Original Research Article

STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN SECOND AND THIRD TRIMESTER OF PREGNANT WOMEN AT GOVERNMENT MEDICAL COLLEGE (GMC) PURNEA, BIHAR.

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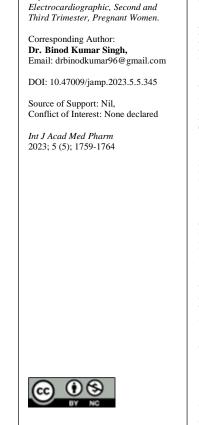[3]Assistant Professor, Department of Physiology, Government Medical College (GMC) Purnea, Bihar, India

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Abstract

Background: Electrocardiograms are necessary in the diagnosis and therapy of heart disease in pregnancy, which remains a severe problem despite the availability of other diagnostic and therapeutic tools. However, in order to make correct use of this information, it is necessary to be familiar with the changes that occur in an ECG during a normal pregnancy. **Materials and Methods:** The research was conducted at Government Medical College (GMC) Purnea, Bihar. The study was conducted from March 2021 to September 2022 for a period of 18 months. 50 healthy pregnant women between the ages of 20 and 35 who were in their second and third trimesters were arbitrarily chosen from the Government Medical College and Hospital's obstetric outpatient department. A separate group of 50 healthy non-pregnant women in the same age range was also assembled by drawing names at random from the general populace. The twelve-channel HEWLETT PACKARD Page Writer electrocardiograph, made by Philips Electronics Ltd., was used to record the electrocardiogram (ECG). **Result:** Out of a total of fifty patients in each group, three subjects during the second trimester and four subjects during the third trimester had ST segment depression ranging from 0.5 mm to 1 mm. The non-pregnant group's participants lacked any ST segment depression. However, none of the three groups showed a statistically significant difference. In comparison to the control group, the QTC interval showed a substantial increase in pregnant women during the second trimester and pregnant women during the third trimester. The QTC interval among pregnant women in the third trimester increased statistically significantly compared to those in the second trimester. **Conclusion:** Our study is by no means comprehensive, it does give a look into the range of cardiovascular system modifications that occur during a typical pregnancy and cause alterations in ECG even in the absence of any cardiac illness.

INTRODUCTION

Heart conditions play a significant part in the worldwide phenomena of maternal mortality. Although heart disease during pregnancy is exceedingly uncommon, there is a rising tendency among women who have had heart disease in the past or who may be at risk of doing so to choose to become mothers.[1] Cardiovascular disease affects about 1% of individuals worldwide, a number that is constantly rising.[2]

It places a primary emphasis on the adjustments that women are need to make in order to provide an optimum environment for fertilization, nourish the developing fetus, ensure a safe birth, and subsequently meet the nutritional requirements of the infant.[3] The cardiovascular system is the one that is most commonly affected during a regular pregnancy, but there are other physiological changes that occur as well.[4] The physiological changes that take place during a regular pregnancy are extremely important for the purpose of assisting the cardiovascular system in adapting to the mother's higher metabolic requirements. Because of these alterations, the peripheral tissues as well as the developing baby are certain to receive the appropriate volumes of oxygenated blood.[5] The physiological process of pregnancy has an effect on each and every one of the mother's bodily processes.[6]

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Changes in a woman's cardiovascular system are a normal part of pregnancy, but these changes can sometimes bring on or make an existing heart condition worse.[6] additionally, a woman's physical findings during a healthy pregnancy may fluctuate due to physiological changes, and these fluctuations may provide the erroneous impression that heart illness is present.[7]

In a healthy pregnancy, functional systolic cardiac murmurs are frequently observed, and the effort required to breathe is highlighted. As frequently mentioned, dyspnea and edema emerge in the third trimester.[7] when a pregnant woman has a systolic murmur heard over the precordium, an obstetrician will almost always refer the patient to a cardiologist for further assessment. Recording systolic murmurs is necessary in order to ascertain whether or not they are present and to identify whether or not they are an indicator of heart disease.[8]

