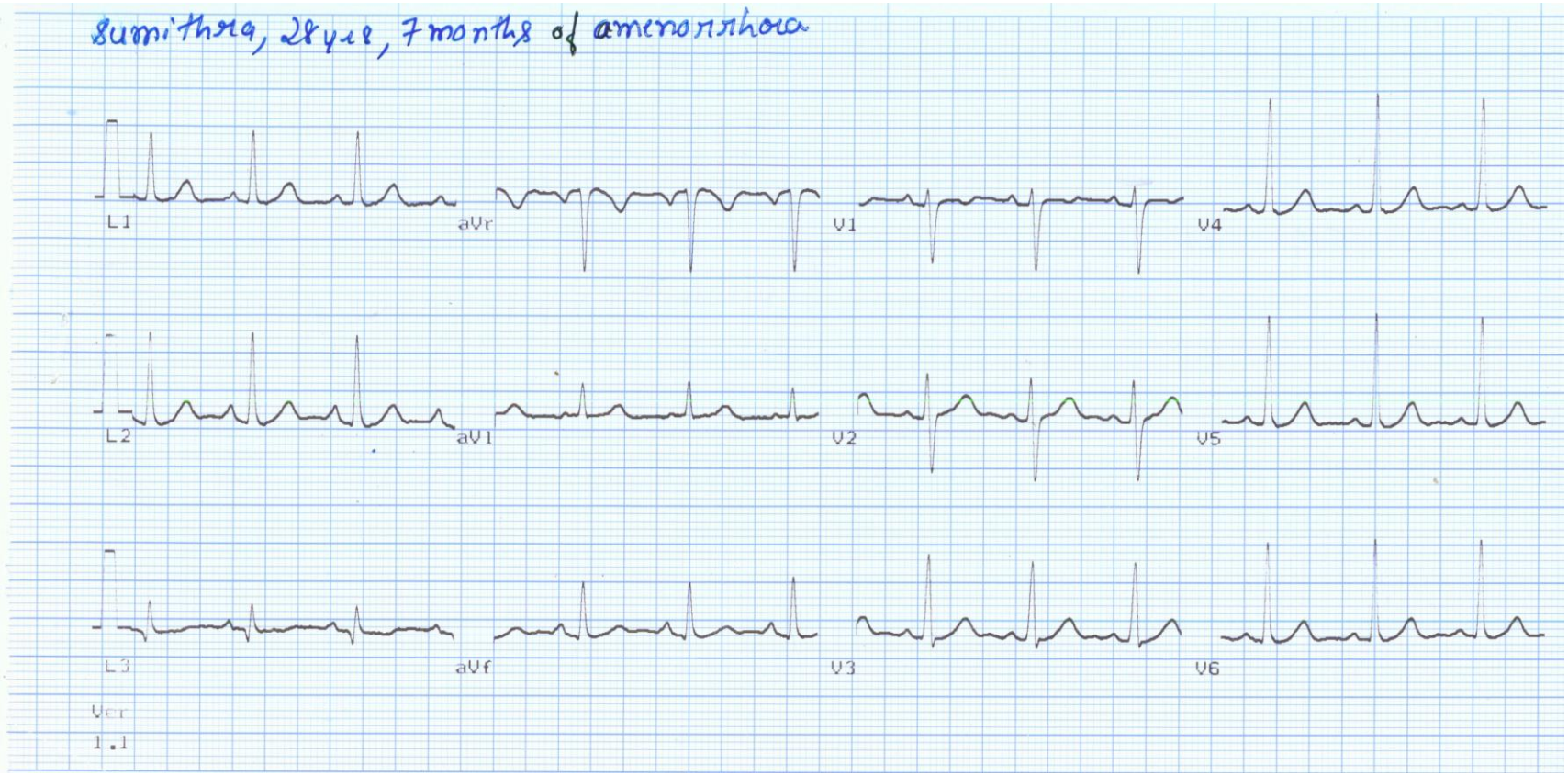
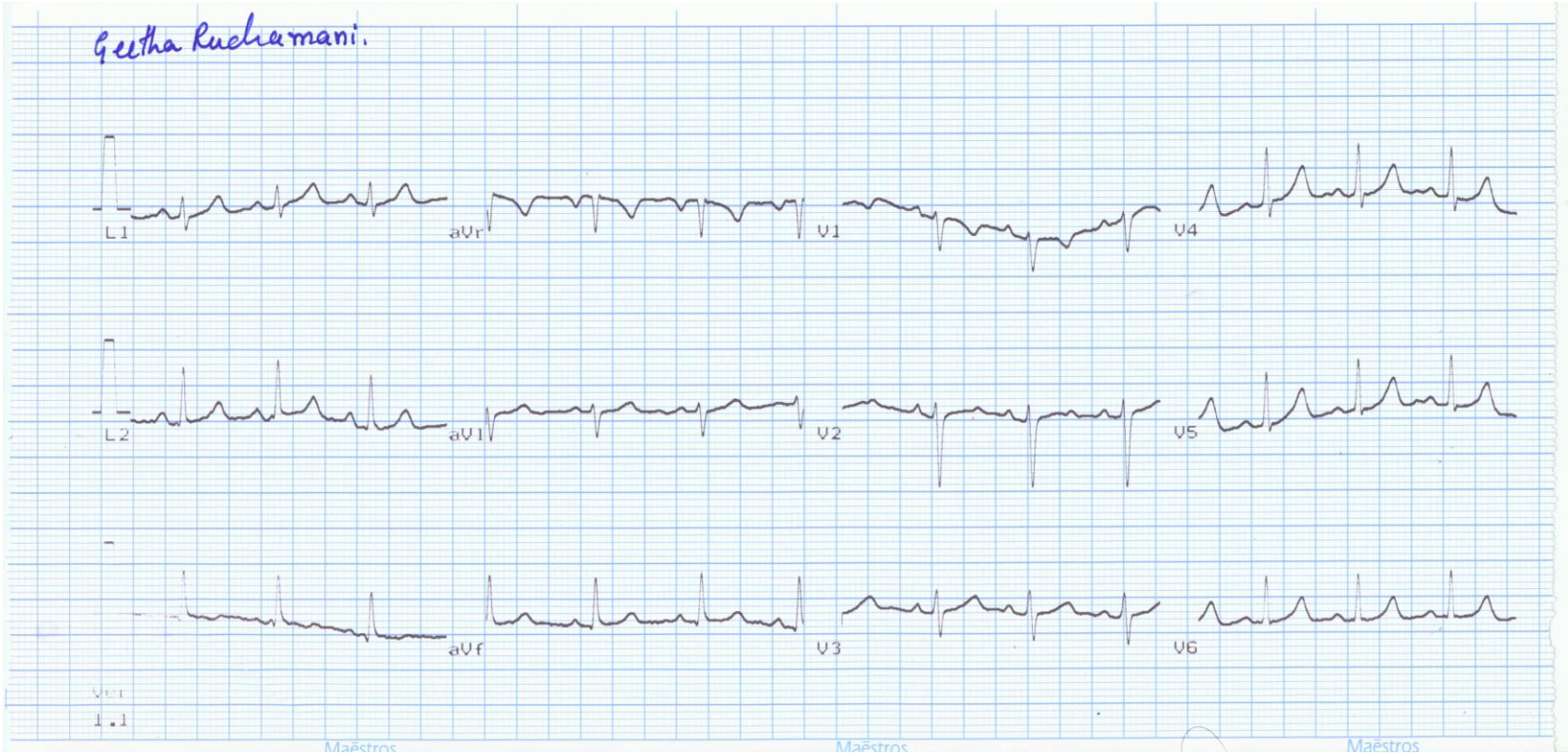


FIG 11: Showing ECG of Non-pregnant women (Control Group)



RESULTS

AGE:

Mean Age ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 26 ± 3.43, 25.12 ± 3.37, 24.12±4.02 and 24.58 ± 3.47 yrs respectively. (Table 1)

HEIGHT:

Mean Height ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 152.2 ± 0.06, 154.4 ± 0.05, 153.2±0.06 and 153.6± 0.06cms respectively. (Table 1)

WEIGHT:

Mean Weight ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 52.38 ± 3.71, 51.2 ± 7.00, 51.22±6.53 and 55.60 ± 9.19 Kgs respectively. (Table 1)

BMI:

Mean BMI ± SD of control, pregnant women in 1st , 2nd and 3rd trimesters were 22.63 ± 2.12, 21.75 ± 3.00, 21.81±2.46 and 23.96 ± 3.42 kg/m^2respectively.(Table 1)

BSA:

Mean BSA ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 1.48 ± 0.06, 1.48 ± 0.10, 1.53±0.45 and 1.53 ± 0.14 m^2 respectively. (Table 1)

PULSE RATE:

Mean Pulse rate ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 76.32 ± 4.12, 82.28 ± 7.84, 87.82 ± 8.70 and 95.12 ± 6.88 beats/ min respectively (Table 2). There was statistically significant increase in the pulse rate in 1st, 2nd and 3rd trimesters of pregnancy when compared to non pregnant women ($p<0.001$), (Table 3). There was also statistically significant increase in the pulse rate in 2nd and 3rd ($p<0.001$) trimesters when compared to 1st trimester of pregnancy and a statistically significant

increase in 3rd trimester ($p<0.001$) when compared to 2nd trimester of pregnancy. (Table 4)

SYSTOLIC BLOOD PRESSURE (SBP):

Mean SBP ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 118.66±4.40, 115.60 ± 7.67, 109.56 ± 5.68 and117.56 ± 7.94 mm Hg respectively (Table 2). There was a statistically significant decrease in SBP in 1st ($p<0.05$) and 2nd ($p<0.001$) trimesters of pregnancy when compared to non pregnant women (Table 3). Similarly, there was a statistically significant decrease in SBP 2nd trimester ($p<0.001$) when compared to 1st and 3rd trimesters of pregnancy (Table 4).

DIASTOLIC BLOOD PRESSURE (DBP):

Mean DBP ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 74.32 ± 4.75, 73.36 ± 5.97, 66.52± 5.68 and 75.60 ± 7.41 mm Hg respectively (Table 2). There was a statistically significant decrease in DBP in 2nd ($p<0.001$) trimester of pregnancy when compared to non pregnant women (Table 3). Similarly, there was a statistically significant decrease in the DBP in 2nd ($p<0.001$) trimester when compared to 1st and 3rd trimesters of pregnancy (Table 4).

PULSE PRESSURE(PP):

Mean PP ± SD of control, pregnant women in 1st, 2nd and 3rd trimesters were 44.34 ± 5.47, 42.44 ± 7.81, 43.20 ± 5.33 and 42.44 ± 8.33 mm Hg respectively(Table 2). There was no statistical significant increase or decrease between study and the control groups and also within the subgroups of study group ($p>0.05$), (Table 3 and 4).

MEAN ARTERIAL BLOOD PRESSURE (MAP):

Mean MAP ± SD of control, pregnant women in 1st , 2nd and 3rd trimesters were 88.94 ± 5.72, 88.32 ± 5.80, 80.9 ± 4.19 and 89.45 ± 7.26 respectively(Table 2). There was a statistically significant decrease in MAP in 2nd ($p<0.001$) trimester of pregnancy when compared to non pregnant women (Table 3). Similarly, there was a statistically significant decrease in MAP in 2nd trimester ($p<0.001$) when compared to 1st and 3rd trimesters of pregnancy (Table 4).

ELECTROCARDIOGRAPHIC FINDINGS:

Heart Rate:

Heart rate is expressed in terms of beats per minute. Heart rate in control, pregnant women in 1st, 2nd and 3rd trimesters were 75.68 ± 3.99, 82.28 ± 7.84, 88.24 ± 9.10 and 95.52 ± 7.04 bpm respectively(Table 5). Heart rate showed a statistically significant increase in 1st, 2nd and 3rd trimesters of pregnant women when compared to non pregnant women ($p <0.001$) (Table 6). Similarly, there was a statistically significant increase in heart rate in 2nd and 3rd trimesters ($p <0.001$) when compared to 1st trimester of pregnant women (Table 6). There was a statistically significant increase in heart rate in 3rd trimester ($p <0.001$) when compared to 1st trimester of pregnant women (Table 7).

P wave:

a. Duration:

P wave duration (sec) in control, pregnant women in 1st , 2nd and 3rd trimesters were 0.08 ± 0.01, 0.08 ± 0.01, 0.08 ± 0.01 and 0.07 ± 0.01 respectively (Table 5). The P wave duration among control and study group and within the subgroups of study group were not statistically significant ($p>0.05$) (Table 6 & 7).

b. Amplitude:

P wave amplitude (mv) in controls, pregnant women in 1st, 2nd and 3rd trimesters were 1.00 ± 0.17, 1.00 ± 0.23, 1.02 ± 0.27 and 1.02 ± 0.28 respectively (Table 5). The P wave amplitude among control and study group and within the subgroup of study group were not statistically significant ($p>0.05$) (Table 6 & 7).

PR Interval:

PR interval (sec) measured in control, pregnant women in 1st, 2nd and 3rd trimesters were 0.15 ± 0.01, 0.14 ± 0.02, 0.14 ± 0.02 and 0.13 ± 0.02 respectively (Table 5). There was a 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 2nd ($p< 0.05$) and 3rd trimesters ($p< 0.001$) when compared to 1st trimester of pregnancy.

Q wave:

Occurrence of Q wave in limb leads

Lead I : (Table 8)

Control: Q wave was noted in 6% of the subjects.

1st Trimester: Q wave was noted in 4% of the subjects.

2nd Trimester: Q wave was noted in 8% of the subjects

3rd Trimester: Q wave was noted in 10% of the subjects

Occurrence of Q wave in lead I showed a slight increase in 2nd and 3rd trimesters of pregnancy when compared to 1st trimester of pregnancy and control group. But, there was no significant statistical difference between the groups ($p>0.05$), (Table 9).

Lead II: (Table 8)

Control: Q wave was noted in 26% of the subjects.

1st Trimester: Q wave was noted in 32% of the subjects.

2nd Trimester: Q wave was noted in 44% of the subjects

3rd Trimester: Q wave was noted in 52% of the subjects.

Occurrence of Q wave in lead II showed a statistically significant increase in 2nd ($p<0.05$) and 3rd trimesters ($p<0.001$) when compared to 1st trimester of pregnancy and control group (Table 9).

Lead III: (Table 8)

Control: Q wave was noted in 32% of the subjects.

1st Trimester: Q wave was noted in 36% of the subjects.

