

## A study of changes in heart rate and ECG during Second and Third trimester of Pregnancy – A comparative study

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### Abstract

**Introduction:** Physiological changes in the cardiovascular system (CVS) that occur during normal pregnancy facilitate the adaptation of the CVS to the increased metabolic needs of the mother, thus enabling adequate delivery of oxygenated blood to the peripheral tissues and fetus. These normally occurring changes in pregnancy sometimes simulate heart disease. It thus becomes essential to distinguish the physiological variations from pathological changes.

**Materials and Method:** A total of 150 healthy subjects were studied (50 non pregnant, 50 from 2<sup>nd</sup> trimester, 50 from 3<sup>rd</sup> trimester) for variations in heart rate and specific characteristic ECG waveforms.

**Results:** Significant increase in the heart rate with advancement of pregnancy as compared to non pregnant individuals was observed. PR interval, QTc showed significant changes not only in 2<sup>nd</sup> trimester as compared to non pregnant subjects but also in 3<sup>rd</sup> trimester as compared to 2<sup>nd</sup> trimester subjects.

**Conclusion:** ECG findings in Pregnancy should be interpreted with caution.

**Keywords:** Heart rate, ECG, Hemodynamics, Pregnancy.

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### Introduction

Pregnancy is the process by which life of a baby begins in the mother's womb and progresses up to the stage when it is safe to expose the baby to the external world. Various physiological changes, especially changes in the cardiovascular system (CVS) occur during normal pregnancy. These physiological changes facilitate the adaptation of the CVS to the increased metabolic needs of the mother, thus enabling adequate delivery of oxygenated blood to the peripheral tissues and to the fetus.<sup>(1)</sup> Pregnancy not only causes remarkable changes in CVS but also brings about various changes in ECG. These normally occurring changes in pregnancy sometimes simulate heart disease.<sup>(2)</sup> In addition, many of the physiological adaptations of normal pregnancy alter the physical findings thus misleading the diagnosis of heart diseases.<sup>(3)</sup> Some studies suggest that fetal sex can be a risk factor for the development of cardiac and hypertensive disorders in pregnancy.<sup>(33)</sup> So it becomes necessary to understand the physiological findings as to distinguish them from those due to cardiovascular pathology.

The effect of pregnancy on the electrocardiogram (ECG) has been a subject of great interest. The objective of the present study is to determine changes in ECG with respect to second and third trimesters of pregnant women as compared to non pregnant women in a population in a tertiary care hospital in a southern state of India.

### Materials and Method

**Study population:** Apparently healthy pregnant women (cases) presenting to the obstetric outpatient department of a tertiary care hospital at Vijayawada for antenatal care. Apparently healthy non-pregnant, age matched (controls) women were selected randomly from the workers of the same hospital. Following an explanation about the purpose and procedure of the study, subjects willing to participate were included after obtaining written informed consent. A detailed assessment was done on a pretested structured proforma.

**Sample size:** A total of 150 subjects were studied as per the calculation of sample size (50 control, 100 cases).

Women aged between 20-30 years who were included in the study were categorized into three groups as follows:

Group 1: 50 non-pregnant healthy women (controls).

Group 2: 50 pregnant women in second trimester (13-28 weeks of gestation)

Group 3: 50 pregnant women in third trimester (29 weeks to term).

**Exclusion criteria:** All women with the following characteristics

1. Age less than 20 and more than 30 years.
2. Any known cardiac diseases or Toxemia of pregnancy.
3. Renal disease.
4. Anemia.
5. Known hormonal or endocrinal disorders.

6. Known diabetes mellitus, hypertension or underwent surgery in the past one year.
7. Women on oral or injectable contraceptive measures.
8. Women on regular physical training (like athletes).

**Study duration:** One year.

**Type of study:** Cross-sectional study

All data was collected, compiled on MS EXCEL worksheets and analysed using SPSS software (version 16). Numerical data was expressed as mean and standard deviation. Statistical significance was determined using student t-test. P value <0.05 was considered statistically significant.

Examination of all the subjects included recording of ECG (after rest of 10 minutes) in standard limb lead II (lead derived from placement of the negative electrode on the right arm and positive electrode on left leg) in supine position.

Recording of ECG

- Lead wires identified with letters RA(right arm), LA(left arm), LL(left leg) and RL(right leg) were fixed to the electrodes & were placed on the respective sites.
- Right foot was connected for grounding.