Electrocardiograms are necessary in the diagnosis and therapy of heart disease in pregnancy, which remains a severe problem despite the availability of other diagnostic and therapeutic tools. However, in order to make correct use of this information, it is necessary to be familiar with the changes that occur in an ECG during a normal pregnancy.[9] In addition to bringing about significant alterations in the cardiovascular system, pregnancy is also associated with a variety of ECG shifts. It is important to have a good comprehension of the physiological data in this case so that one can detect the pathological alterations.[10] it is crucial to point out that the impact that pregnancy has on an EKG has been a major concern ever since the technique of electrocardiography was first developed.[9]

Alterations in the patient's ECG are taken into account when determining whether or not they have cardiovascular disease during pregnancy. In the event that diagnostic specificity is required, these alterations have to be greater than the typical variances.[11]

Aim and Objectives
The study of electrocardiographic changes in II and III trimester of pregnant women between 20-35 years of age group and normal non pregnant women of the same age group.
The comparison of ECG changes of the above three groups.

MATERIALS AND METHODS

The research was conducted at Government Medical College (GMC) Purnea, Bihar. The study was conducted from March 2021 to September 2022 for a period of 18 months. 50 healthy pregnant women between the ages of 20 and 35 who were in their second and third trimesters were arbitrarily chosen from the Government Medical College and Hospital's obstetric outpatient department. A separate group of 50 healthy non-pregnant women in the same age

range was also assembled by drawing names at random from the general populace.

Individuals who showed a desire to participate in the study were enrolled after being told about its purpose and nature and having their informed permission.

A thorough evaluation was carried out, and each chosen person's information was collected using a pretested organized proforma. The data collection procedure was carried out in the morning.

A thorough physical and systemic examination of the chosen participants was performed. During the physical examination, the height in centimeters and weight in kilograms were measured. The radial artery was palpated to determine the resting pulse rate, and blood pressure was calculated using a mercury sphygmomanometer with a cuff of the proper size. The respiratory and cardiovascular systems were also thoroughly examined clinically.

The participants were carefully examined before being screened to see if they matched the inclusion and exclusion requirements. A person was later removed from the research if they did not match the inclusion criteria or any exclusion criteria.

The twelve-channel HEWLETT PACKARD Page Writer electrocardiograph, made by Philips Electronics Ltd., was used to record the electrocardiogram (ECG).

Statistical Analysis
The results for continuous data are presented as Mean ± SD (standard deviation), while categorical data is expressed as numbers and percentages. Multiple group comparisons were conducted using one-way ANOVA, followed by post-hoc Tukey tests for group-wise comparisons. Categorical data was analyzed using the Chi-square test.
Statistical significance was determined by considering a 'p' value of 0.05 or less.

RESULTS

These are the outcomes: When compared to the control group, there was a statistically significant rise in heart rate among pregnant women in the second and third trimesters. Additionally, pregnant women in the third trimester saw statistically significant heart rate increases compared to those in the second trimester.

In comparison to the control group, there was a statistically significant reduction in the PR interval in both the second and third trimesters of pregnancy. The PR interval among pregnant women in the third trimester decreased statistically significantly compared to those in the second trimester.

Q waves occurred more frequently in pregnant women, although they still happened within the usual range.

Out of a total of fifty patients in each group, three subjects during the second trimester and four subjects during the third trimester had ST segment depression ranging from 0.5 mm to 1 mm. The non-pregnant group's participants lacked any ST segment

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depression. However, none of the three groups showed a statistically significant difference.

In comparison to the control group, the QTC interval showed a substantial increase in pregnant women during the second trimester and pregnant women during the third trimester. The QTC interval among pregnant women in the third trimester increased statistically significantly compared to those in the second trimester.

As the pregnancy went on, there was a noticeable leftward deviation in the QRS frontal axis.