2nd Trimester: Q wave was noted in 48% of the subjects

3rd Trimester: Q wave was noted in 58% of the subjects

Occurrence of Q wave in lead III showed a statistically significant increase in 2nd ($p<0.05$) and 3rd trimesters ($p<0.001$) when compared to 1st trimester of pregnancy and control group (Table 9).

QRS Complex:

Duration of QRS Complex (seconds) in control, pregnant women in 1st, 2nd and 3rd trimesters were 0.08 ± 0.01, 0.08 ± 0.01, 0.08 ± 0.01 and 0.08 ± 0.01 respectively (Table 10). There was no statistical difference in duration of QRS complex among the control and study groups or within the subgroups of study group ($p> 0.05$) (Table 11 & Table 12).

QT Interval:

QT intervals (sec) in control, pregnant women in 1st, 2nd and 3rd trimesters were 0.35 ± 0.02, 0.35 ± 0.02, 0.35 ± 0.02 and 0.36 ± 0.01 respectively (Table 10). There was no statistical significant difference between the control and the study groups or within in the subgroup of study group ($p > 0.05$) (Table 11 & 12).

QTc Interval:

QTc interval (sec) in controls, pregnant women in 1st, 2nd and 3rd trimesters were 0.38 ± 0.01, 0.39 ± 0.01, 0.40 ± 0.01 and 0.41 ± 0.01 respectively (Table 10). QTc interval showed a statistically significant increase 1st, 2nd and 3rd trimester of pregnancy when compared to control group ($p < 0.001$) (Table 11). Similarly, a statistically significant increase in QTc interval was observed in 2nd and 3rd trimester ($p < 0.001$) of pregnancy when compared to 1st trimester and also in 3rd trimester ($p < 0.001$) of pregnancy when compared to 2nd trimester (Table 12).

ST segment:

In the present study, we observed ST segment depression in 2nd and 3rd trimesters of pregnancy i.e 4 subjects in 2nd trimester and 6 subjects in 3rd trimester. None of subjects in control group and in 1st trimester of pregnancy showed ST segment depression. There was no statistically significant difference between control and study groups or within the subgroups of study group (Table 13) ($p > 0.05$).

T wave:

a. Duration

T wave duration (sec) in control, pregnant women in 1st, 2nd and 3rd trimesters were 0.19 ± 0.04, 0.19 ± 0.04, 0.18 ± 0.03 and 0.19 ± 0.04 respectively (Table 14). The T

wave duration among control and study groups and within the subgroups of study group was not statistically significant ($p>0.05$) (Table 15).

b. Amplitude

T wave amplitude (mv) in control, pregnant women in 1st, 2nd and 3rd trimesters were 2.72 $\pm$ 1.01, 2.70 $\pm$ 1.11, 2.78 $\pm$ 0.89 and 2.45 $\pm$ 0.93 respectively (Table 14). The T wave amplitude among control and study groups and within the subgroups of study group was not statistically significant ($p>0.05$) (Table 15).

c. Abnormalities in the pattern of T wave

An upright T wave was observed in leads I and II in both control and study group which was normal.

T wave abnormalities were in Lead III (Table 16)

Lead III:

Control – 52% of the subjects showed normal (upright), 38% 0f the subjects showed inverted and 10% of them showed flat T wave pattern.

1st Trimester- 60% of the subjects showed normal (upright), 32% 0f the subjects showed inverted and 8% of them showed flat T wave pattern.

2nd Trimester- 48% of the subjects showed normal (upright), 42% 0f the subjects showed inverted and 10% of them showed flat T wave pattern.

3rd Trimester- 44% of the subjects showed normal (upright), 48% 0f the subjects showed inverted and 8% of them showed flat T wave pattern.

T wave abnormalities in lead III showed a statistically significant increase in both 2nd and 3rd trimesters of pregnancy when compared to non pregnant women.

QRS Frontal axis:

QRS Frontal axis (degrees) in control, pregnant women in 1^{st} , 2^{nd} and 3^{rd} trimesters were 64.56 $\pm$ 7.66, 60.48 $\pm$ 11.05, 55.70 $\pm$ 12.61 and 45.4 $\pm$ 22.54 respectively (Table 10). QRS Frontal axis showed a statistically significant decrease in 1^{st}, 2^{nd} and 3^{rd} trimesters ($p<0.001$) of pregnancy when compared to non pregnant women (Table 11). Similarly, a statistically significant decrease in QRS frontal axis was observed in 2^{nd} and 3^{rd} trimesters ($p<0.001$) when compared 1^{st} trimester and 3^{rd} trimester when compared to 2^{nd} trimester of pregnancy ($p<0.001$) (Table 12).

Amplitude of R wave:

Amplitude of R wave (mm) in control, pregnant women in 1^{st}, 2^{nd} and 3^{rd} trimesters were 12.72 $\pm$ 2.86, 12.58 $\pm$ 3.28, 12.50 $\pm$ 3.61 and 13.18 $\pm$ 3.68 respectively (Table 17). There was no statistically significant difference when compared between the control and the study groups or within the subgroups of study group ($p>0.05$) (Table 18 &19).

Amplitude of S wave:

Amplitude of S wave (mm) in control, pregnant women in 1^{st} , 2^{nd} and 3^{rd} trimesters were 10.12 $\pm$ 2.06, 9.84 $\pm$ 2.02, 9.96 $\pm$ 2.05and 9.82 $\pm$ 2.20 respectively (Table 17). There was no statistically significant difference when compared between the control and the study groups or within the subgroups of study group ($p>0.05$) (Table 18 &19).

Table 1: Mean ± SD and Range of Age and Anthropometric Parameters of subjects in Control and Study Groups

PARAMETERS	CONTROL		1ST TRIMESTER		2ND TRIMESTER		3RD TRIMESTER	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean± SD	Range
Age (yrs)	26 ± 3.43	20-34	25.12 ± 3.37	19 - 33	24.12±4.02	18-34	24.58 ± 3.47	19-32
Height (cms)	152.2 ± 0.06	142-164	154.4 ± 0.05	144-163	153.2±0.06	140-165	153.6± 0.06	1.3-1.64
Weight (kg)	52.38 ± 3.71	46 -60	52.2 ± 7.00	40-75	55.22±6.53	40–64	59.60 ± 9.19	42-82
BMI (kg/m^2)	21.63 ± 2.12	26.6-19.07	21.75 ± 3.00	17.6–30.4	23.81±2.46	17.39-27.2	25.96 ± 3.42	18.5-33.7
BSA (Sq m)	1.48 ± 0.06	1.36-1.58	1.48 ± 0.10	1.22-1.76	1.53±0.45	1.24-4.58	1.53 ± 0.14	1.26-1.8

Table 2: Mean ± SD and Range of Physiological Parameters of subjects in Control and Study groups

PARAMETERS	CONTROL		1ST TRIMESTER		2ND TRIMESTER		3RD TRIMESTER	
	Mean± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
PR(beats/min)	76.32 ± 4.12	66-84	82.28 ± 7.84	66-100	87.82 ± 8.70	74-106	95.12 ± 6.88	84-110
SBP (mm Hg)	118.66±4.40	107-126	115.60 ± 7.67	98-134	109.56 ± 5.68	98-122	117.56±7.94	98-144
DBP (mm Hg)	74.32 ± 4.75	58-82	73.36 ± 5.97	60-82	66.52± 5.68	60-74	75.60 ± 7.41	60-90
PP (mm Hg)	44.34 ± 5.47	32-56	42.44 ± 7.81	24-64	43.20 ± 5.33	32-60	42.44 ± 8.33	26-66
MAP (mm Hg)	88.94 ± 5.72	71.3-98.6	88.32 ± 5.80	72.6-98.6	80.90 ± 4.19	73.3-89.3	89.45 ± 7.26	73.3-108

Table 3: Test of Significance for Physiological Parameters Using Z Statistics between Control and Study groups

PARAMETERS	CONTROL & 1ST TRIMESTER		CONTROL & 2ND TRIMESTER		CONTROL & 3RD TRIMESTER	
	Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
PR (beats/min)	4.75	0.0001***	4.37	0.0001***	5.25	0.0001***
SBP (mm Hg)	2.44	0.0146*	3.01	0.0001***	1.38	0.17
DBP (mm Hg)	0.88	0.378	3.09	0.0001***	0.77	0.441
PP (mm Hg)	1.40	0.161	1.75	0.080	1.34	0.180
MAP (mm Hg)	0.53	0.596	2.59	0.0003*	0.47	0.638

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

Table 4: Test of Significance for Physiological Parameters Using Z Statistics within the subgroups of Study group

PARAMETERS	1ST & 2ND TRIMESTERS		1ST & 3RD TRIMESTERS		2ND & 3RD TRIMESTERS	
	Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
PR (beats/min)	3.34	0.0001***	7.75	0.0001***	4.1	0.0001***
SBP (mm Hg)	4.47	0.0001***	1.45	0.147	7.04	0.0001***
DBP (mm Hg)	6.47	0.0001***	2.11	0.034*	10.09	0.0001***
PP (mm Hg)	0.56	0.575	0.0	1	0.71	0.477
MAP (mm Hg)	7.33	0.0001***	1.12	0.262	10.21	0.0001***

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

Table 5: Mean ± SD and Range of Heart rate, P wave and PR Interval of subjects in Control and Study groups

PARAMETERS		CONTROL		1ST TRIMESTER		2ND TRIMESTER		3RD TRIMESTER	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
HR (beats/min)		75.68 ± 3.99	68 – 84	82.28 ± 7.84	66 – 100	88.24 ± 9.10	72 – 108	95.52 ± 7.04	84 – 112
P wave	Dur (sec)	0.08 ± 0.01	0.06 – 0.1	0.08 ± 0.01	0.04 – 0.1	0.08 ± 0.01	0.04 – 0.1	0.07 ± 0.01	0.04 – 0.1
	Amp (mv)	1.00 ± 0.17	0.5 – 1.5	1.00 ± 0.23	0.5 – 2	1.02 ± 0.27	0.5 – 2	1.02 ± 0.28	0.5 – 2
PR Interval (sec)		0.15 ± 0.01	0.12 – 0.16	0.14 ± 0.02	0.1 – 0.16	0.14 ± 0.02	0.1 – 0.16	0.13 ± 0.02	0.014 – 0.16

Table 6: Test of Significance of Heart rate, P wave and PR Interval using Z Statistics b/n Control and Study groups.

PARAMETERS		CONTROL & 1ST TRIMESTER		CONTROL & 2ND TRIMESTER		CONTROL & 3RD TRIMESTER	
		Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
HR (beats/min)		5.30	0.0001***	4.69	0.0001***	5.76	0.0001***
P wave	Dur (sec)	0.43	0.667	0.39	0.696	0.37	0.711
	Amp (mv)	0	1	0	1	0	1
PR Interval (sec)		2.53	0.0003**	2.51	0.0003**	2.11	0.0003**

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 Heart rate, P wave and PR Interval using Z Statistics within the subgroups of Study group.

PARAMETERS		1^{ST} & 2^{ND} TRIMESTERS		1^{ST} & 3^{RD} TRIMESTERS		2^{ND} & 3^{RD} TRIMESTERS	
		Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
HR (beats/min)		3.50	0.0001***	7.79	0.0001***	3.99	0.0001***
P wave	Dur (sec)	0.39	0.696	1.23	0.218	0.49	0.624
	Amp (mv)	0	1	0.40	0.689	0	1
PR Interval (sec)		2.51	0.012*	4.46	0.0001***	2.93	0.0034**

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

Table 8: Occurrence of Q Wave in limb leads in subjects of control and study groups.

LIMB LEADS	OCCURRENCE OF Q WAVE			
	Control	1st Trimester	2nd Trimester	3rd Trimester
I	3	2	4	5
II	13	16	22	26
III	16	18	24	29

Table 9: 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			
	Chi Square Value	Control&1st Trimester	Control & 2nd Trimester	Control & 3rd Trimester
I	0.82 p>0.05	NS	NS	NS
II	15.7 P<0.001	2.32 p>0.05	8.12* P<0.05	16.12*** P<0.001
III	13.5 P<0.05	4.32 p>0.05	9.23* P<0.05	17.3*** P<0.001

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

Table 10: Mean ± SD and Range of QRS Complex, QT Interval, QTc Interval and QRS frontal axis in subjects of Control and Study groups

PARAMETERS	CONTROL		1ST TRIMESTER		2ND TRIMESTER		3RD TRIMESTER	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
QRS Complex(sec	0.08 ± 0.01	0.06 - 0.1	0.08 ± 0.01	0.06 - 0.08	0.08 ± 0.01	0.06 - 0.1	0.08 ± 0.01	0.04 - 0.08
QT Interval (sec)	0.35 ± 0.02	0.32 - 0.36	0.35 ± 0.02	0.32 - 0.36	0.35 ± 0.02	0.32 - 0.36	0.36 ± 0.01	0.32 - 0.36
QTc Interval (sec)	0.38 ± 0.01	0.34 - 0.40	0.39 ± 0.01	0.35 - 0.42	0.40 ± 0.01	0.37 - 0.42	0.41 ± 0.01	0.37- 0.42
QRS frontal axis (degree)	64.56 ± 7.66	48 - 80	60.48 ± 11.05	26 - 82	55.70 ± 12.61	22 – 82	45.4 ± 22.54	-3 - 80

Table 11: Test of Significance for QRS Complex, QT Interval, QTc Interval and QRS frontal axis using Z Statistics between Control and Study groups

PARAMETERS	CONTROL & 1ST TRIMESTER		CONTROL & 2ND TRIMESTER		CONTROL & 3RD TRIMESTER	
	Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
QRS Complex (sec)	1.83	0.067	1.62	0.105	1.69	0.091
QT Interval (sec)	0.84	0.401	0.87	0.384	1.05	0.293
QTc Interval (sec)	3.96	< 0.0001***	4.44	< 0.0001***	3.88	< 0.0001***
QRS frontal axis (degree)	2.14	0.032*	3.95	<0.0001***	4.21	<0.0001***

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

Table 12: 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^{ST} & 2^{ND} TRIMESTERS		1^{ST} & 3^{RD} TRIMESTERS		2^{ND} & 3^{RD} TRIMESTERS	
	Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
QRS Complex (sec)	1.62	0.105	0.21	0.833	0.19	0.849
QT Interval (sec)	0.87	0.384	2.21	0.027	2.16	0.030
QTc Interval (sec)	4.44	0.0001***	6.39	0.0001***	3.88	0.0001***
QRS frontal axis (degree)	1.95	0.051	6.36	0.0001***	4.08	0.0001***