- Lead selector knob was adjusted to record ECG of the 12 leads in the following order: I, II, III, aVR, aVL, and aVF.
- Chest electrodes (V<sub>1</sub>-V<sub>6</sub>) were placed in appropriate position.
- ECG was evaluated for different parameters like heart rate, P wave, PR interval, QRS Complex, T wave, QT interval, QTc interval, axis deviation and results were drawn.

The Electrocardiograph used was a single channel ECG machine-CARDIART 108T /MK VII manufactured by BPL Electronic Limited with frequency response 0.3Hz to 120Hz (AHA) without filter@ 5mm/mV, 0.3-100Hz (IEC) without filter@ 10mm/mV and sensitivity continuously variable from 0 to 15 mm/mV with marking for 10mm/mV calibration. On ECG paper, the vertical axis represents voltage (amplitude) and horizontal axis represents time (duration). With normal standardization, each 1mm represents 0.1 mV vertically and horizontally 1 mm represents 0.04 sec.

#### Observations

Mean distribution of age in different study groups is shown in Table 1 which were comparable.

**Table 1: Age Wise Distribution in Three Groups**

Age Group(yrs)	Group 1 Controls	Group 2 2 <sup>nd</sup> Trimester	Group 3 3 <sup>rd</sup> Trimester
20-25	29	34	22
26-30	21	16	28
Total	50	50	50
Mean age ± SD	25.1 ± 2.7	24.6 ± 2.3	26 ± 1.7

Mean distribution of Heart rate, P wave, PR interval, QRS complex, QT interval, QTc interval between Non pregnant with Second trimester of pregnancy have been described in Table 2.

**Table 2: Mean distribution of Heart rate, P wave, PR interval, QRS complex, QT interval, QTc interval between Non pregnant with Second trimester of pregnancy**

Parameters	Group1 (non pregnant), n=50	Group 2 (2 <sup>nd</sup> trimester), n=50	'p' value
Heart rate	79 ± 6.6	87.3± 6.1	< 0.0001*
P wave	0.08 ± 0.01	0.08 ± 0.01	1.000
PR interval	0.15 ± 0.01	0.14 ± 0.01	< 0.0001*
QRS complex	0.08 ± 0.01	0.08 ± 0.01	1.000
QT interval	0.35 ± 0.02	0.35 ± 0.01	1.000
QTc interval	0.38 ± 0.01	0.40 ± 0.01	< 0.0001*

\*p value <0.05 considered statistically significant

Table 3 shows comparison of Mean distribution of Heart rate, P wave, PR interval, QRS complex, QT interval, QTc interval between Second and Third trimesters of pregnancy.

**Table 3: Mean distribution of Heart rate, P wave, PR interval, QRS complex, QT interval, QTc interval between Second and Third trimesters of pregnancy**

Parameters	Group 2 (2 <sup>nd</sup> trimester), n=50	Group 3 (3 <sup>rd</sup> trimester), n=50	'p' value
Heart rate	87.3± 6.1	94.3± 8.5	< 0.0001*
P wave	0.08 ± 0.01	0.08 ± 0.01	1.000
PR interval	0.14 ± 0.01	0.13 ± 0.01	< 0.0001*
QRS complex	0.08 ± 0.01	0.08 ± 0.01	1.000
QT interval	0.35 ± 0.01	0.36 ± 0.01	< 0.0001*
QTc interval	0.40 ± 0.01	0.41 ± 0.01	< 0.0001*

\*p value <0.05 considered statistically significant

## Discussion

The present study was designed to analyse the changes in heart rate and determine the incidence of physiological variations in ECG of normal pregnant women compared with non pregnant women of same race and age group. Most of the ECG changes that occur during pregnancy can be explained by physiological adaptations in response to normal pregnancy.

In the present study, age matching was done. However it was observed by Zhao XQ et al<sup>(31)</sup> that the older pregnancy women with the age of over 35 are more likely to have more ECG abnormalities, arrhythmia, myocardial ischemia and so on as compared to pregnant women below 35 years of age.