In the pregnant groups compared to the non-pregnant group, T wave abnormalities, such as flat and inverted T waves in lead III and chest leads V1-V3, were more commonly seen.

Although the R wave amplitude (mm) was higher in third-trimester pregnant women than in second-trimester pregnant women and the control group, there was no statistically significant difference between the three groups.

Table 1: age wise distribution in three groups

Age Group (yrs)	Controls [A]	2nd TM [B]	3rd TM [C]
20 - 22	8	13	8
23 - 25	25	22	18
26 - 28	15	12	16
29 - 30	2	3	8
Total	50	50	50
Mean Age (yrs) + SD	24.7 + 2.2	24.3 + 2.6	25.5 + 2.8

Table 2: comparison of heart rate (bpm), pwave (sec) and pr interval (sec) between three groups

Variables		Particulars	Controls [A]	2 nd TM [B]	3rd TM [C]	Difference between groups			
						F value	A-B	A-C	B-C
Heart Rate (beats/ Min)		Mean + SD	79.0 + 6.60	87.2+ 6.16	94.2 + 8.5	F - 56.06 p < 0.05	p<0.01 S	p<0.01 S	p<0.01 S
		Range	68-100	76-104	68-110				
P wave (sec)	Duration	Mean + SD	0.08 + 0.01	0.08 + 0.01	0.08 + 0.01	F - 1.13 p > 0.05	NS	NS	NS
		Range	0.06-0.1	0.01-0.1	0.04-0.1				
	Amplitude	Mean + SD	1.01+0.15	0.99+0.25	1.00+0.24	F - 0.098 p > 0.05	NS	NS	NS
		Range	1.0 - 2.0	1.0 - 2.0	1.0 - 2.0				
PR Interval (sec)		Mean + SD	0.15 + 0.01	0.14 + 0.02	0.13 + 0.02	F - 19.8 p <0.05	p<0.01 S	p<0.01 S	p<0.05 S
		Range	0.12 - 0.16	0.10 - 0.16	0.10 - 0.16				

Multiple group comparison: one-way Anova, F-Test Group wise comparison: 'Post-Hoc- Tukey' Test p<0.05, P<0.01: S Significant p<0.001: HS- Highly Significant p>0.05: NS- Not Significant.

Table 3: comparison of occurence of q wave in limb leads between three groups

Lead	Control (A)		2nd TM (B)		3rd TM (C)		Difference between groups			
	n	(%)	n	(%)	n	(%)	Chi square value X2	A-B	A-C	B-C
I	2	4%	3	6%	4	8%	0.71 p>0.05 NS	NS	NS	NS
II	13	26%	25	50%	34	68%	17.8 p<0.001 S	6.11 p<0.05 S	17.7 p<0.01 S	3.35 p>0.05 NS
III	20	40	27	54	33	66	12.1 p<0.05 S	1.97 p>0.05 NS	6.78 p<0.01 S	1.50 p>0.05 NS
avR	-		-		-		-	-	-	-
avL	2	4	-		3	6	2.89 p>0.05 NS	NS	NS	NS
avF	19	38	25	50	32	64	6.78 p<0.05 S	1.46 p>0.05 NS	6.76 p <0.01 S	1.99 p >0.05 NS

Categorical Data was analysed by: Chi-Square Test p < 0.05, p< 0.01: S – Significant.
p < 0.001: HS – Highly Significant.
p > 0.05: NS – Not Significant.

Table 4: comparison of occurrence OFQ wave in chest leads between three groups

Lead	Control (A)		2nd TM (B)		3rd TM (C)		Difference between groups			
	n	%)	n	(%)	n	(%)	Chi square value X 2	A-B	A-C	B-C
V1	-		-		-		-	-	-	-
V2	-		-		-		-	-	-	-
V3	-		-		-		-	-	-	-
V4	4	8	6	12	8	16	1.52 p>0.05 NS	NS	NS	NS
V5	25	50	29	58	32	64	2.02 p>0.05 NS	NS	NS	NS
V6	27	54	30	60	32	64	1.05 p> 0.05 NS	NS	NS	NS

Categorial Data was analysed by: Chi-Square Test p < 0.05, P< 0.01: S – Significant.
p < 0.001: HS – Highly Significant.
p > 0.05: NS – Not Significant.