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

Table 13: Comparison of ST segment depression in Subjects of Control and Study groups

Particulars	Controls	1^{st} Trimester	2^{nd} Trimester	3^{rd} Trimester
ST segment depression	-	-	4	6

Table 14: Mean ± SD and Range of T wave in subjects of Control and Study groups

PARAMETERS		CONTROL		1ST TRIMESTER		2ND TRIMESTER		3RD TRIMESTER	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
T wave	Dur (sec)	0.19 ± 0.04	0.12 – 0.3	0.19 ± 0.04	0.12 – 0.3	0.18 ± 0.03	0.16 – 0.28	0.19 ± 0.04	0.12 – 0.3
	Amp (mv)	2.72 ± 1.01	1 - 5	2.70 ± 1.11	1 – 5	2.78 ± 0.89	1 - 5	2.45 ± 0.93	1 – 4

Table 15: Test of significance for T wave using Z Statistics between Control and Study groups

PARAMETERS		CONTROL & 1ST TRIMESTER		CONTROL & 2ND TRIMESTER		CONTROL & 3RD TRIMESTER	
		Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
T wave	Dur (sec)	0.19	0.275	0.21	0.833	0.19	0.275
	Amp (mv)	0.09	0.928	0.10	0.920	0.10	0.920

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

Table 16: Percentage of T wave Abnormalities in Limb Leads in Subjects of Control and Study groups

Leads	Control						1st Trimester						2nd Trimester						3rd Trimester					
	I		F		N		I		F		N		I		F		N		I		F		N	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
I	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
II	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
III	19	36	5	10	26	52	16	32	4	8	30	60	21	42	5	10	24	48	24	48	4	8	22	44

Note: I = Inverted, F = Flat, N = Normal

Table 17: Mean ± SD and Range of Amplitude of R and S waves in Subjects of Control and Study groups

PARAMETERS	CONTROL		1ST TRIMESTER		2ND TRIMESTER		3RD TRIMESTER	
	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
Amp of R wave (mm)	12.72 ± 2.86	6-19	12.58 ± 3.28	4 – 18	12.50 ± 3.61	4 – 19	13.18 ± 3.68	4 – 18
Amp of S wave (mm)	10.12 ± 2.06	5-16	9.84 ± 2.02	4 – 14	9.96 ± 2.05	4 – 13	9.82 ± 2.20	4 – 14

Table 18: Test of significance for Amplitude of R and S wave using Z Statistics between Control and Study groups

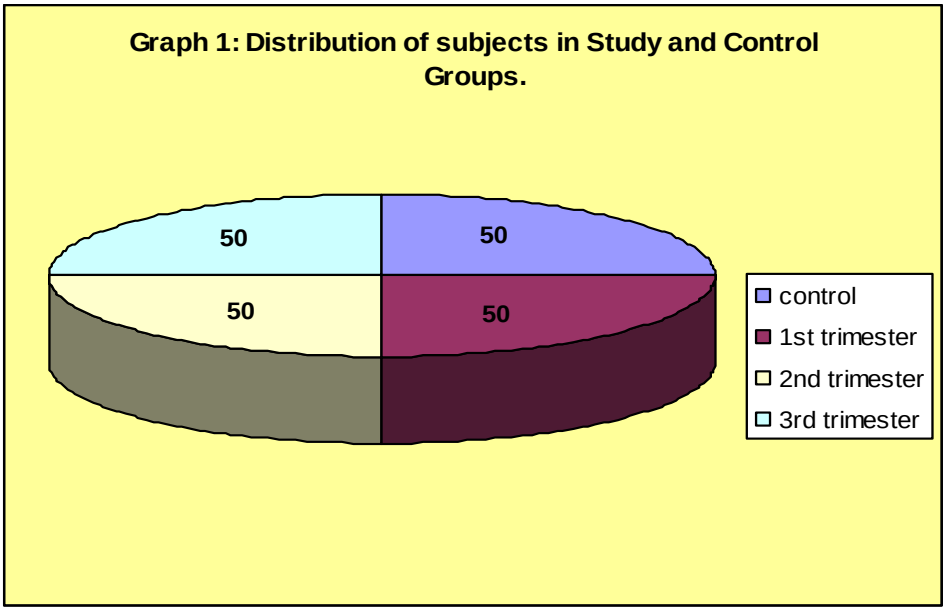
PARAMETERS	CONTROL & 1ST TRIMESTER		CONTROL & 2ND TRIMESTER		CONTROL & 3RD TRIMESTER	
	Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
Amp of R wave (mm)	0.22	0.825	0.21	0.833	0.21	0.833
Amp of S wave (mm)	0.68	0.496	0.68	0.496	0.65	0.515

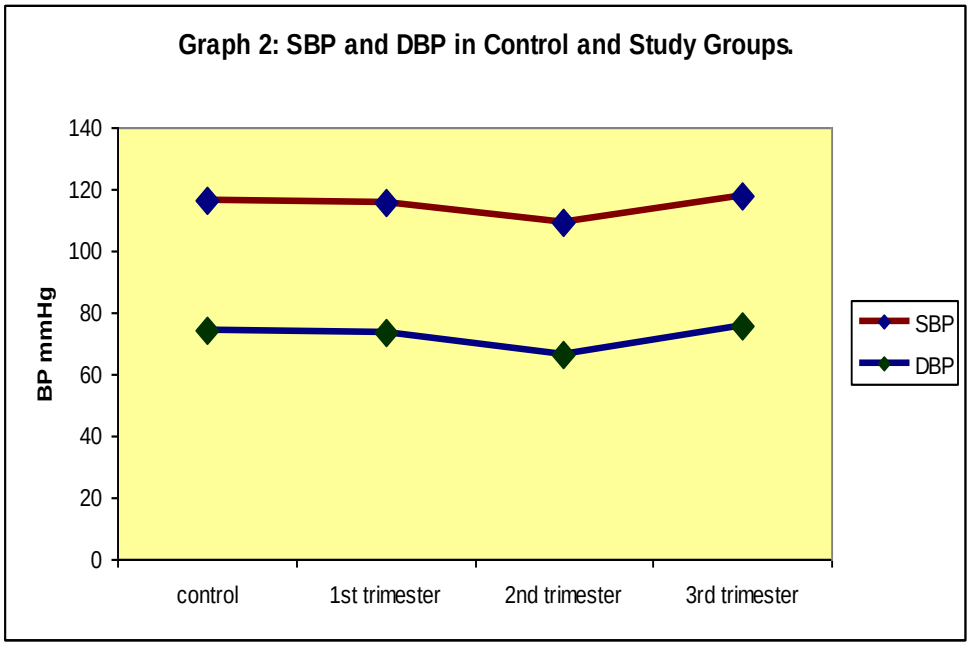
p>0.05: Not Significant, *p: <0.05: Significant, ** p: <0.01: Highly significant, *** p: <0.001: Very highly significant.

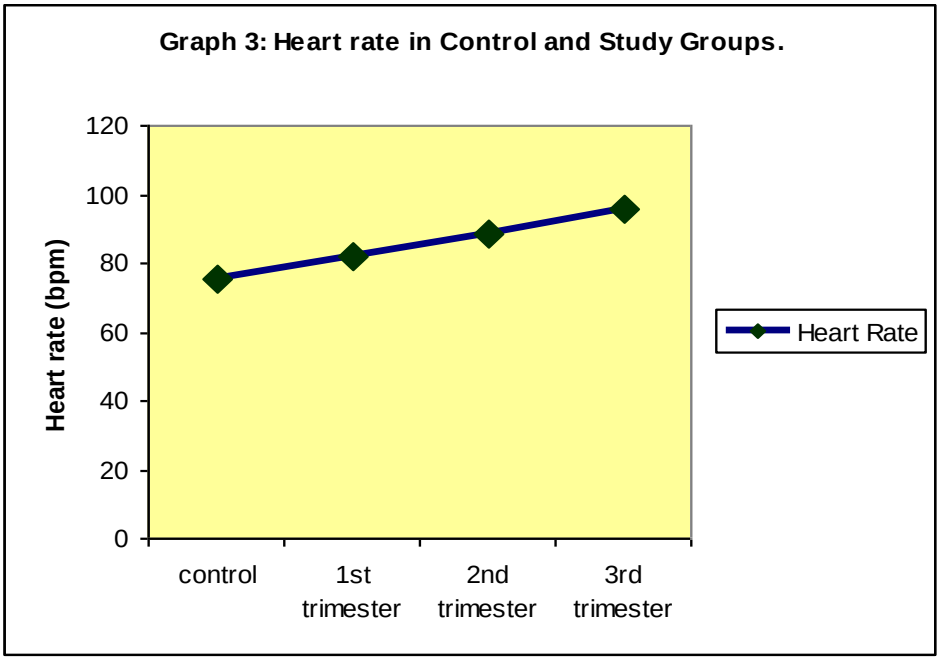
Table 19: Test of significance for Amplitude of R and S wave using Z Statistics between subgroups within the Study group

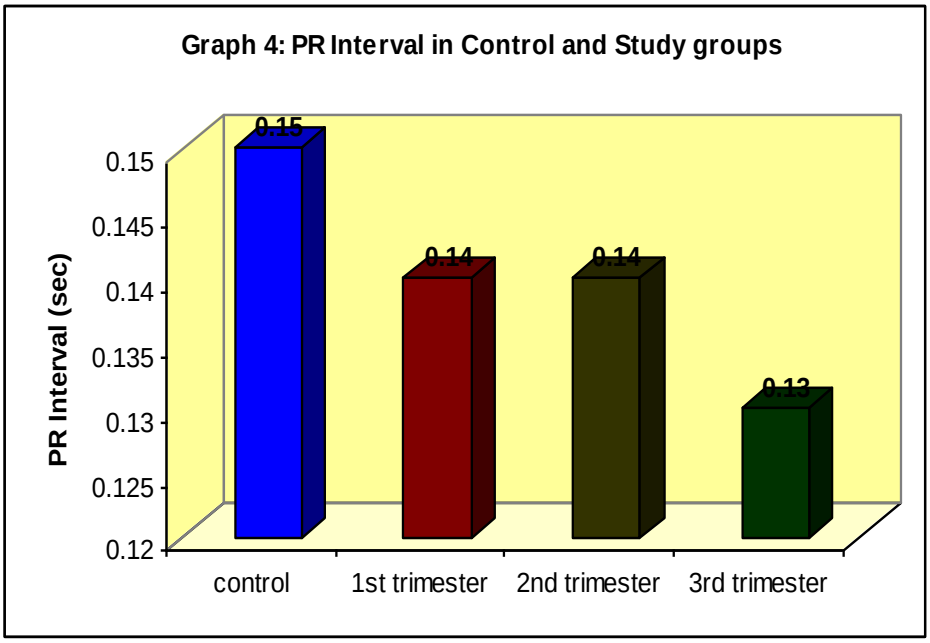
PARAMETERS	1ST & 2ND TRIMESTERS		1ST & 3RD TRIMESTERS		2ND & 3RD TRIMESTERS	
	Z-Value	P-Value	Z-Value	P-Value	Z-Value	P-Value
Amp of R wave (mm)	0.21	0.833	0.86	0.389	0.94	0.347
Amp of S wave (mm)	0.68	0.496	0.68	0.968	0.65	0.733

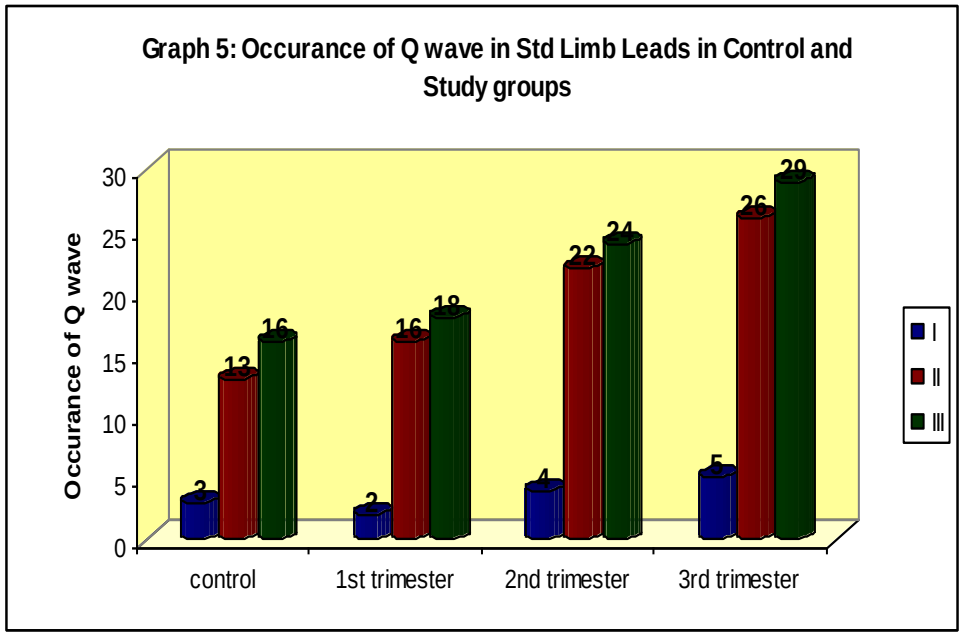
p>0.05: Not Significant, *p: <0.05: Significant, ** p: <0.01: Highly significant, *** p: <0.001: Very highly significant.