**Heart Rate:** The study shows statistically significant increase in heart rate ( $p < 0.0001$ ) in group 2 and group 3 when compared to group 1. There was also statistical significant increase between groups 2 and 3. Increase in 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.<sup>(4)</sup> The increase in heart rate in early pregnancy can be linked to the production of chorionic gonadotropin,<sup>(5)</sup> with the later increase being related to the vascular changes that accompany placental and fetal growth.<sup>(6)</sup>

The increase in heart rate may have been triggered to maintain the cardiac output in a state of relative hypovolemia<sup>(7)</sup> or due to decrease in vagal baroreflex as well as parasympathetic tone.<sup>(8)</sup> Duvekot JJ et al<sup>(9)</sup> concluded that heart rate of a pregnant women steadily increased throughout pregnancy which is in correlation with present study. Findings on heart rate changes with pregnancy in studies conducted by Halphen et al,<sup>(6)</sup> Capeless et al,<sup>(27)</sup> Van open AC et al<sup>(28)</sup> and Simmons et al<sup>(29)</sup> are in line with present study.

Heart rate variability during pregnancy could be attributed to psychogenic factors too. Mizuno T<sup>(32)</sup> et al noticed that anxiety during pregnancy decreased heart rate variability and anxiety in second trimester pregnancy promoted a subsequent increase in sympathetic activity.

ECG waveform changes analysed are as follows:

**P Wave:** P wave duration neither showed any statistical significant difference in group 2 and 3 when compared to group 1 nor between groups 2 and 3. Nandini. B.N et al<sup>(10)</sup> observed that there was no change in duration of P wave in pregnancy which is in consonance with present study.

**PR Interval:** In this study PR interval showed statistically significant decrease in group 2 and 3 when compared to group 1. There was also statistically significant decrease between groups 2 and 3. The decrease in PR interval during pregnancy could be due to shortening of atrioventricular (A-V) conduction with respect to increase in heart rate during pregnancy<sup>(11)</sup> or accelerated A-V node conduction. Accelerated A-V conduction may be attributed to high sympathetic tone, A-V node bypass, small A-V node, increased heart rate, individual differences, fever, exercise, hypoxia, hyperthyroidism, anaemia and other sympathomimetic drugs.

In late pregnancy as compared to early pregnancy, PR shortening was higher and this may be due to the increased maternal blood volume, increased oxygen demand, relative hypoxia. The findings of the present study are in line with observations made by Carruth JE et al<sup>(12)</sup> and Nandini B.N et al.<sup>(10)</sup>

**QRS Complex:** QRS Complex measurement in this study showed no increase or decrease in duration in group 2 and 3 when compared to group 1. There was no statistically significant difference even between groups 2 and 3. N.N Iwobi et al(2002),<sup>(13)</sup> MutiGoloba et al(2010),<sup>(15)</sup> B.N. Nandini et al(2014)<sup>(14)</sup> in their studies during different trimesters of pregnancy concluded that there was no change in duration of QRS complex which is correlating with findings of the present study. However, Mameli P et al<sup>(16)</sup> showed that during pregnancy, mean electrical axis of QRS significantly diverted to the left side at the end of III trimester by about 6 degrees.

**QT Interval:** In this study, QT interval showed no change in duration in groups 2 and 3 when compared to group 1. There was no difference even between group 2 and group 3. Majid Zamani et al<sup>(24)</sup> concluded that there

was no change in duration of QT interval in pregnancy which is in correlation with present study.

**QTc interval:** In this study, QTc showed statistically significant increase in group 2 and 3 when compared to group 1. There was also statistically significant increase between groups 2 and 3. Gradual increase in QTc with advance in duration of pregnancy could be due to various causes which affect electrical activity of heart.<sup>(17)</sup> The surface electrocardiographic QTc interval reflects complex and interrelated aspects of cardiac electrophysiology, cardiac geometry, torso shape, tissue impedance and biological signal processing.<sup>(17)</sup> 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.<sup>(18)</sup> An increase in the QTc interval may be also due to tachycardia.<sup>(19)</sup>

The studies conducted by Komal Ruikar et al,<sup>(20)</sup> N Ozmen et al,<sup>(22)</sup> Oram S et al,<sup>(23)</sup> Kadish AH et al<sup>(17)</sup> and Lechmanová et al<sup>(21)</sup> conclude that there was prolongation of QTc interval in pregnancy which is in line with the present study.