Table 5: comparison of ST segment depression between three groups

Variables	Particulars	Controls [A] n%	2nd TM [B] n%	3rd TM [C] n%	Difference between groups			
					Chi square value X2	A-B	A-C	B-C
S T Segment	S T Depression	-	36%	48%	X2 =3.86 p > 0.05	N S	N S	N S

Categorial Data was analysed by: Chi-Square Test p < 0.05,

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P< 0.01: S – Significant.
p < 0.001: HS – Highly Significant.
p > 0.05: NS – Not Significant.

Table 6: comparison of QRS complex (sec), qt interval (sec), QTC interval (sec) and QRS frontal axis (degrees) between three groups

Variables	Particulars	Controls [A]	2nd TM [B]	3rd TM [C]	Difference between groups			
					F value	A-B	A-C	B-C
QRS Complex (in sec)	Mean + SD	0.08 + 0.01	0.08 + 0.01	0.08 + 0.01	F -0.95 p > 0.05 NS	NS	NS	NS
	Range	0.06 - 0.08	0.06 - 0.1	0.06 - 0.1				
QT interval (in Sec)	Mean + SD	0.35 + 0.02	0.35 + 0.01	0.36 + 0.01	F- 0.72 p > 0.05 NS	NS	NS	NS
	Range	0.32 - 0.36	0.32 - 0.36	0.32 - 0.36				
QTc interval (in sec)	Mean + SD	0.38 + 0.01	0.40 + 0.01	0.41 + 0.01	F-61.44 P< 0.001 HS	p < 0.01 S	p < 0.01 S	p<0.01 S
	Range	0.35 - 0.41	0.37 - 0.42	0.37 - 0.44				
QRS Frontal axis (in degrees)	Mean + SD	64.10 + 6.8	53.0 + 16.7	45.9 + 22.1	F - 15.31 p < 0.001 HS	p < 0.01 S	p < 0.01 S	p<0.09 NS
	Range	48 - 82	(-3) -82	(-3) - 80				

Table 7: comparison of t wave abnormalities in limb leads between three groups

Lead	Control (A)			2nd TM (B)			3rd TM (C)			Difference between groups			
	Flat n %	Inverted n %	Total n %	Flat n %	Inverted n %	Total n%	Flat n%	Inverted n%	Total n%	Chi square value X2	A-B	A-C	B-C
I	-	-	-	-	-	-	-	-	-	-	-	-	-
II	-	-	-	-	-	-	-	-	-	-	-	-	-
III	510	20 40	2550	1224	23 46	3570	1326	2754	4080	X2=10.5 p < 0.01 S	4.17 P<0.05 S	9.89 p<0.01 S	1.33 p>0.05 NS
avR	-	-	-	-	-	-	-	-	-	-	-	-	-
AvL	-	-	-	-	-	-	-	-	-	-	-	-	-
AvF	-	-	-	-	-	-	-	-	-	-	-	-	-

Categorial Data was analysed by Chi-Square Test p < 0.05, p < 0.01: S – Significant, p < 0.001: HS – Highly Significant. p > 0.05: N S – Not Significant.

DISCUSSION

The cardiovascular system experiences notable alterations during pregnancy. Due to these changes, the ECG during a healthy pregnancy may deviate significantly from the established norm in the absence of cardiac illness that can be clearly seen.[12-15]

Pregnancy-related electrocardiographic alterations might result from:

The diaphragm is pushed higher by the uterus during pregnancy, changing the location of the heart.

The heart is moved laterally, raised, and somewhat rotated to its long axis due to the altered spatial configuration of the chest organs.