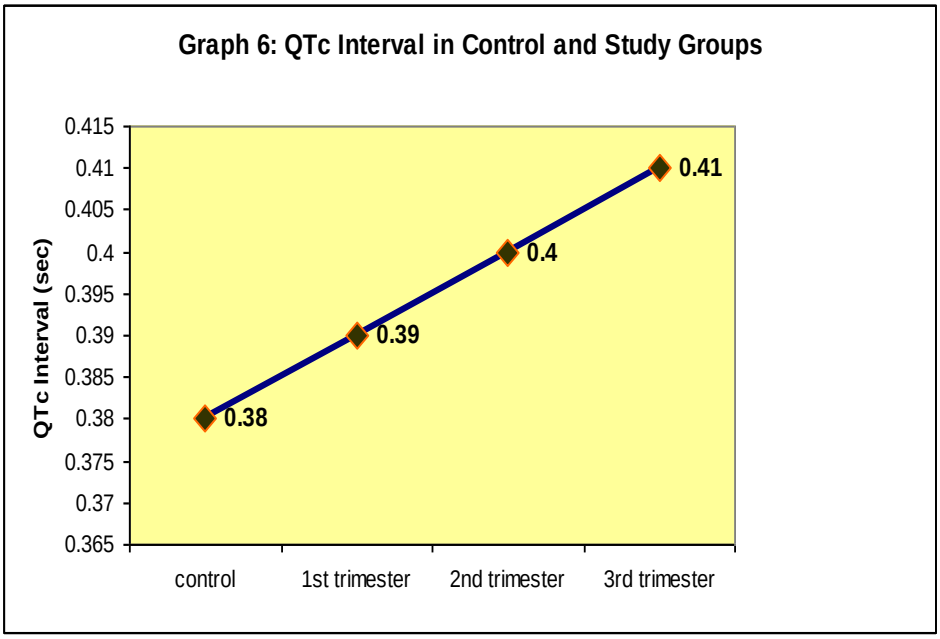

Graph 1: Distribution of subjects in Study and Control Groups.
50
50
50
50
control
1st trimester
2nd trimester
3rd trimester

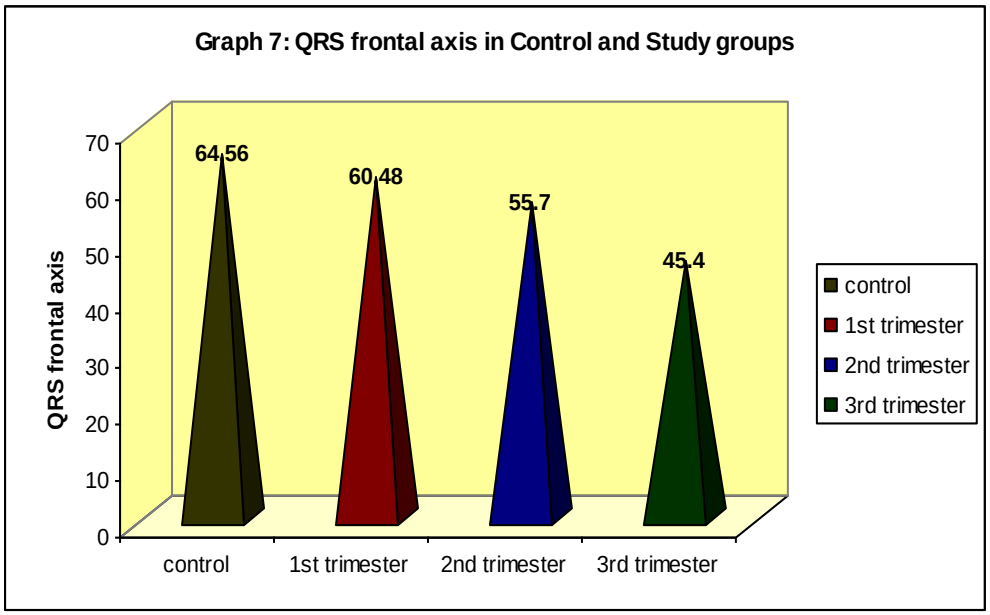

Graph 2: SBP and DBP in Control and Study Groups.
140
120
100
80
60
40
20
0
BP mmHg
control
1st trimester
2nd trimester
3rd trimester
SBP
DBP

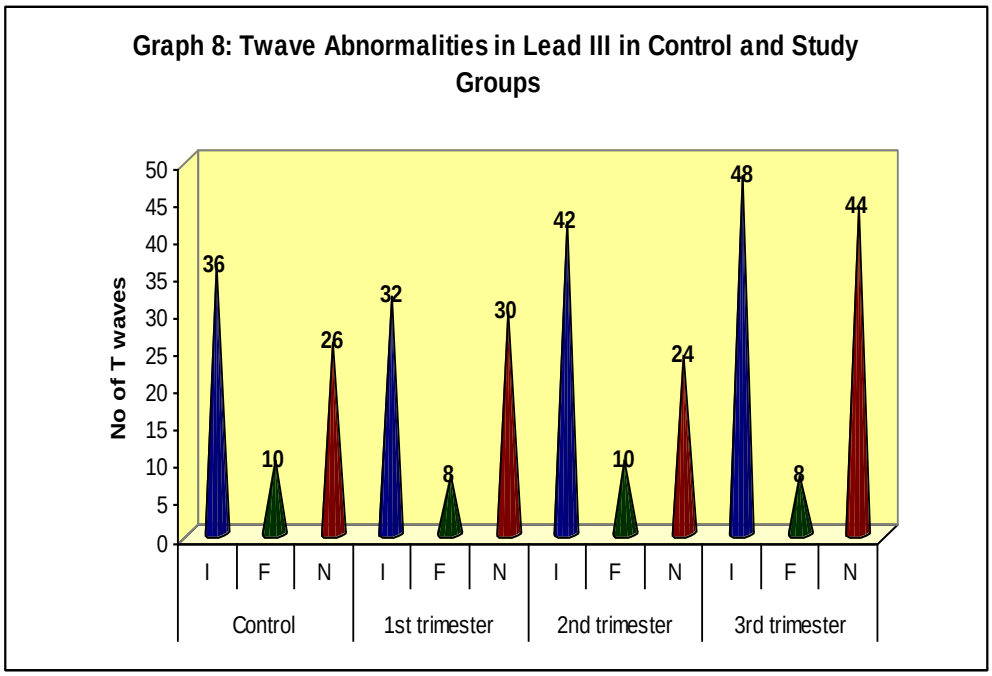

Graph 3: Heart rate in Control and Study Groups.
Heart rate (bpm)
120
100
80
60
40
20
0
control
1st trimester
2nd trimester
3rd trimester
Heart Rate


Graph 4: PR Interval in Control and Study groups
PR Interval (sec)
0.15
0.145
0.14
0.135
0.13
0.125
0.12
0.15
0.14
0.14
0.13
control
1st trimester
2nd trimester
3rd trimester


Graph 5: Occurance of Q wave in Std Limb Leads in Control and Study groups
Occurance of Q wave
30
25
20
15
10
5
0
3
13
16
2
16
18
4
22
24
5
26
29
control
1st trimester
2nd trimester
3rd trimester
I
II
III


Graph 6: QTc Interval in Control and Study Groups
QTc Interval (sec)
0.415
0.41
0.405
0.4
0.395
0.39
0.385
0.38
0.375
0.37
0.365
0.38
0.39
0.4
0.41
control
1st trimester
2nd trimester
3rd trimester


Graph 7: QRS frontal axis in Control and Study groups
QRS frontal axis
70
60
50
40
30
20
10
0
64.56
60.48
55.7
45.4
control
1st trimester
2nd trimester
3rd trimester
control
1st trimester
2nd trimester
3rd trimester


Graph 8: Twave Abnormalities in Lead III in Control and Study Groups
No of T waves
50
45
40
35
30
25
20
15
10
5
0
36
10
26
32
8
30
42
10
24
48
8
44
I
F
N
I
F
N
I
F
N
I
F
N
Control
1st trimester
2nd trimester
3rd trimester

DISCUSSION

Pregnancy is a normal physiological process. It induces widespread circulatory adaptations in the mothers. The pregnancy induced changes in the cardiovascular system develop primarily to meet the increased metabolic demands of mother & fetus.

Both structural and functional changes are known to occur in the heart and vessels due to pregnancy. Ventricular dimensions, heart rate, cardiac output, vascular compliance and capacitance will increase whereas peripheral resistance and blood pressure decrease during pregnancy. Many of these changes are induced by gestational hormonal milieu which influences vessel structure, basal tone and reactivity via receptors for chorionic gonadotropin, estradiol and progesterone located in vascular endothelium and smooth muscle.[30]

Despite the increased work load on the heart during gestation, the healthy pregnant women have no impairment of cardiac reserve. An understanding of these changes and the mechanism involved would be helpful in deciding on the optimal management of pregnant women with preexisting cardiovascular diseases as well as potentially useful in the prevention of gestational complications associated with a inadequate maternal hemodynamic adaptation.

Electrocardiography is one of basic tools in the investigation of cardiovascular diseases. The electrocardiogram during normal pregnancy may show wide variation from the normal accepted. These variations may be due to the changed spatial arrangement of the chest organs as well as changed electrical properties of the myocardium.These changes are inturn due to sympathetic and hormonal modulation of cardiac electrical activity during pregnancy. [47]

A cross-sectional study was carried in 150 healthy pregnant women and 50 healthy non pregnant women in the age range of 18-35 years of BLDEA's Shri B M Patil Medical College, Hospital and Research Centre, Bijapur. The subjects were distributed in two groups, i.e control group and the study group. The study group was comprising of subjects in 1^{st} , 2^{nd} & 3^{rd} trimesters of pregnancy.

We have recorded various physical, physiological & ECG parameters in control and study groups.

Physical Parameters

In our study, there was no significant change in height of the subjects between study and control groups.

There was a significant change in the weight of the subjects in the 3^{rd} trimester of pregnancy when compared to the control group.

There was a also significant change in the BMI of the subjects in the 3^{rd} trimester of pregnancy when compared to the control group.

There was no significant change in the BSA of the subjects in the study and the control groups.

Physiological Parameters

Pulse Rate

There was a significant increase in the pulse rate of the subjects in the 1^{st}, 2^{nd} and 3^{rd} trimesters of pregnancy when compared to the control group.

Systolic Blood Pressure

Our study revealed a significant decrease in SBP in the 2nd trimester of pregnancy compared to control group and also when compared to 1st and 3rd trimesters of pregnancy.

DBP was shown to be significantly decreased in the 2nd trimester of pregnancy compared to control group and also when compared to 1st and 3rd trimesters of pregnancy. In the normal course of pregnancy, the blood pressure during the first few months is similar to that of pre-pregnant women. During the middle 3 months of gestation, the blood pressure tends to fall by an average of 3-5mm Hg. Sometimes, the drop may be up to 20-30 mm Hg[10]

The blood pressure both SBP and DBP tend to fall in early pregnancy reach nadir in the second trimester of pregnancy and return towards pre-pregnant level at term.Fall in blood pressure is due to fall in systemic vascular resistance. The overall decrease in vascular tone in response to a yet unknown endocrine stimulus represents the very first adaptive change in cardiovascular system giving rise to both an increased vascular capacity and a decreased filling state. In early pregnancy, an overall decrease in vascular tone leading to a systemic vasodilatation and rise in arterial compliance. The arterial blood pressure and vascular resistance tend to normalize during the 3rd trimester of pregnancy.[49]

Our study is in agreement with the findings of studies conducted by Duvekot JJ et al[49],Schier RW, Briner VA[50],Voss A et al[51], Hytten et al[10], Clap IF & Capeless E[30],Ekholm EKM et al[52], Thompson CA & Tarto DL[53], Blake MJ et al[54], Oakely CM[55].

ELECTROCARDIOGRAPHIC PARAMETERS:

Heart Rate:

In our study there was a statistically significant increase in the heart rate in 1st, 2nd and 3rd 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 5th week. It increased after 8th 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.[49]The increase in the heart rate is linked to autonomic nervous system changes that produce alterations in cardiac autonomic modulation. 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[50]. The increase in heart rate was due to a decrease in vagal baroreflex as well as a decrease in parasympathetic tone.[51] The increase in heart rate mainly during third trimester of pregnancy compensates for the fall in the stroke volume resulting from caval compression.[54]

The observations made in our study are in agreement with the findings of studies conducted by Duvekot JJ et al[49], Schier RW & Briner VA[50], Voss A et al[51], Hytten et al[10], Clap IF & Capeless E[30], EKholm EKM et al.[52]

P wave:

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:

PR interval was shown to be statistically significantly decreased in 1st, 2nd and 3rd trimesters of pregnancy as compared to control group. There was also a statistically significant decrease in 2nd & 3rd trimesters of pregnancy compared to 1st trimester of pregnancy and in 3rd trimester of pregnancy when compared to 2nd 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.[55]

Similar report was made by Joseph E Carruth et al. In their study, they found that mean PR interval was shorter at 3rd trimester when compared to 1st and 2nd trimesters of normal pregnancy & it was statistically significant.[32]

Q wave:

In the present study, there was a statistically significant increase in occurrence of Q wave in the 2nd and 3rd trimesters when compared to 1st 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.[36] The frequent occurring of Q wave during pregnancy when compared to normal non pregnant women may be to due altered position of the heart.[48]

Similar findings were reported by Misra J et al[25], Veille JC et al[36], Carruth JE et al [32] & Singh AD[48], in their studies.

QRS Complex

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.

ST segment:

In the present study, ST segment depression was noticed in 4 subjects in 2nd trimester and 6 subjects in the 3rd trimester of pregnancy. There was no change in ST segment in the subjects of control group and 1st 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.[56]

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. Anxiety might be accompanied by an increase in circulating humoral agents which would directly affect myocardial electrical activity. [57]

Our findings are in accordance with the observations made by Boyle DM et al[13], Veille JC et al[36], Oram S et al[56], Singh AD et al[48] in their study.

QT Interval:

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:

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^{st}, 2^{nd} and 3^{rd} trimesters of pregnancy when compared to control group. There was also a statistically significant increase in QTc interval in 2^{nd} and 3^{rd} trimesters when compared to 1^{st} trimester of pregnancy and also in the 3^{rd} when compared to 2^{nd} 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.[57]

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. [47]

Similar reports were given by Lechmanova et al[47], Ozmen N et al[2], Carruth JE et al[32], Oram S et al[56] in their studies

T wave:

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.

In the present study, T wave abnormalities like flat and inverted T waves in Lead III were more frequently observed in pregnant women when compared to the non pregnant women.

During pregnancy, there is an increase in blood volume ,which inturn results in a temporary increase in workload on heart. Eventually temporary ischemia develops which is represented by T wave inversion.[25] 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.[58]

Similar findings were observed by Misra J et al[25], Singh R et al[58], Oram S et al[56] & Veille JC et al[36] in their studies.

QRS Frontal axis:

In the present study, QRS axis showed a statistically significant decrease in the 1^{st}, 2^{nd} and 3^{rd} trimesters of pregnancy when compared to non pregnant women. There was also a statistically significant decrease in the 3^{rd} trimester when compared to 1^{st} and 2^{nd} trimester of pregnancy.

With increase in gestational age, position of heart changes from vertical to intermediate indicating that heart shifted to left with increase in gestational period.