**T Wave:** In this study, T wave was found to be upright with normal amplitude in groups 2 and 3 when compared to group 1. Cunningham GF et al<sup>(3)</sup> concluded that pregnancy does not alter the voltage findings. Veille JC et al<sup>(25)</sup> observed that T wave inversion in V<sub>2</sub> was more frequent in the pregnant than in the non pregnant patients. One pregnant lady had runs of premature ventricular contractions. Two patients had marked T wave peaking, and one had a biphasic T wave in V<sub>2</sub>. In spite of these findings, there was no difference in the overall impression regarding the normality of the ECG between either of the two stages of pregnancy and controls.

L. Wallace et al<sup>(26)</sup> found that inversion of the T wave in leads V<sub>2</sub> and V<sub>4</sub> occurred during the last trimester of pregnancy, which reverted back to its normal upright form within one week following delivery. This is possibly as a result of dilatation of the heart in pregnant women.

Glenna C L Bett<sup>(30)</sup> opined that gestational cardiac hypertrophy and a physical shift of the heart contribute to changes in the ECG. Some changes in the ECG are clearly the result of changes in ion channel expression and behaviour, but little is known about the ionic basis for this electrical remodelling. Most information comes from animal models, and implicates changes in the delayed-rectifier channels. However, it is likely that there are additional roles for sodium channels as well as changes in calcium homeostasis.<sup>(30)</sup>

## Conclusion

It is established by our study that significant ECG changes do occur in the second and third trimesters of pregnancy. These changes are due to the overall changes in the cardiovascular function of pregnant women due to the existence and growth of the foreign body, the fetus. Pregnancy changes the biophysical architecture, as well as the hemodynamics of the women which is reflected by these changes. ECG findings in pregnant subjects especially in 2<sup>nd</sup> and 3<sup>rd</sup> trimester may be normal inspite of being different from non-pregnant status. Hence the interpretation of ECG findings in pregnant subjects required caution.

## References

1. Bijalani RL, S. Manjunath. Physiology of pregnancy in Lactation. In: Understanding Medical Physiology. A text book of medical students. 4th ed. New Delhi: Jaypee Brothers; 2011:p.498.
2. 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.
3. Cunningham GF, Leveno KJ, Bloom SL, Hauth JC, Roule, Spong. Cardiovascular disease. In: William obstetrics. 23rd ed. USA: McGraw Hill Publications; 2010:p.958.
4. Stein PK, Hagley MT, Cole PL, Domitrovic PP, Kleigler RE, Rottman JN. Changes in 24 hours heart rate variability during normal pregnancy. Am J Obstet Gynecol. 1999;180(4):978-85.
5. Singh AD, Devi L, Singh L, Devi R, Singh J. Electrocardiographic findings at term, labour and immediate postpartum. J Obstet & Gynecol of India. 1986;36:316-19.
6. Halphen C, Leguludec D, Valent R, Haiat R. Electrocardiographic study of left ventricular performance in normal pregnancy. Arch Mal Coeur Vasis 1984;77(2):212-17.
7. Schier RW, Briner VA. Peripheral arterial vasodilatation hypothesis of sodium and water retention in pregnancy: implications for pathogenesis of pre-eclampsia. Obstet Gynecol. 1991;77:632-639.
8. Voss A, Malberg H, Schumann A, Wessel N, Walther T, Stepan H. Baroreflex sensitivity, heart rate and blood pressure variability in normal pregnancy. Am J Hypertens. 2000;13:1218-1225.
9. Duvekot JJ, Cheriex EC, Pieters FA, Meheere PP, Peters LL. Early pregnancy changes in hemodynamics and volume homeostasis are consecutive adjustments triggered by a primary fall in systemic vascular tone. Am J Obstet Gynecol. 1993;169:1382- 1392.
10. Nandini. B.N, Shiva Kumar D.G, Manjunath, Girish Babu. Shortening of PR-Interval in different trimesters of pregnancy-a cross-sectional study. International Journal of Biomedical and Advance Research. 2011;2(11):421-26.
11. Adamson DL, Piercy CN. Managing palpitations and arrhythmias during pregnancy. Postgrad Med J 2008;84:66-72.
12. Carruth JE, Mirvis SB, Brogan DR, Wenger NK. The electrocardiogram in normal pregnancy. Am Heart J. 1981;102:1075-78.
13. Iwobi NN, Dapper DV. QRS axis deviation in Nigerian women during normal pregnancy. West Afr J Med. 2002;21(3):241-43.

