Original research article

Comparison of electrocardiographic parameters between the asymptomatic pregnant women above 32nd week of pregnancy and the non-pregnant women of comparative age

Suranjana Sur Mukherjee[1], Aditya Chowdhury[2]

1. Associate Professor, Department of Physiology, Nil Ratan Sircar Medical College, Kolkata, West Bengal.
2. Student of Final MBBS Part II at Nil Ratan Sircar Medical College , Kolkata, West Bengal.

Corresponding author: Suranjana Sur Mukherjee[1]

ABSTRACT

Introduction: We assessed the electrocardiographic parameters in 200 asymptomatic pregnant women beyond 32nd weeks of pregnancy and compared them with that of 200 non-pregnant healthy women of 18-35 years of age.

Methodology: The study populations were interviewed for general complaints (if any) and then electrocardiography (ECG) was done to determine the heart rate (HR), P wave duration, PR interval, corrected QT interval (QTc), axis and duration of QRS complex, Cardiac axis, ST segment, axis of T wave. Those with hypertension, gestation diabetes mellitus, preeclampsia, eclampsia, cardiovascular/liver/thyroid/renal/immunological diseases were excluded.

Results: The variables which have been found to be significantly deviated (i.e. $p<0.05$) in the study group were : Increase in HR (<0.001), decrease in duration of P wave ($p<0.001$), shortening of PR interval ($p<0.001$), increase in corrected QT interval (QTc) ($p=0.04$), degree of QRS wave deviation to the left ($p<0.001$), degree of T wave deviation to the left ($p<0.001$), and deviation of cardiac axis cardiac axis towards left (<0.001).

Conclusion: Electrocardiographic parameters of the study group were significantly different from that of the control group. As the study group had no symptoms of cardiac abnormality, the findings can be taken as a normative database of ECG changes in normal pregnancy in Indian population. Electrocardiography should be a routine test in antenatal check up to prevent cardiac arrhythmias and sudden maternal death although it should be interpreted with caution.

Key words: Gestation, Electrocardiography, ECG parameters.

INTRODUCTION:

Pregnancy is a high risk state where both the lives of mother and baby are crucial. Mother and child are always a single unit [1] . There are several changes in pregnancy which may be either physiological or pregnancy may unearth some hidden pathological conditions in mother's body. It will be easier for the obstetrician to take timely precaution and modify the intervention against some pregnancy induced events if data of simple baseline tests are available. In India, there is a paucity of data on the changes in the electrocardiographic parameters during pregnancy. In an earlier study by Briller et al, done during pregnancy till one year postpartum period, 22.2% maternal deaths were found to be due to cardiovascular causes [2]. Aslan and Atici concluded that the most common cause of maternal death was cardiomyopathy while arrhythmia was the 3rd most common cause and 25% of these deaths were possibly preventable [3].

Our study aimed to compare various electrocardiographic parameters between 200 asymptomatic pregnant women beyond 32 weeks of pregnancy and 200 non-pregnant unmarried healthy women of comparable age and also to prepare a normative database of Indian population for future reference.

AIM(S) AND OBJECTIVE(S):

1. To compare the Electrocardiographic parameters like Heart Rate, morphology of major ECG waves, duration of segments and intervals and the direction of cardiac vector between the asymptomatic pregnant women beyond 32nd week of pregnancy (case) and non-pregnant healthy unmarried women (control) of comparable age.
2. To determine the range of physiological variations of each parameter of ECG in advanced uncomplicated pregnancy.
3. To describe the changes in ECG parameters with respect to the potential changes in the cardiac dipoles during a cardiac cycle.

MATERIALS AND METHODS:

An analytical observational clinical type of study of cross-sectional design was done at the Department of Physiology and Department of Gynaecology and Obstetrics, NRSMCH from 1st July to 30th August, 2018. 200 pregnant women of above 32nd week of gestation between 18 to 35 years and 200 non-pregnant unmarried women of comparable age were taken as case and control group respectively.

A predesigned pretested schedule, Sphygmomanometer, Electrocardiograph (Model name-BPL CARDIART 6208 VIEW) and data from Indoor and Outdoor records were used for this research. All the study population were first interviewed and then a 12 lead electrocardiography was done for each of them in lying down position. Those with hypertension, gestation diabetes mellitus, preeclampsia, eclampsia, cardiovascular/liver/thyroid/renal/immunological diseases were excluded. Women more than 35 years and less than 18 years were excluded. Gestational age, Heart rate, P wave duration, PR interval, QTC interval, axis and duration of QRS complex, Cardiac axis, ST segment, axis of T wave were the variables in the study. The institutional ethical clearance was duly obtained before starting the procedure and all the participants voluntarily consented after being fully explained about the procedure. Statistical analysis was done by Microsoft Excel and SPSS Software.

RESULTS:

Data collected are analysed in three ways which are sub-grouped in A, B & C.