The change in the electrical axis may be due to raise in the diaphragm during pregnancy.[48] 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[59] 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. [25]

Similar findings were also reported from Singh AD et al[48], Misra J et al[25], Lechmanova M et al[29] & Carruth JE et al[32] in their studies.

Amplitude of R wave:

R wave is produced by depolarization of interventricular septum and apices of ventricles. In the present study, Amplitude of R wave showed no statistical significance among the study and the control groups or between subgroups within study group.

Amplitude of S wave:

Amplitude of S wave showed no statistical significance among the study and the control groups or between subgroups within study group.

CONCLUSION

We conducted a cross-sectional study to evaluate the different patterns of ECG in 1st, 2nd and 3rd trimesters of pregnancy in "BLDEU'S Shri B M Patil Medical College,Hospital and Research centre, Bijapur. We concluded from our study that:

1. There was a statistically significant increase in the pulse rate in 1st, 2nd and 3rd trimesters of pregnancy when compared to non pregnant women. A similar increase was observed in 2nd and 3rd trimesters when compared to 1st trimester of pregnancy and an increase in 3rd trimester when compared to 2nd trimester of pregnancy.
2. There was a statistically significant decrease in SBP in 1st and 2nd trimesters of pregnancy when compared to non pregnant women. A similar decrease was observed in 2nd trimester when compared to 1st and 3rd trimesters of pregnancy.
3. There was a statistically significant decrease in DBP in 2nd trimester of pregnancy when compared to non pregnant women. A similar decrease was observed in 2nd trimester when compared to 1st and 3rd trimesters of pregnancy.
4. There was statistically significant decrease in MAP in 2nd trimester of pregnancy when compared to non pregnant women. A similar decrease was observed in 2nd trimester when compared to 1st and 3rd trimester of pregnancy.
5. Heart rate showed a statistically significant increase in 1st, 2nd and 3rd trimesters of pregnancy when compared to non pregnant women. A similar increase was observed in 2nd and 3rd trimesters when compared to 1st trimester of pregnancy and in 3rd trimester when compared to 2nd trimester of pregnancy.

6. There was a statistically significant decrease in PR interval in all the trimesters of pregnancy when compared to control group. A similar decrease was observed in 2nd and 3rd trimesters when compared to 1st trimester of pregnancy and in 3rd trimester when compared to 2nd trimester of pregnancy.
7. Occurrence of Q wave in leads II and III showed a statistically significant increase in 2nd and 3rd trimesters of pregnancy when compared to 1st trimester of pregnancy and control group.
8. ST segment depression was noticed in 4 subjects in 2nd trimester and 6 in 3rd trimester of pregnancy. There was no ST segment depression in 1st trimester of pregnancy and in the control group. There was no statistically significant difference between the control and the study groups or between subgroups within study group.
9. QTc interval showed a statistically significant increase in pregnant women in 1st, 2nd and 3rd trimesters of pregnancy when compared to control group. A similar increase was observed in 2nd and 3rd trimesters of pregnancy when compared to 1st trimester and also in 3rd trimester when compared to 2nd trimester of pregnancy.
10. T wave abnormalities in lead III showed a statistically significant increase in both 2nd and 3rd trimesters of pregnancy when compared to non pregnant women.
11. QRS frontal axis showed a statistically significant decrease in 1st, 2nd and 3rd trimesters of pregnancy when compared to non pregnant women. A similar decrease was observed in QRS frontal axis in 2nd & 3rd trimesters of pregnancy when compared 1st trimester of pregnancy and in 3rd trimester when compared to 2nd trimester of pregnancy.

SUMMARY

The study entitled "A study of ECG patterns in 1st, 2nd and 3rd trimesters of pregnancy" was conducted in the Department of Physiology, BLDEU's Shri B M Patil Medical College,Hospital and Research centre, Bijapur during 2009-2010. The aim of the study was to know electrocardiographic changes in pregnant women in 1st, 2nd and 3rd trimesters of pregnancy and in age matched nonpregnant women and to compare the results between and within them.

The cross-sectional study was carried in 150 healthy pregnant women. The study group was in turn divided into 3 subgroups each with 50 women in first, second and third trimesters of pregnancy and 50 healthy non pregnant women in the age group of 18-35 years were selected randomly who were attending the OPD of OBGy of Shri B.M. Patil Medical College, Hospital and Research centre, Bijapur.

Statistically significant observations were made such as an increase in the pulse rate in 1st, 2nd and 3rd trimesters of pregnancy, decrease in SBP in 1st and 2nd trimesters of pregnancy, a decrease in DBP in 2nd trimester of pregnancy, a decrease in MAP in 2nd trimester of pregnancy, Heart rate was increased in 1st, 2nd and 3rd 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 2nd and 3rd trimesters of pregnancy, ST segment depression was noticed in 4 subjects in 2nd trimester and 6 in 3rd trimester of pregnancy, QTc interval showed an increase in pregnant women in 1st, 2nd and 3rd trimesters of pregnancy , T wave abnormalities in lead III showed an increase in both 2nd and 3rd trimesters of pregnancy and QRS frontal axis was decreased in 1st, 2nd and 3rd trimesters of pregnancy.

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ANNEXURE – 1

B.L.D.E.A'S SHRI B.M.PATIL MEDICAL COLLEGE , BIJAPUR.
INSTITUTIONAL ETHICAL COMMITTEE

Dr. Vijay Ganjoo
Chairperson, I.E.C.
B.L.D.E.A'S Shri B.M.Patil Medical college
Bijapur-586103

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 22-09-2008

at 03-15 pm **to scrutinize the Synopsis / Research projects of post graduate student / undergraduate student / Faculty members of this college from ethical clearance point of view. After scrutiny the following original / corrected & revised version Synopsis of the Thesis/ Research project has been accorded Ethical Clearance.**

Title A study of ECG patterns in 1st 2nd & 3rd Trimesters of Pregnancy

Name of P.G / U.G student / Faculty member Dr. Nandini B.N.

Name of Guide Dr. Manjunatha. Astekar. ASSOC. Prof of Physiology

Dr. Vijay Ganjoo
Chairperson
Institutional Ethical Committee

Date:

Following documents were placed before E.C. for scrutinization:
1)Copy of Synopsis / Research project
2) Copy of informed consent form
3) Any other relevant document/s

ANNEXURE – 2

B. L. D. E UNIVERSITY, SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, BIJAPUR
RESEARCH INFORMED CONSENT FORM

Title of the Project:

"A STUDY OF ECG PATTERNS IN 1ST, 2ND AND 3RD TRIMESTERS OF PREGNANCY"

Principal investigator/ P.G.Guide's name: **DR.MANJUNATHA.AITHALA MD**

ASSOCIATE PROFESSOR,

DEPARTMENT PHYSIOLOGY.

1: PURPOSE OF RESEARCH: I have been informed that this study will test influence of age on cardiovascular autonomic functions. This study will be useful academically as well as for clinically to interpret ECG findings in different trimesters of pregnancy and control group.

2: PROCEDURE: I understand that, the procedure of the study will involve recording of various physiological physical parameters. The procedure will not interfere with any of my physiological parameters and they are noninvasive.

3: RISK AND DISCOMFORTS: I understand determination of ECG changes will not cause any discomfort to me and do not involve any risk to my health.

4: BENEFITS: I understand that my participation in the study may not have a direct benefit to me but this may have a potential beneficial effect in the field of electrocardiography changes in future.

5: CONFIDENTIALITY: I understand that medical information produced by this study will become part of institutional records and will be subject to the confidentiality and

privacy regulation of the said institute. Information of a sensitive personal nature will not be a part of medical record, but will be stored in investigators research file and identified only by a code number. The code key connecting name two numbers will be kept in a separate secured location.

If the data are used for publication in the medical literature and for teaching purposes no names will be used and other identities such as photographs, audio and video tapes will be used only with my special written permission. I understand I may see the photographs and the video tapes and have the audio tapes before giving this permission.

6: REQUEST FOR MORE INFORMATION: I understand that I may ask more questions about the study at any time. Concerned researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study which might influence my continued participation. If during the study or later, I wish to discuss my participation in all concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful re-reading.

7: REFUSAL OR WITHDRAWAL OF PARTICIPATION: I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that researcher may terminate my participation in this study at any time after she/he has explained the reasons for doing so and had helped arrange for my continued care by my physician or physical therapist if this is appropriate.

8: INJURY STATEMENT: I understand that in unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, then medical treatment will be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____ (Patient/Relevant guardian)

the purpose of the research, procedures required and the possible risk and benefits to the best of my ability.

Investigator/ PG (Guide) Date

I confirm that _____ (Name of the P.G. Guide /Chief researcher) has explained to me the purpose of research, the study procedure that I will undergo, and the possible risk and discomforts as well as benefits that I may experience. Alternative to my participation in the study have also been to give my consent from. Therefore I agree to give consent to participate as a subject and this research project.

Participant / Guardian Date:

Witness to signature Date:

Modified from Portney L.G, Watkins M.P., in Foundation of Clinical Research, Second Edition, New Jersey, Prentice Hall Health 2000.

ANNEXURE – 3

PROFORMA

BLDE UNIVERSITY
SHRI B.M.PATIL MEDICAL COLLEGE, BIJAPUR
DEPARTMENT OF PHYSIOLOGY

S.NO. DATE:

NAME: OCCUPATION:

AGE:

RELIGION:

ADDRESS:

PRESENTING COMPLAINTS:

OBSTETRIC HISTORY:

PAST HISTORY:

PERSONAL HISTORY:

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATION:

ANTHROPOMETRY:

HEIGHT: WEIGHT: BSA: BMI:

VITAL SIGNS:

SYSTEMIC EXAMINATION:

RESPIRATORY SYSTEM

CARDIOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM

PER ABDOMEN:

HEIGHT OF UTERUS: LIE:

FETAL HEART RATE: FETAL MOVEMENTS:

ECG REPORT:

1. HEART RATE:
2. RHYTHM
3. P WAVE
4. PR INTERVAL
5. Q WAVE
6. ST SEGMENT
7. QRS COMPLEX
8. QT INTERVAL
9. QTc INTERVAL
10. QRS FRONTAL AXIS
11. T WAVE
12. AMPLITUDE OF R WAVE:
13. AMPLITUDE OF S WAVE

Signature of Guide Signature of PG student Signature of HOD

ANNEXURE 4a: MASTER CHART- CONTROL GROUP

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP(mv)	DUR(sec)
1	GANGAMMA	24	153	46	19.65	1.44	74	110	68	42	82.33	76	S.R	1	0.08
2	REKHA BAI	32	154	58	24.47	1.56	80	120	82	38	98.33	80	S.R	1	0.08
3	SUSHILA	26	147	50	23.14	1.4	75	107	58	49	71.33	72	S.R	1	0.08
4	SOMESHWARI	28	156	51	20.98	1.48	74	110	70	40	85.33	74	S.R	1	0.08
5	SHEELA	27	162	53	20.22	1.54	76	118	72	46	87.33	76	S.R	1	0.06
6	PREMA	20	150	48	20	1.44	80	120	74	46	89.33	80	S.R	1	0.08
7	SUMANGALA	30	149	53	23.87	1.46	74	116	70	46	86.67	74	S.R	1	0.08
8	SHARADA	34	150	56	22.2	1.58	72	122	72	50	88	72	S.R	1	0.08
9	GIRIJA	32	161	53	20.46	1.52	71	124	76	48	89.33	70	S.R	1	0.08
10	SAROJA	30	157	54	21.95	1.52	83	120	80	40	96	82	S.R	1	0.08
11	SAVITHA	31	151	52	25.43	1.52	76	116	68	48	80.67	74	S.R	1.5	0.08
12	JASMINE	28	148	53	24.2	1.46	68	112	74	38	88	68	S.R	1	0.08
13	RUDRI BAI	28	156	50	20.57	1.48	76	118	76	42	92	78	S.R	1	0.08
14	NAGAMMA	25	163	54	20.37	1.58	75	122	74	48	88	74	S.R	1	0.06
15	SOUMYA	32	151	52	22.81	1.5	80	120	78	42	94.67	78	S.R	1	0.08
16	SUDHA	24	158	51	20.48	1.5	73	124	74	50	89.33	76	S.R	1	0.08
17	SUMATHI	32	164	56	21.5	1.6	80	118	72	46	88.67	80	S.R	1	0.08
18	RUKMINI	29	153	49	20.94	1.44	84	120	70	50	84	82	S.R	1	0.08
19	HEMALI	27	156	57	22.61	1.58	73	122	80	42	98.67	72	S.R	1	0.08
20	SMITHA	29	162	57	21.75	1.6	70	124	68	56	82.67	70	S.R	1	0.06
21	SARITHA	26	159	51	20.23	1.5	76	118	74	44	88.67	74	S.R	1	0.08
22	SAVITRI	25	150	54	24	1.56	74	116	72	44	89.33	72	S.R	1	0.08
23	VANI	24	164	53	19.77	1.56	81	122	70	52	85.33	80	S.R	0.5	0.08
24	GOURAMMA	25	147	53	24.53	1.44	66	124	78	46	95.33	68	S.R	1	0.1
25	SUMATHI	22	148	51	23.28	1.44	80	120	68	52	82	82	S.R	1	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
1	0.14	absent	absent	absent	0.08	IE		0.32	0.395	82	13	9	3	0.2	N	N	N
2	0.16	absent	absent	present	0.08	IE		0.36	0.385	68	14	12	3	0.2	N	N	N
3	0.12	absent	absent	absent	0.08	IE		0.32	0.358	48	11	10	2	0.22	N	N	I
4	0.16	absent	absent	absent	0.08	IE		0.36	0.395	73	15	10	3	0.26	N	N	N
5	0.16	absent	present	present	0.08	IE		0.36	0.388	64	12	10	1	0.2	N	N	I
6	0.16	absent	absent	absent	0.08	IE		0.32	0.381	60	14	11	4	0.28	N	N	I
7	0.16	absent	absent	absent	0.08	IE		0.32	0.384	56	12	11	4	0.3	N	N	I
8	0.16	absent	absent	absent	0.06	IE		0.36	0.377	60	15	10	4	0.16	N	N	N
9	0.16	absent	absent	absent	0.08	IE		0.36	0.388	68	14	9	2	0.16	N	N	F
10	0.16	absent	present	present	0.08	IE		0.36	0.395	72	16	10	2	0.2	N	N	I
11	0.16	absent	absent	absent	0.08	IE		0.36	0.362	60	15	10	3	0.2	N	N	N
12	0.16	present	present	present	0.06	IE		0.36	0.388	72	13	9	3	0.2	N	N	N
13	0.14	absent	present	present	0.08	IE		0.36	0.384	59	13	10	3	0.16	N	N	I
14	0.16	absent	absent	absent	0.08	IE		0.32	0.391	66	12	11	2	0.2	N	N	I
15	0.16	absent	absent	absent	0.08	IE		0.36	0.388	60	14	12	1	0.2	N	N	I
16	0.12	absent	absent	absent	0.08	IE		0.34	0.393	64	9	10	4	0.16	N	N	N
17	0.16	absent	absent	absent	0.08	IE		0.36	0.398	70	14	13	4	0.24	N	N	N
18	0.16	absent	present	present	0.06	IE		0.36	0.409	59	16	14	1	0.12	N	N	I
19	0.16	absent	present	present	0.08	IE		0.32	0.377	68	13	10	1	0.2	N	N	F
20	0.16	absent	absent	absent	0.08	IE		0.36	0.395	56	12	11	3	0.3	N	N	N
21	0.16	absent	absent	absent	0.08	IE		0.36	0.39	60	14	11	4	0.2	N	N	N
22	0.14	absent	absent	absent	0.08	IE		0.36	0.388	64	12	10	1	0.28	N	N	N
23	0.16	absent	absent	absent	0.08	IE		0.36	0.388	73	15	10	3	0.12	N	N	N
24	0.16	absent	absent	present	0.08	IE		0.36	0.381	48	11	10	3	0.16	N	N	I
25	0.12	absent	absent	absent	0.08	IE		0.36	0.381	68	14	12	5	0.16	N	N	N