Modifications in the sympathetic and hormonal regulation of the electrical cardiac activity during pregnancy, which have an impact on the myocardium's electrical characteristics.

It is noteworthy to note that these ECG abnormalities may be explained by the possibility that pregnancy is linked to a concentric expansion of the left ventricle in response to the demands of hemodynamics.[16]

Heart rate

Both pregnant women in their second and third trimesters in this research had statistically significant heart rate increases when compared to controls.

Dramatic circulatory modifications, such as a rise in heart rate, are linked to pregnancy. The modifications in the autonomic nerve system that result in changes in cardiac autonomic modulation are responsible for this rise in heart rate. Obstetric complications might arise if these modifications fail. HR rises by 10–12 beats per minute.

Early pregnancy heart rate increases are caused by hormonal mechanisms. Early in pregnancy, chorionic gonadotropin (hCG) production may be associated with the first shift in heart rate, whereas the later, steady rise may be associated with vascular changes that go along with placental and fetal development.[12] This rise in heart rate, which occurs mostly in the third trimester, makes up for the decline in stroke volume brought on by caval compression.[13] Additionally, the cardiac output starts to increase as early as week 5 and reaches a peak of 45% above the baseline by week 24 of pregnancy. Increases in heart rate and stroke volume are used to achieve this.[17,18]

Previous research by Ueland K. et al., Charles.B. et al,[19] Katz R. et al,[10] Ibrahim S. et al,[14] Suzanne M. et al,[19] and Carla A. et al,[18] also revealed findings of a similar kind.

P wave

When compared to controls, P wave measurements in the second and third trimesters of pregnancy did not reveal any statistically significant differences.

PR interval

When compared to controls, the PR interval in our research significantly decreased in both the second and third trimesters for pregnant women. When compared to pregnant women in the second trimester, there was a statistically significant decrease in PR interval in the third trimester.

The shortening of A-V conductance in response to the pregnancy-related rise in heart rate may be the cause of the decrease in PR interval.[14]

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Similar studies by Joseph E. Carruth et al. indicated that, although statistically significant, the mean PR interval was clinically undetectable during the third trimester when compared to the first and second trimesters of normal pregnancy.[11]

Q Wave

In this study, pregnant women saw an increase in Q Wave incidence compared to controls; however it was still within normal ranges in lead II, III, and avF. These ECG alterations might be the consequence of an increase in the amount of vasopressors in the blood or they could be due to pregnancy-related diaphragmatic abnormalities.[11] When compared to typical non-pregnant women, pregnant women experience Q waves more frequently, which may be caused by the heart's changing location.[13]

Similar results were found in studies by Misra J et al,[6] Veille JC et al,[11] Singh AD et al,[13] and Carruth JE et al.[11]

QRS Complex

When compared to controls, the length of the QRS Complex in this study's pregnant participants' second and third trimesters did neither increase or decrease.

ST segment

In asymptomatic individuals, ST Segment departure from the isoelectric line is a predictor of coronary events.

ST segment depression is the sort of ST segment alteration identified in this investigation. In the second and third trimesters, only 3 and 4 individuals, respectively, of healthy pregnant women demonstrated ST depression. The depression in every individual ranged from 0.5 mm to 1 mm. None of the control group participants displayed ST depression. All three groups did not statistically vary from one another.

Previous investigations have demonstrated non-ischemic alterations in women caused by the digoxin-like effects of estrogen. Preogesterone level fluctuations have also been linked to such ST segment depression in young women.[16]

Electrolyte imbalance, such as hypokalaemia, of a serious enough degree to result in depression of the ST Segment due to persistent vomiting during pregnancy may be one of the possibilities for ST Segment depression during pregnancy. None of the trial participants reported having frequent vomiting. So, hypokalaemia may be ruled out as the reason for ST segment depression.[17]

Drug therapy: Since digitalis and any other medications that can have an impact on the cardiogram were administered during pregnancy, the investigation can rule out the possibility that digitalis caused the ST segment to droop. 17It has been proposed that short-lived ST segment depression is triggered by anxiety-inducing events and that this is due to a "hypersensitivity" to endogenous adrenalin. There is no clear explanation for how adrenaline causes ST segment depression. One of the theories is that, in addition to the increased muscle activity and coronary dilatation that adrenalin evokes, it also causes higher oxygen demand. An increase in circulating humoral substances that would have a direct impact on cardiac electrical activity may accompany anxiety15.