14. B.N. Nandini, D.G. Shivakumar, Manjunath Aithal, Sunkeswari Sreepadma. Occurrence of Q wave, QTc interval and QRS frontal axis during different trimesters of Pregnancy- a cross sectional study. *International Journal of Current Research and Academic Review*. 2014;2(7):79-88.
15. Muti Goloba, Scott Nelson, Peter Macfarlane. The Electrocardiogram in Pregnancy. *Computing in cardiology 2010*; 37:693-696.
16. Mamelì P, Girasudi D, Viridis A, Firinuc, Marini S, Marrosu P et al. Determination of the range of physiologic variation of the electrical axis using P, QRS and T waves in pregnancy. *Boll Soc Ital Biol Sper* 1989;65(5):391-98.
17. Kadish AH, Greenland P, Limacher MC, Frishman WH, Daugherty SA, Schwartz JB. Estrogen and progestin use and the QT interval in postmenopausal women. *Ann Noninvasive Electrocardiol* 2004;9:366-74.
18. Zachary HB. Duration of the Q-T interval in normal pregnant women. *Am Heart J*. 1949; 1(38):119-122.
19. Lechmanova M, Parizek A, Halaska M, Slavicek J, Kittnar O. Changes of the electrical heart field and hemodynamic parameters in 34th to 40th weeks of pregnancy and after delivery. *Arch Gyneol Obstet*. 2002;266:145-51.
20. Komal Ruikar, Vitthal Khode, Neelam Deokar. Corrected QT Interval (QTc) Among Pregnant Women During Different Gestational Periods. *Heart India*. 2014;2(2):43-46.
21. Lechmanova M, Kittnar O, Mleek M, Kolarick J, Parizek A. QT dispersion and T-loop morphology in late pregnancy and after delivery. *Physiol Res*.2002;51:121-29.
22. N Ozmen, BS Cebeci, O Yiginer, M Muhcu, E Kardesoglu and M Dincturk. P-wave Dispersion is increased in Pregnancy Due to Shortening of Minimum Duration of P: Does This Have Clinical Significance? *Journal of International Medical Research*. 2006;34:468.
23. Oram S, Holt M. Innocent depression of the ST segment and flattening of the T-wave during pregnancy. *J Obstet & Gynecol*. 1961;68(5):765-70.
24. Majid Zamani, Mehrdad Esmailian, Zahra Yoosefian. QT Interval in Pregnant and Non-pregnant Women. *Emergency* (2014);2(1):22-25.
25. Veille JC, Kitzman DW, Bacevice AE. Effect of pregnancy on electrocardiogram in healthy subjects during strenuous exercise. *Am J Obstet Gynecol*. 1996;175(1):1360-64.
26. Wallace J, Katz LM, Langendorf R, Buxbaum H. Electrocardiogram in toxemias of pregnancy. *Arch Int Med*. 1946;77:405-19.
27. Capeless EL, Clapp JF. Cardiovascular changes in early phase of pregnancy. *Am J Obstet Gynecol*. 1989;161(6):1449-53.
28. Carla A, Oppen V, Tweel IVD, Alsbach GPJ, Heethar RM, Bruinse HW. A longitudinal study of maternal haemodynamics during normal pregnancy. *Obstet & Gynecol*.1996;88(1):40-46.
29. 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.
30. Glenna C.L. Bett. Hormones and sex differences: changes in cardiac electrophysiology with pregnancy. *Clinical Science* 2016;130(10):747-759.
31. Zhao XQ, Wang CG, Song YX, Jiao H. The relationship of ECG and pregnancy outcome of older pregnant woman in late pregnancy. *Zhongguo Ying Yong Sheng Li Xue Za Zhi*. 2014 Jan;30(1):44-7.
32. Mizuno T, Tamakoshi K, Tanabe K. Anxiety during pregnancy and autonomic nervous system activity: A longitudinal observational and cross-sectional study. *J Psychosom Res*. 2017;99:105-111.
33. Shiozaki A, Matsuda Y, Satoh S, Saito S. Impact of fetal sex in pregnancy-induced hypertension and preeclampsia in Japan. *J Reprod Immunol*. 2011 May;89(2):133-9.