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP (mv)	DUR (sec)
26	SABREENA	23	150	48	21.33	1.42	76	122	80	42	92.67	74	S.R	1	0.08
27	SUHASINI	26	151	60	26.31	1.56	72	120	82	38	96.67	72	S.R	1	0.08
28	JAYASHREE	22	157	55	22.31	1.54	80	118	74	44	86.67	78	S.R	1	0.08
29	ALKA	28	149	52	23.42	1.44	78	116	78	38	92	76	S.R	1	0.08
30	MAMTHA	27	142	50	24.8	1.38	76	118	76	42	93.33	74	S.R	1	0.06
31	SANGEETA	23	152	49	21.21	1.44	83	122	70	52	88	84	S.R	1	0.08
32	SWAPNA	26	154	47	19.82	1.42	80	126	72	54	86.67	80	S.R	1	0.08
33	MEENAKSH	21	149	53	23.87	1.48	72	122	78	44	92.67	74	S.R	0.5	0.08
34	REKHA BAI	25	145	55	26.16	1.44	70	120	76	44	88.67	72	S.R	1	0.08
35	POORNIMA	24	148	58	26.48	1.5	80	118	80	38	95.33	82	S.R	1	0.06
36	PRABHA	23	152	51	22.07	1.48	81	116	70	46	85.33	80	S.R	1	0.08
37	MANJULA	22	145	56	26.63	1.46	76	120	74	46	89.33	78	S.R	1	0.08
38	LAKSHMI	26	151	54	24	1.48	75	122	76	46	92.67	72	S.R	1	0.08
39	SHANTI	28	151	48	21.05	1.4	80	110	78	32	88.67	76	S.R	1	0.08
40	MANGALA	25	155	55	22.89	1.52	80	112	80	32	94.67	78	S.R	1	0.08
41	RATHNA	24	147	60	27.77	1.54	78	114	70	44	86.67	80	S.R	1	0.06
42	ARUNA	21	153	50	21.36	1.46	76	124	74	50	90.67	74	S.R	1.5	0.08
43	PREMA	22	145	45	21.4	1.44	74	122	72	50	85.33	76	S.R	1	0.08
44	GOURI	27	142	47	23.31	1.36	80	118	78	40	94.67	78	S.R	1	0.08
45	HARSHITA	30	154	60	25.3	1.58	76	120	70	50	83.33	74	S.R	1	0.08
46	PRIYA	22	145	50	23.78	1.4	82	122	82	40	96.67	78	S.R	1	0.1
47	SUMA	23	152	52	22.51	1.48	74	124	80	44	93.33	72	S.R	1.5	0.08
48	KAMALA	27	150	54	24	1.5	70	118	78	40	92.67	68	S.R	1	0.08
49	ASHA	22	157	47	19.07	1.38	76	116	72	44	72	74	S.R	1	0.08
50	SOUMYA	23	151	48	21.05	1.42	80	110	78	32	88.67	76	S.R	0.5	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
26	0.16	absent	absent	absent	0.08	IE		0.32	0.395	82	13	9	2	0.2	N	N	I
27	0.16	present	present	present	0.08	IE		0.36	0.392	62	6	7	3	0.16	N	N	I
28	0.16	absent	absent	absent	0.08	IE		0.36	0.388	58	12	5	3	0.16	N	N	I
29	0.12	absent	absent	absent	0.06	IE		0.36	0.402	60	14	9	3	0.16	N	N	F
30	0.16	absent	absent	absent	0.08	IE		0.32	0.395	74	13	9	3	0.16	N	N	N
31	0.16	absent	present	present	0.08	IE		0.36	0.388	70	15	10	2	0.16	N	N	N
32	0.16	absent	present	present	0.08	IE		0.36	0.381	80	9	10	4	0.2	N	N	N
33	0.12	absent	absent	absent	0.08	IE		0.36	0.389	65	14	10	3	0.16	N	N	I
34	0.16	absent	absent	absent	0.08	IE		0.36	0.382	60	13	10	2	0.16	N	N	I
35	0.14	absent	absent	absent	0.08	IE		0.36	0.396	69	12	11	5	0.16	N	N	F
36	0.16	absent	present	present	0.08	IE		0.36	0.374	59	14	11	3	0.16	N	N	N
37	0.16	absent	absent	absent	0.08	IE		0.36	0.384	60	7	5	2	0.16	N	N	I
38	0.16	absent	absent	absent	0.08	IE		0.34	0.356	66	8	11	3	0.2	N	N	N
39	0.16	absent	absent	present	0.08	IE		0.36	0.391	70	19	12	2	0.16	N	N	N
40	0.12	absent	absent	absent	0.08	IE		0.36	0.391	59	12	14	2	0.16	N	N	N
41	0.16	absent	absent	absent	0.08	IE		0.36	0.402	72	15	9	3	0.16	N	N	N
42	0.16	present	present	present	0.08	IE		0.32	0.392	69	7	5	3	0.2	N	N	N
43	0.14	absent	absent	absent	0.1	IE		0.36	0.355	72	12	16	3	0.2	N	N	N
44	0.16	absent	absent	absent	0.08	IE		0.36	0.391	60	9	9	1	0.16	N	N	I
45	0.12	absent	absent	absent	0.08	IE		0.36	0.381	62	6	7	3	0.2	N	N	N
46	0.14	absent	present	present	0.06	IE		0.36	0.379	60	14	9	3	0.2	N	N	I
47	0.16	absent	absent	absent	0.08	IE		0.36	0.358	64	14	10	3	0.2	N	N	I
48	0.16	absent	absent	absent	0.08	IE		0.36	0.343	70	19	12	1	0.16	N	N	N
49	0.16	absent	present	present	0.08	IE		0.32	0.387	49	16	10	3	0.16	N	N	F
50	0.16	absent	absent	absent	0.08	IE		0.36	0.356	58	10	11	2	0.12	N	N	N

ANNEXURE 4b: MASTER CHART-1ST TRIMESTER OF PREGNANCY

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP(mv)	DUR(sec)
1	NAZIMA	25	145	43	20.47	1.32	74	120	78	42	94.67		S.R	1	0.08
2	SHANTAMMA	33	154	58	18.14	1.56	80	118	76	42	92	80	S.R	1	0.08
3	SUREKHA	32	156	60	25.31	1.58	75	122	74	48	88	75	S.R	1	0.08
4	NAZIMA	27	163	58	21.88	1.62	74	124	68	56	82.67	74	S.R	1	0.08
5	RUDRAMMA	30	154	50	21.09	1.46	76	118	74	44	88.67	76	S.R	1	0.08
6	REKHA	23	158	44	17.6	1.4	80	120	74	46	86.67	80	S.R	1	0.06
7	PARVATHI	24	148	52	23.4	1.44	74	120	82	38	96.67	74	S.R	1	0.08
8	BANU	24	157	45	18.29	1.42	72	116	78	38	92	72	S.R	1	0.08
9	RUKMINI	28	149	56	25.22	1.5	78	122	80	42	98.67	78	S.R	1	0.08
10	SOUNDARYA	26	160	54	21.09	1.54	83	118	72	46	87.33	83	S.R	1	0.08
11	SRILEKHA	27	157	51	20.73	1.48	76	110	70	40	85.33	76	S.R	1.5	0.06
12	LALITHA BAI	19	158	53	21.28	1.52	82	126	74	52	85.33	82	S.R	1	0.08
13	PRATHIMA	22	153	60	25.64	1.58	76	118	76	42	92	76	S.R	1	0.08
14	RADHA	28	149	54	20.27	1.48	94	122	70	52	85.33	94	S.R	1	0.08
15	SABREEN TAJ	23	161	58	22.39	1.6	80	120	82	38	96.67	80	S.R	1	0.08
16	TEJASWINI	28	148	53	24.2	1.46	73	112	68	44	82.6	73	S.R	1	0.08
17	GIRIJA	30	154	60	25.31	1.58	80	110	62	48	78	80	S.R	1	0.06
18	BANUMMA	25	154	45	19.23	1.4	84	116	78	38	92	84	S.R	1	0.08
19	MAHANANDA	28	156	52	21.23	1.48	86	124	80	44	93.33	86	S.R	1	0.08
20	MAHADEVI	22	155	46	19.6	1.4	82	122	70	52	87.33	82	S.R	1	0.08
21	SHARADA	23	156	48	19.75	1.44	76	110	80	30	90	76	S.R	1	0.08
22	ASHA	22	153	42	17.9	1.36	74	100	60	40	73.3	74	S.R	1	0.1
23	GAYATRI	26	150	40	17.7	1.38	81	126	72	54	86.67	81	S.R	0.5	0.08
24	VEENA	24	154	59	23.4	1.56	66	120	68	52	85.33	66	S.R	1	0.08
25	SUSHILA	21	150	46	19.4	1.4	80	110	80	30	90	80	S.R	1	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
1	0.12	absent	absent	absent	0.08	IE		0.32	0.395	68	14	9	3	0.2	N	N	N
2	0.16	absent	absent	present	0.08	IE		0.36	0.385	72	16	10	3	0.2	N	N	I
3	0.12	absent	absent	absent	0.08	IE		0.32	0.358	60	15	10	2	0.22	N	N	N
4	0.14	absent	absent	absent	0.08	IE		0.36	0.395	72	13	9	3	0.26	N	N	N
5	0.16	absent	present	present	0.06	IE		0.36	0.388	59	13	10	1	0.2	N	N	N
6	0.12	absent	absent	absent	0.08	IE		0.32	0.381	66	12	11	4	0.28	N	N	I
7	0.1	absent	absent	absent	0.08	IE		0.32	0.384	60	14	12	4	0.3	N	N	I
8	0.12	absent	absent	absent	0.08	IE		0.36	0.377	64	9	10	4	0.16	N	N	I
9	0.14	absent	absent	absent	0.08	IE		0.36	0.388	70	14	13	2	0.16	N	N	N
10	0.16	absent	present	present	0.08	IE		0.36	0.395	59	16	14	2	0.2	N	N	N
11	0.12	absent	absent	absent	0.08	IE		0.36	0.362	68	13	10	3	0.2	N	N	I
12	0.12	present	present	present	0.08	D	III,V4,V5	0.36	0.388	56	12	11	3	0.2	N	N	F
13	0.14	absent	present	present	0.08	IE		0.36	0.384	60	14	11	3	0.16	N	N	N
14	0.16	absent	absent	absent	0.08	IE		0.32	0.391	64	12	10	2	0.2	N	N	N
15	0.12	absent	absent	absent	0.08	IE		0.36	0.388	73	15	10	1	0.2	N	N	N
16	0.16	absent	absent	absent	0.06	IE		0.34	0.393	48	11	10	4	0.16	N	N	N
17	0.16	absent	absent	absent	0.08	IE		0.36	0.398	68	14	12	4	0.24	N	N	N
18	0.14	absent	present	present	0.08	IE		0.36	0.409	82	13	9	1	0.12	N	N	N
19	0.12	absent	present	present	0.08	IE		0.32	0.377	62	6	7	1	0.2	N	N	I
20	0.12	absent	absent	absent	0.08	IE		0.36	0.395	58	12	5	3	0.24	N	N	I
21	0.16	absent	absent	absent	0.08	IE		0.36	0.39	60	14	9	4	0.2	N	N	N
22	0.14	absent	present	present	0.06	IE		0.36	0.388	60	15	12	1	0.12	N	N	F
23	0.14	absent	absent	absent	0.08	IE		0.36	0.388	52	13	7	3	0.12	N	N	I
24	0.14	present	present	present	0.08	IE		0.32	0.386	53	9	9	3	0.22	N	N	I
25	0.16	absent	absent	absent	0.06	IE		0.32	0.405	46	17	10	5	0.16	N	N	N