Therefore, anxiety may be a significant contributing factor to ST segment depression along with pregnancy-specific variables such changes in heart position.

In their studies, Oram S. et al,[17] Boyle DM et al,[9] Velle JC et al,[11] and Singh AD et al,[13] reported similar findings.

QT interval

When compared to controls, there was no difference in the QT interval between pregnant women in the second and third trimesters of this research.

QTC Interval

The electrocardiogram's QTC Interval measures how long the ventricular myocardium needs to depolarize and repolarize. When adjusted for heart rate, the QT interval is (QTC).

Although the QT interval (Sec) did not change statistically significantly in this study, it was discovered that when the QT interval was corrected for heart rate, which was significantly elevated in our study, both pregnant women in the second and third trimesters showed statistically significant increases in the QTC interval (Sec) when compared to controls. Therefore, an increase in heart rate might be the cause of the rise in QTC. This may be related to variations in the patterns of ventricular depolarization and repolarization during pregnancy, and these variations in regulatory systems must be taken into account as a complicated result.[15] The study comes to the conclusion that extended QTc should be seen as "an unspecific sign of changed course of repolarization".[16]

Similar findings were reported in studies by Carruth JE et al,[11] Lechmanova et al,[16] Ozmen N et al,[5] and Oram S et al.[17]

T wave

According to this study, pregnant women were more likely than non-pregnant women to experience T Wave abnormalities including flat and inverted T Waves in lead III and chest leads V1–V3.

The aforementioned discovery may be explained by a momentary increase in cardiac workload brought on by pregnancy's increased blood volume, which may result in a brief ischaemia, as seen by a T Wave inversion.[6]

Similar results were observed in studies by Misra J et al,[6] Singh R et al,[4] Oram S et al,[7] and Veille JC et al.[11]

According to certain reports, normal pregnant women may have ST depression and flat or negative T Waves during their pregnancies. This information should be kept in mind while reading pregnant women's electrocardiograms.[4]

QRS Frontal axis: In this study QRS frontal axis showed statistically significant decrease in both pregnant women in 2nd trimester and 3rd trimester when compared to controls. There was no statistically significant difference between pregnant women in 2nd trimester and pregnant women in 3rdtrimester.

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Ventricular activation time (VAT): Ventricular activation time is the time taken for impulse to traverse the myocardium from the endocardial to the epicardial surface.

In this study Ventricular activation time (sec) was compared between controls, pregnant women in 2nd trimester and 3rd trimester. There was no statistically significant difference in the VAT in pregnant women in 2nd trimester and 3rd trimester when compared to controls.

Amplitude of R wave: R wave is produced by depolarization of interventricular septum and apices of ventricles.

In this study, though amplitude of R wave (mm) slightly increased in pregnant women in 3rd trimester when compared to pregnant women in 2nd trimester and controls, there was no.

Statistically significant difference in the values among all the three groups.

Amplitude of S wave

S waves are created when the basal regions of both ventricles depolarize.

In this investigation, there was no statistically significant difference in the amplitude of the S wave (mm) between pregnant women in the second and third trimesters and controls.

Pregnancy has been linked to physiological ventricular hypertrophy, which is thought to happen as an adaptive reaction to increased preload and work done, as shown by a higher R/S ratio in leads V1 and V2 of the ECG.19 none of the pregnant research participants had any electrocardiographic signs of heart hypertrophy.

CONCLUSION

Our study is by no means comprehensive, it does give a look into the range of cardiovascular system modifications that occur during a typical pregnancy and cause alterations in ECG even in the absence of any cardiac illness. Further investigation is required to fully understand the impact of normal pregnancy on electrocardiograms, despite the fact that we do understand these alterations to some extent and that only a small number of studies have been conducted on this topic.

REFERENCES

1. Kessel E, Awan A, Martin JF, Omran AR,Ullman D. Magnitude and causes of maternal mortality as a basis for its prevention. In : Maternal and child care in developing countries. Assessment – Promotion Implementation. Switzerland: 1989 .p.80.
2. Chia P, Chia H, Subramanian R. A clinical approach to heart disease in pregnancy part 1 : General considerations in management. The obstetrician and gynecologist. 2002 July;4(3):162-68.
3. Bijalani RL, Bhole MV, Dutta S, Dikshi MB, Kashalikar SJ,Kothari LK et al. Physiology of pregnancy in Lactation. In: Understanding Medical Physiology A text book of medical students. 3rd ed. New Delhi: Jaypee Brothers; 2006. p.582.
4. Singh R, Gahlaut DS, Kishore N, Hingorant P. Electrocardiographic changes during normal pregnancy. J Obstet Gynaecol India 1968; 18:34-38.
5. Oakeley C, Warnes CA. Physiological changes in pregnancy. In: Heart disease in pregnancy. 2nd ed.Boston: Wiley Blackwell; 2007 .p.6.
6. Misra J, Dutta B, Ganguly D. Electrocardiographic study in pregnant women in normal and toxemia of pregnancy. J Obstet Gynecol India 1986; 36:635-38.
7. Cunnigham GF, Leveno KJ, Bloom SL, Hauth JC, Gilstrap LC, Wenstrom KD. Cardiovascular disease. In: William obstetrics. 22nd ed. USA: Mc Graw Hill Publications; 2005 .p.1018.
8. Harvey PW. Alternations of the cardiac physical examination in normal pregnancy. Clin Obstet Gynecol 1975; 18:54.
9. Boyle DM, Jones RLL. The electrocardiographic ST segment in normal pregnancy. J Obstet Gynecol Br CWlth 1966; 73:986-87.
10. Williams L, Wilkins. The heart and pregnancy. In: Text book of cardiovascalr medicine. 2nd ed. USA: A wolters Kluwer Company; 2004 .p.736.
11. Simmons LA, Gilun AG, Jeremy RW. Structural and functional changes in left ventricle during normotensive and pre-eclamptic pregnancy. Am J Physiol Heart Circ Physiol 2002;283(4):1627-33.
12. Iwobi NN, Dapper DV. QRS axis deviation in Nigerian women during normal pregnancy. West Afr J Med 2002;21(3):241-43.
13. Desai DK, Moodley J, Naidoo DP. Echocardiographic assessment of cardiovascular haemodynamics in normal pregnancy. obstetrics and gynecology 2004;104(1):20-27.
14. Ozmen N, Cenbeci BS, Yiginer O, Machu M,Kardesoglu E,Dincturk M. P wave dispersion is increased in pregnancy due to shortening of minimum duration of P. Does this have clinical significance. The Journal of International Medical Research 2006;34:468-74.
15. Wiley J, Sons. Maternal left ventricular transverse and long axis systolic function during pregnancy. Ultrasound in obstetrics and gynecology. 2009;18(5):465-74.
16. Kottari CR. Sampling fundamenta ls In : Research methodology by C.R. Kottari, 2nd ed. 1994 .p.213-20.
17. Clapp III, James F. Maternal heart rate in pregnancy. Am J Obstet Gynecol 1985;152:659- 60.
18. Julian DG, Wenger NK. Heart disease and heart surgery during pregnancy. In : women and heart disease. United Kingdom : 2000 .p.196.
19. Mitchell JH, Shapiro AP, Tex D. The relationship of adrenalin and T-wave changes in anxiety state. Am Heart J 1954;48(3):323-26.

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