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP (mv)	DUR (sec)
26	ASHA	22	153	44	18.8	1.38	76	114	80	34	91.33	76	S.R	1	0.08
27	RACHANA	24	156	46	18.9	1.42	94	124	74	50	83.33	94	S.R	1.5	0.08
28	POORNIMA	29	162	56	21.3	1.58	76	104	72	32	82.66	76	S.R	1	0.08
29	SRIDEVI	27	154	48	20.25	1.44	92	114	72	52	89.33	92	S.R	1	0.08
30	RUPA	24	162	54	20.6	1.64	86	104	68	36	90	86	S.R	1	0.06
31	SHILPA	23	148	50	22.83	1.42	84	98	60	38	72.6	84	S.R	1	0.08
32	PRATHIBA	26	153	44	18.8	1.38	98	116	70	46	85.33	98	S.R	1	0.1
33	SHOBHA	24	146	53	24.8	1.44	76	124	76	48	92	76	S.R	1	0.06
34	PREETI	20	156	49	20.16	1.44	82	110	80	30	90	82	S.R	1	0.08
35	SUMANGALA	28	152	54	23.4	1.5	84	112	78	34	89.3	84	S.R	0.5	0.08
36	ANITA	24	157	75	30.4	1.76	94	122	82	40	95.3	94	S.R	1	0.06
37	RATHANMMA	26	148	48	21.9	1.4	90	102	72	30	92	90	S.R	1	0.1
38	REKHA	28	144	52	26	1.4	83	108	64	44	98.6	83	S.R	1	0.08
39	PUSHPALATHA	27	156	42	25.53	1.62	84	114	74	40	87.3	84	S.R	1	0.08
40	POORNIMA	28	160	60	23.4	1.62	78	102	78	24	86	78	S.R	1	0.08
41	REKHA	21	146	50	23.4	1.4	82	134	70	64	91.3	82	S.R	2	0.06
42	SUDHA	29	156	48	19.75	1.46	98	108	62	46	77.3	98	S.R	0.5	0.08
43	KALPANA	21	148	54	24.65	1.48	100	112	68	44	92.6	100	S.R	1	0.08
44	JAGADEVI	30	156	45	20.7	1.42	86	124	82	42	96	86	S.R	1	0.08
45	VEENA	20	149	51	22.9	1.44	89	110	70	40	83.3	89	S.R	1	0.08
46	JAYASHREE	19	157	44	17.88	1.4	74	120	70	50	86.6	74	S.R	0.5	0.08
47	SARIKA	25	159	45	17.85	1.42	92	108	68	40	81.3	92	S.R	1	0.08
48	SAVITHA	20	147	54	25	1.48	98	114	80	34	91.3	98	S.R	1	0.04
49	KAVITHA	25	153	67	29.13	1.68	80	118	72	46	87.3	80	S.R	1	0.08
50	BOURAKKA	26	146	40	18.77	1.22	82	120	80	40	93.3	82	S.R	1	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
26	0.16	absent	absent	absent	0.08	IE		0.36	0.407	59	18	12	2	0.16	N	N	N
27	0.16	present	present	present	0.06	IE		0.36	0.405	56	10	11	3	0.2	N	N	I
28	0.12	absent	absent	absent	0.08	IE		0.36	0.397	67	16	11	3	0.2	N	N	N
29	0.16	absent	absent	absent	0.08	IE		0.34	0.412	70	14	11	5	0.2	N	N	N
30	0.16	absent	absent	absent	0.08	IE		0.36	0.409	50	17	10	5	0.22	N	N	N
31	0.16	absent	present	present	0.08	IE		0.36	0.413	63	18	12	3	0.16	N	N	I
32	0.12	absent	present	present	0.08	IE		0.34	0.391	54	15	10	2	0.16	N	N	I
33	0.16	absent	absent	absent	0.06	IE		0.36	0.402	58	16	11	2	0.16	N	N	I
34	0.14	absent	absent	absent	0.08	IE		0.34	0.395	32	12	10	2	0.2	N	N	N
35	0.16	absent	absent	absent	0.08	IE		0.36	0.388	68	11	9	2	0.2	N	N	F
36	0.16	absent	present	present	0.06	IE		0.32	0.405	64	6	7	3	0.12	N	N	N
37	0.16	absent	absent	absent	0.08	IE		0.34	0.412	37	10	11	2	0.2	N	N	N
38	0.16	absent	absent	absent	0.08	IE		0.36	0.388	60	11	10	2	0.16	N	N	N
39	0.12	absent	present	present	0.08	IE		0.36	0.384	26	9	9	1	0.12	N	N	N
40	0.16	absent	absent	present	0.08	IE		0.36	0.379	82	10	11	4	0.2	N	N	N
41	0.16	absent	absent	absent	0.06	IE		0.32	0.409	70	10	11	4	0.12	N	N	N
42	0.14	present	present	present	0.08	IE		0.36	0.418	56	4	4	1	0.16	N	N	I
43	0.16	absent	absent	absent	0.08	IE		0.36	0.42	76	16	11	3	0.2	N	N	I
44	0.12	absent	absent	absent	0.06	IE		0.36	0.408	50	14	10	3	0.16	N	N	N
45	0.14	absent	absent	absent	0.08	IE		0.34	0.401	66	13	7	2	0.2	N	N	N
46	0.16	absent	present	present	0.08	IE		0.32	0.419	72	9	9	3	0.2	N	N	I
47	0.16	absent	absent	absent	0.08	IE		0.34	0.388	50	15	10	3	0.12	N	N	F
48	0.16	absent	absent	absent	0.06	IE		0.36	0.405	54	14	12	3	0.2	N	N	N
49	0.16	absent	present	present	0.08	IE		0.36	0.398	56	4	4	2	0.22	N	N	N
50	0.12	absent	absent	absent	0.06	IE		0.36	0.401	68	11	9	1	0.12	N	N	N

ANNEXURE 4c: MASTER CHART – 2ND TRIMESTER OF PREGNANCY

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP(mv)	DUR(sec)
1	SHANTALA	28	162	58	22.16	1.6	76	108	62	46	77.3	78	S.R	1	0.08
2	SHAMSHAD	33	156	62	25.5	1.62	78	116	72	44	86.6	80	S.R	1	0.08
3	PRATIBHA	20	151	46	20.17	1.38	76	100	60	40	73.3	76	S.R	1.5	0.08
4	UMADEVI	34	155	64	26.6	1.62	92	118	74	44	88.6	94	S.R	1	0.06
5	MANGALA	24	143	53	25.9	1.42	86	116	74	42	88	86	S.R	1	0.08
6	MEENAKSHI	22	146	53	24.88	1.44	82	110	70	40	83.3	84	S.R	1	0.1
7	GEETA	20	147	49	22.6	1.4	98	108	64	44	78.6	100	S.R	1	0.06
8	MEENA	26	146	58	27.2	1.5	76	120	60	60	80	78	S.R	1	0.08
9	KAVITHA	26	159	47	18.65	1.46	82	98	60	42	74	84	S.R	1	0.08
10	PARVATI	28	154	60	25.31	4.58	84	104	68	36	80	84	S.R	1	0.06
11	SUREKHA	22	150	42	18.66	1.42	86	110	66	44	80.67	88	S.R	1	0.1
12	NEELAVVA	28	158	62	24.8	1.62	84	106	70	36	82	84	S.R	0.5	0.08
13	KASTURI	20	150	44	19.5	1.36	80	112	72	40	85.3	78	S.R	1	0.08
14	GOURAMMA	26	152	45	19.48	1.38	83	114	68	46	83.3	84	S.R	1	0.08
15	KASTURI	26	157	55	22.35	1.54	77	116	74	42	88	76	S.R	1	0.06
16	MANJULA	30	148	47	21.46	1.38	81	108	64	44	78.67	80	S.R	2	0.08
17	SUMANGALA	22	153	54	22.64	1.5	98	104	60	44	74.6	100	S.R	1	0.08
18	TRIVENI	24	140	40	20.4	1.24	100	106	62	44	76.6	100	S.R	1	0.08
19	BABY	26	145	50	23.8	1.4	86	110	66	44	80.67	84	S.R	1	0.08
20	NILAMMA	28	164	63	23.5	1.68	89	114	70	44	84.6	88	S.R	1	0.08
21	NETRA	24	158	56	22.48	1.56	74	108	68	40	81.3	72	S.R	1	0.08
22	SUKADEVI	18	152	56	24.24	1.62	92	104	72	32	82.6	94	S.R	1	0.04
23	BHAGYASHREE	23	163	55	20.75	1.58	98	120	60	60	80	100	S.R	1	0.08
24	SUNANDA	19	161	48	18.53	1.56	100	118	70	48	86	100	S.R	1	0.06
25	SUMA	24	154	55	23.5	1.52	96	110	72	42	86	94	S.R	1.5	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
1	0.12	absent	absent	present	0.08	IE		0.32	0.386	49	12	9	3	0.2	N	N	I
2	0.16	absent	present	present	0.08	IE		0.32	0.405	53	15	9	3	0.2	N	N	I
3	0.12	absent	absent	absent	0.08	IE		0.36	0.407	56	6	7	5	0.2	N	N	I
4	0.14	absent	absent	absent	0.08	IE		0.36	0.405	61	14	10	5	0.16	N	N	N
5	0.16	absent	absent	absent	0.06	IE		0.36	0.397	68	11	9	2	0.16	N	N	N
6	0.12	absent	present	present	0.08	IE		0.34	0.412	42	10	13	3	0.24	N	N	N
7	0.1	absent	present	present	0.06	IE		0.36	0.409	50	17	13	3	0.28	N	N	N
8	0.12	absent	present	present	0.08	IE		0.36	0.413	54	12	11	3	0.2	N	N	F
9	0.14	absent	absent	present	0.08	IE		0.34	0.391	60	15	12	2	0.16	N	N	N
10	0.16	absent	absent	absent	0.08	IE		0.36	0.402	52	13	7	2	0.16	N	N	N
11	0.12	absent	absent	absent	0.1	IE		0.34	0.395	53	9	9	3	0.16	N	N	F
12	0.12	absent	absent	absent	0.08	D	III, V4,V5	0.36	0.388	46	17	10	3	0.2	N	N	I
13	0.14	absent	absent	absent	0.08	IE		0.32	0.405	59	18	12	3	0.16	N	N	I
14	0.16	absent	absent	absent	0.06	IE		0.34	0.412	56	10	11	1	0.16	N	N	N
15	0.12	absent	absent	absent	0.06	IE		0.36	0.388	67	16	11	3	0.16	N	N	N
16	0.16	absent	present	present	0.08	IE		0.36	0.384	70	14	11	2	0.16	N	N	N
17	0.16	absent	present	present	0.08	IE		0.36	0.379	50	17	10	3	0.16	N	N	N
18	0.14	absent	present	present	0.08	IE		0.32	0.409	63	18	12	3	0.2	N	N	I
19	0.12	absent	absent	absent	0.08	IE		0.36	0.418	54	15	10	3	0.16	N	N	I
20	0.12	absent	present	present	0.06	IE		0.36	0.42	58	16	11	1	0.16	N	N	I
21	0.16	absent	absent	absent	0.08	IE		0.36	0.408	32	12	10	3	0.16	N	N	I
22	0.14	present	present	present	0.08	D	III, V3,V4,V5	0.34	0.401	68	11	9	2	0.16	N	N	N
23	0.14	absent	absent	absent	0.06	IE		0.32	0.419	64	6	7	3	0.16	N	N	N
24	0.14	absent	absent	absent	0.08	IE		0.34	0.405	37	10	11	3	0.2	N	N	I
25	0.12	absent	absent	absent	0.06	IE		0.36	0.388	60	11	10	3	0.2	N	N	N

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP (mv)	DUR (sec)
26	SUJATHA	22	151	47	20.6	1.4	90	120	74	46	89.3	88	S.R	1	0.08
27	GEETHA	19	150	48	21.05	1.4	92	108	68	40	81.3	94	S.R	1	0.1
28	MEENAKSHI	22	148	44	20.09	1.34	74	110	70	40	83.3	72	S.R	1	0.08
29	SUREKHA	20	153	50	21.36	1.46	100	106	66	40	79.3	100	S.R	1	0.08
30	MAHADEVI	33	152	54	23.37	1.5	106	104	68	36	80	108	S.R	1	0.06
31	DAKSHAYANI	22	149	54	24.32	1.48	98	112	64	48	80	100	S.R	1	0.08
32	MOHINI	24	156	60	24.69	1.6	96	116	62	54	80	94	S.R	1	0.08
33	VEENA	26	157	58	23.57	1.58	88	110	68	42	82	90	S.R	0.5	0.08
34	PADMAVAT	26	157	44	17.88	1.38	84	106	70	36	82	84	S.R	1	0.08
35	SAVITRI	23	165	64	23.52	1.7	75	122	72	50	88.67	76	S.R	1	0.04
36	GEETHA	19	145	46	21.9	1.34	92	108	62	46	77.3	94	S.R	1	0.08
37	ANITHA	20	152	50	20.6	1.46	77	104	64	40	77.3	76	S.R	1	0.08
38	DHAKSHA	21	152	49	21.21	1.44	98	108	66	42	80	100	S.R	1	0.08
39	RAJESHWARI	22	153	44	19.04	1.36	85	116	68	48	84	86	S.R	2	0.08
40	REKHA NAYAGI	18	146	40	18.77	1.28	80	110	70	40	83.3	78	S.R	0.5	0.08
41	KAVITHA	23	154	47	19.8	1.42	100	112	64	48	80	100	S.R	1	0.06
42	SHRIDEVI	25	160	44	17.39	1.42	92	106	60	46	75.3	94	S.R	1	0.08
43	RIYANNA	22	149	49	22.07	1.42	90	102	62	40	75.3	88	S.R	1	0.08
44	INDINA	23	158	50	20.08	1.48	92	104	68	36	80	94	S.R	0.5	0.06
45	SUJATHA	24	156	48	19.75	1.44	75	100	60	40	73.3	76	S.R	1	0.08
46	SUNITHA	32	154	48	20.25	1.44	87	110	70	40	83.3	88	S.R	1	0.08
47	SOUMYASHREE	31	158	49	19.67	1.48	84	108	66	42	80	84	S.R	1	0.08
48	MALLAMMA	20	154	60	25	1.58	88	106	64	42	78	88	S.R	1	0.08
49	SAVITHA	24	154	49	20.6	1.44	92	108	62	46	77.3	94	S.R	1	0.06
50	KANTHA	24	150	43	19.1	1.36	102	104	60	44	74.6	100	S.R	1	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
26	0.16	absent	present	present	0.08	IE		0.36	0.405	26	9	9	3	0.16	N	N	F
27	0.12	absent	present	present	0.08	IE		0.36	0.391	82	10	11	2	0.2	N	N	I
28	0.16	absent	absent	absent	0.08	IE		0.36	0.411	70	10	11	4	0.2	N	N	N
29	0.16	absent	absent	absent	0.08	IE		0.36	0.391	56	4	4	3	0.2	N	N	N
30	0.12	absent	present	present	0.08	D	III, V4,V5	0.36	0.402	-3	6	6	2	0.16	N	N	I
31	0.12	absent	present	present	0.06	IE		0.32	0.386	79	17	13	5	0.16	N	N	I
32	0.12	present	present	present	0.08	IE		0.36	0.405	58	16	8	3	0.24	N	N	N
33	0.12	absent	present	present	0.08	IE		0.36	0.407	53	12	11	2	0.28	N	N	N
34	0.14	absent	present	present	0.08	IE		0.34	0.405	52	8	9	3	0.2	N	N	F
35	0.14	absent	absent	absent	0.08	IE		0.36	0.397	64	11	10	2	0.16	N	N	I
36	0.16	absent	absent	absent	0.06	IE		0.32	0.412	50	18	12	2	0.16	N	N	N
37	0.1	absent	absent	absent	0.08	IE		0.34	0.409	34	13	7	3	0.16	N	N	I
38	0.12	absent	present	present	0.08	IE		0.36	0.413	22	15	10	3	0.2	N	N	N
39	0.16	absent	absent	absent	0.06	IE		0.36	0.392	53	7	10	3	0.16	N	N	N
40	0.16	absent	present	present	0.08	IE		0.36	0.405	48	13	11	1	0.16	N	N	I
41	0.14	absent	present	present	0.08	IE		0.36	0.411	59	19	13	3	0.16	N	N	I
42	0.14	absent	absent	absent	0.08	IE		0.36	0.406	60	10	11	3	0.16	N	N	I
43	0.16	absent	absent	absent	0.06	IE		0.32	0.405	54	16	12	3	0.16	N	N	N
44	0.12	absent	present	present	0.08	D	III, V3,V4	0.34	0.41	50	15	10	1	0.2	N	N	N
45	0.14	absent	absent	absent	0.1	IE		0.36	0.388	36	12	8	3	0.16	N	N	I
46	0.16	absent	present	present	0.06	IE		0.36	0.405	72	11	10	2	0.16	N	N	N
47	0.14	absent	absent	absent	0.08	IE		0.36	0.398	55	13	11	3	0.16	N	N	I
48	0.12	absent	absent	absent	0.08	IE		0.32	0.392	58	12	9	3	0.16	N	N	N
49	0.16	absent	present	present	0.08	IE		0.34	0.396	76	9	5	3	0.16	N	N	I
50	0.16	absent	absent	absent	0.06	IE		0.36	0.416	73	14	13	4	0.2	N	N	F

ANNEXURE 4d: MASTER CHART – 3RD TRIMESTER OF PREGNANCY

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP(mv)	DUR(sec)
1	SUSHILA	30	1.46	49	23	1.4	94	110	70	40	83.3	94	S.R	1	0.08
2	SHOBHA	25	1.45	46	29.9	1.36	98	130	80	50	96.6	98	S.R	1	0.08
3	CHANDRIKA	19	1.5	76	33.7	1.72	104	120	80	40	93.3	104	S.R	1	0.08
4	PAVITRA	24	1.64	70	26.7	1.76	98	128	76	52	93.3	96	S.R	1	0.08
5	PALLAVI	26	1.59	63	25	1.64	96	110	72	38	84.6	98	S.R	1.5	0.06
6	SHALU	25	1.59	63	23.8	1.62	88	120	60	60	80	88	S.R	1	0.08
7	JASMINE	22	1.52	55	23.8	1.5	94	120	80	40	93.3	96	S.R	1	0.1
8	BUVHANES	28	1.47	54	25	1.46	94	120	80	40	93.3	94	S.R	0.5	0.08
9	JAYASHREE	20	1.53	55	23.5	1.52	94	114	72	42	86	94	S.R	1	0.08
10	NIRMALA	26	1.57	65	26.42	1.66	104	110	78	32	88.6	104	S.R	1	0.06
11	SAKAMMA	32	1.62	68	25.95	1.84	88	120	70	50	76.6	88	S.R	1	0.08
12	SAVITHA	22	1.5	50	22.22	1.54	94	124	80	44	92.6	94	S.R	1	0.08
13	SRIDEVI	24	1.49	56	25.22	1.44	84	114	70	44	84.6	84	S.R	1	0.08
14	BHARATI	23	1.52	49	21.21	1.44	88	120	80	40	93.3	88	S.R	1.5	0.08
15	SRIDEVI	24	1.58	52	23.11	1.46	94	130	80	50	96.6	94	S.R	1	0.08
16	SAVITHA	29	1.56	51	20.73	1.48	100	120	70	50	86.6	102	S.R	1	0.06
17	SUMASANJU	28	1.43	67	27.12	1.68	90	124	90	50	101.3	90	S.R	2	0.08
18	SMITHA	26	1.63	81	33.3	1.86	92	128	84	34	98.6	92	S.R	1	0.04
19	RAJINI	22	1.43	42	20.09	1.26	108	116	80	44	92	108	S.R	1	0.08
20	VIJAYLAXMI	25	1.63	82	30.9	1.88	84	144	88	36	106	84	S.R	1	0.08
21	LAKSHMI	25	1.59	67	26.58	1.68	102	110	70	56	83.3	102	S.R	1	0.08
22	RANJANNA	22	1.59	54	21.42	1.56	96	120	80	40	93.3	98	S.R	1	0.06
23	PRATHIBA	22	1.53	47	20.08	1.42	94	110	80	40	93.3	94	S.R	1	0.08
24	GANGAMMA	32	1.53	49	20.94	1.44	110	120	90	30	100	110	S.R	1	0.08
25	JAYAMMA	25	1.52	46	19.9	1.4	98	124	70	40	83.3	98	S.R	0.5	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
1	0.12	absent	absent	absent	0.08	IE		0.36	0.405	22	17	13	2	0.16	N	N	I
2	0.1	absent	present	present	0.08	IE		0.36	0.408	76	15	9	1	0.2	N	N	I
3	0.014	absent	present	present	0.08	IE		0.36	0.412	50	16	11	2	0.16	N	N	I
4	0.12	absent	absent	absent	0.06	IE		0.36	0.392	66	14	10	2	0.24	N	N	F
5	0.12	absent	present	present	0.08	IE		0.34	0.411	72	13	7	3	0.2	N	N	N
6	0.16	present	present	present	0.08	IE		0.36	0.409	50	9	9	4	0.2	N	N	N
7	0.16	absent	present	present	0.04	IE		0.36	0.405	54	15	10	2	0.24	N	N	I
8	0.16	absent	absent	absent	0.08	IE		0.36	0.388	7	18	12	2	0.16	N	N	I
9	0.14	absent	present	present	0.08	D	III,V4,V5	0.36	0.417	60	16	9	3	0.16	N	N	N
10	0.12	absent	absent	absent	0.08	IE		0.36	0.43	45	15	11	3	0.2	N	N	N
11	0.12	absent	present	present	0.08	IE		0.36	0.419	62	18	9	4	0.2	N	N	N
12	0.12	absent	present	present	0.08	IE		0.36	0.423	0	17	12	2	0.2	N	N	N
13	0.12	absent	present	present	0.08	IE		0.36	0.439	63	5	9	2	0.16	N	N	I
14	0.12	absent	present	present	0.08	D	III,V3,V4,V5	0.36	0.391	8	13	12	3	0.12	N	N	I
15	0.14	absent	absent	absent	0.06	IE		0.36	0.432	39	17	12	1	0.22	N	N	N
16	0.12	present	present	present	0.08	IE		0.36	0.411	0	11	5	2	0.2	N	N	F
17	0.12	absent	absent	absent	0.08	IE		0.36	0.398	80	16	8	2	0.24	N	N	I
18	0.14	absent	absent	absent	0.06	IE		0.34	0.421	18	10	9	4	0.2	N	N	I
19	0.16	absent	present	present	0.08	IE		0.34	0.423	-2	10	7	2	0.2	N	N	I
20	0.12	absent	absent	absent	0.08	IE		0.36	0.405	47	17	14	2	0.16	N	N	N
21	0.12	absent	absent	absent	0.08	IE		0.36	0.409	30	10	8	3	0.16	N	N	I
22	0.12	absent	present	present	0.08	IE		0.36	0.414	38	12	9	1	0.16	N	N	I
23	0.12	absent	present	present	0.08	IE		0.36	0.418	54	13	10	4	0.16	N	N	N
24	0.12	absent	present	present	0.08	IE		0.36	0.392	49	18	9	2	0.2	N	N	N
25	0.12	absent	absent	absent	0.08	IE		0.34	0.405	76	17	13	2	0.16	N	N	N

SL.NO	NAMES	AGE	HEIGHT	WEIGHT	BMI	BSA	PR	SBP	DBP	PP	MAP	HR	RHYTHM	P- wave	
														AMP (mv)	DUR (sec)
26	PREMA	19	1.45	53	25.23	1.46	90	126	60	54	78	92	S.R	1	0.08
27	MANJULA	22	1.46	50	23.47	1.4	86	120	86	66	108	88	S.R	1	0.04
28	LAKSHMI	22	1.46	50	23.4	1.4	84	120	84	34	95.3	84	S.R	0.5	0.1
29	KALLAMMA	29	1.5	49	21.7	1.42	88	110	70	40	83.3	88	S.R	1	0.06
30	BNAGARAM	28	1.49	58	26.12	1.52	90	100	60	40	73.3	90	S.R	1	0.06
31	SAVITHA	24	1.62	50	19.08	1.52	94	110	70	40	83.33	94	S.R	1	0.08
32	VIJAYA	29	1.5	58	25.7	1.5	108	114	80	34	91.33	112	S.R	1	0.08
33	VANI	23	1.54	52	21.9	1.48	94	110	80	30	90	94	S.R	1	0.08
34	MANGALA	24	1.5	45	20	1.36	100	120	70	50	86.6	100	S.R	1	0.08
35	SULABHA	30	1.53	58	24.7	1.64	102	98	72	26	80.6	102	S.R	1	0.08
36	SHILPA	20	1.5	56	23.3	1.64	104	114	78	36	90	104	S.R	1	0.08
37	SUMATI	28	1.56	55	22.6	1.54	94	122	64	58	83.3	94	S.R	1	0.06
38	NEETHA	27	1.53	60	25.6	1.58	100	120	74	46	89.3	102	S.R	1.5	0.08
39	LAKSHMI	22	1.48	64	29.22	1.58	92	114	72	42	86	94	S.R	1	0.08
40	SAVITHA	19	1.3	45	24.06	1.28	100	118	70	48	86	100	S.R	1	0.08
41	SHARADA	25	1.49	46	20.72	1.38	84	112	78	34	89.33	84	S.R	1	0.08
42	SUJATHA	25	1.56	60	24.69	1.6	94	126	82	44	96.6	96	S.R	0.5	0.08
43	REKHA	20	1.48	56	25.5	1.5	94	108	74	34	85.33	94	S.R	1	0.06
44	HEENA	20	1.61	54	20.84	1.56	96	112	82	30	92	96	S.R	1	0.08
45	SHOBA	25	1.5	48	21.33	1.42	108	116	80	36	92	108	S.R	1	0.04
46	KAVITHA	23	1.54	47	19.8	1.42	98	112	64	48	80	98	S.R	1	0.06
47	POORNIMA	24	1.48	58	26.48	1.5	86	118	80	38	95.33	86	S.R	1	0.08
48	JAGADEVI	30	1.56	45	20.7	1.42	94	124	82	42	96	94	S.R	1	0.08
49	SUNANDA	19	1.61	48	18.53	1.56	104	118	70	48	86	104	S.R	0.5	0.08
50	VEENA	26	1.57	58	23.57	1.58	86	110	68	42	82	86	S.R	2	0.08

SL. NO	PR interval (Sec)	Q wave			QRS (sec)	ST segment		QT (sec)	QTc (sec)	QRS Frontal Axis	Amp of R wave in mm	Amp of S wave in mm	T wave		T wave abnor malites		
		Lead I	Lead II	Lead III		IE/D	Lead						Amp (mv)	Dur (sec)	Lead I	Lead II	Lead III
26	0.14	absent	present	present	0.06	IE		0.36	0.401	22	15	9	3	0.16	N	N	I
27	0.14	absent	absent	absent	0.08	IE		0.36	0.421	76	16	11	2	0.22	N	N	I
28	0.16	absent	present	present	0.08	IE		0.34	0.389	50	14	10	4	0.24	N	N	N
29	0.1	absent	present	present	0.08	D	III,V4,V5	0.36	0.41	66	13	7	1	0.3	N	N	I
30	0.12	absent	absent	absent	0.08	IE		0.36	0.423	72	9	9	2	0.12	N	N	N
31	0.12	absent	absent	absent	0.08	IE		0.36	0.399	50	15	10	3	0.16	N	N	I
32	0.1	present	present	present	0.08	IE		0.36	0.423	54	14	12	3	0.2	N	N	N
33	0.16	absent	absent	absent	0.06	IE		0.36	0.411	56	4	4	4	0.16	N	N	N
34	0.14	absent	absent	absent	0.08	IE		0.36	0.409	-3	6	6	3	0.16	N	N	I
35	0.16	absent	absent	present	0.06	IE		0.32	0.43	79	17	13	3	0.16	N	N	F
36	0.12	absent	present	present	0.08	IE		0.36	0.422	58	16	8	4	0.2	N	N	I
37	0.12	absent	present	present	0.08	IE		0.34	0.381	53	12	11	2	0.24	N	N	N
38	0.14	absent	absent	present	0.08	IE		0.36	0.419	52	8	9	1	0.2	N	N	N
39	0.12	absent	absent	absent	0.06	IE		0.34	0.412	64	11	10	3	0.12	N	N	N
40	0.16	absent	absent	absent	0.08	IE		0.36	0.409	50	18	12	3	0.16	N	N	I
41	0.1	absent	present	present	0.08	IE		0.36	0.422	34	13	7	3	0.16	N	N	I
42	0.12	absent	absent	absent	0.08	D	III,V4,V5	0.36	0.431	22	15	10	2	0.24	N	N	I
43	0.12	absent	absent	absent	0.06	IE		0.36	0.391	53	7	10	2	0.12	N	N	N
44	0.14	absent	present	present	0.08	IE		0.34	0.416	48	13	11	1	0.2	N	N	N
45	0.12	absent	absent	present	0.06	IE		0.36	0.374	59	19	13	3	0.16	N	N	I
46	0.12	absent	absent	absent	0.06	IE		0.36	0.401	37	10	11	2	0.24	N	N	N
47	0.16	present	present	present	0.08	IE		0.36	0.405	60	11	10	4	0.2	N	N	I
48	0.14	absent	present	present	0.08	IE		0.36	0.421	26	9	9	2	0.12	N	N	I
49	0.12	absent	present	present	0.08	IE		0.36	0.399	30	10	14	1	0.12	N	N	F
50	0.12	absent	absent	absent	0.08	IE		0.34	0.402	38	12	8	2	0.24	N	N	N