A. Comparison of the mean values of each parameter between the two groups:

PARAMETERS	**CASE (x+/-SD) (N=200)**	**CONTROL (x+/-SD) (N=200)**	**p VALUE**
Heart Rate (beats per min)	94.78 +/-13.02	83.08+/-11.42	<0.001
P wave duration (ms)	88.34 +/-7.17	97.82+/-9.87	<0.001
PR interval (ms)	123.24 +/-15.11	138.68+/-19.25	<0.001
PR Segment (ms)	34.9 +/- 9.98	40.86 +/-11.61	<0.001
Corrected QT interval (ms)	417.24+/-14.81	406.28+/-15.23	<0.001
QRS complex axis (deg)	48+/-21.72	66.32+/-18.77	<0.001
T wave axis (deg)	27.38+/-11.50	48.48+/-15.01	<0.001

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

Axis (deg)	45.22+/-21.79	63.64+/-16.83	<0.001
QRS complex duration (ms)	77.22+/-21.72	84.90+/-10.81	<0.001
P wave axis (deg)	48.06+/-28.26	56.04+/-14.59	<0.001
R wave amplitude in lead V5 (mV)	1279.64+/-348.	1538.8+/-544.78	<0.001
S wave amplitude in lead V1 (mV)	708.68+/-300.92	1041.04+/-569.57	<0.001
RV5+SV1 (mV)	1988.32+/-464.26	2579.84+/-965.78	<0.001

Abbreviations: ms = millisecond(s), mV = milliVolt(s), min = minute(s), deg = degree(s)

All the variables showed statistically significant (i.e. $p<0.05$) deviation in the study group.

In short, the mean heart rate and mean corrected QT interval were increased in case group. PR interval was significantly shortened in case group. The axes of QRS complex and T wave significantly deviated towards left. Also, the mean cardiac axis significantly deviated towards left but still within normal range.

B. Range of variation of each parameter:

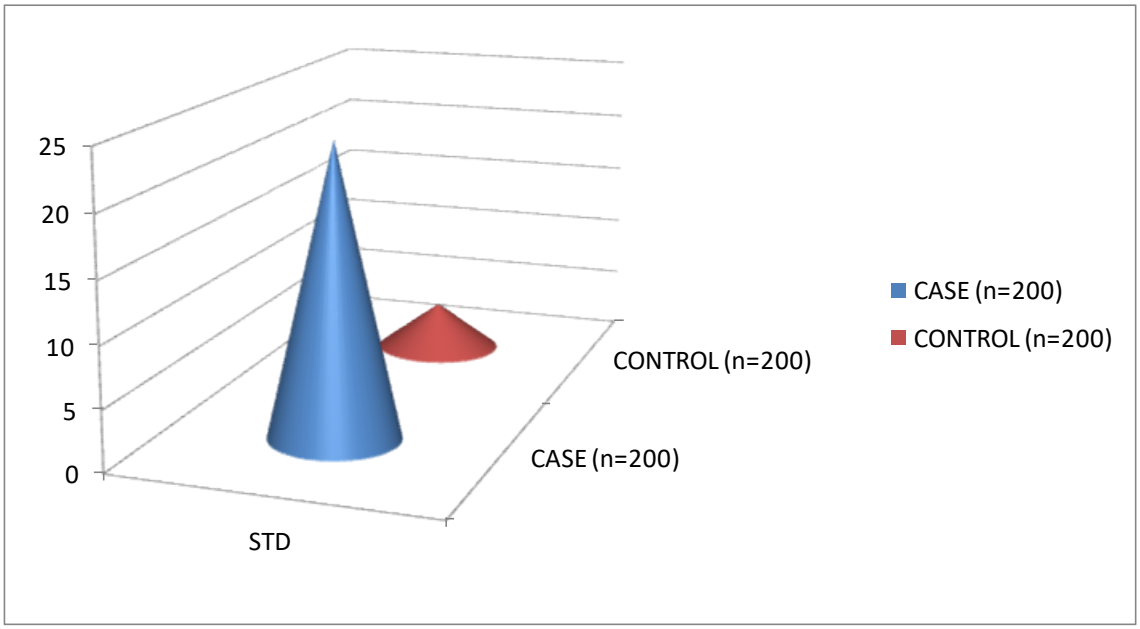
PARAMETER	**CASE**		**CONTROL**	
	min	**max**	**min**	**max**
Heart Rate (beats per min)	61	119	57	105
P wave duration (ms)	72	104	81	125
PR interval (ms)	87	153	103	184
PR segment (ms)	15	57	21	73
QRS wave duration (ms)	66	110	70	115
QTC (ms)	385	452	377	444
P wave axis (deg)	-32	210	0	79
QRS wave axis (deg)	1	108	26	97
T wave axis (deg)	-3	55	26	92
Amp of R wave in V5 (mV)	700	2232	813	2648
Amp of S wave in V1 (mV)	216	1491	315	2316
Cardiac Vector (deg)	7	101	27	90
Sum of R amplitude at V5 and S amplitude at V1 (mV)	1136	3133	1284 mV	4966 mV

Abbreviations: min = minimum, max = maximum

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

C. Other important findings:

1) Figure 1: Showing ST segment changes in two groups

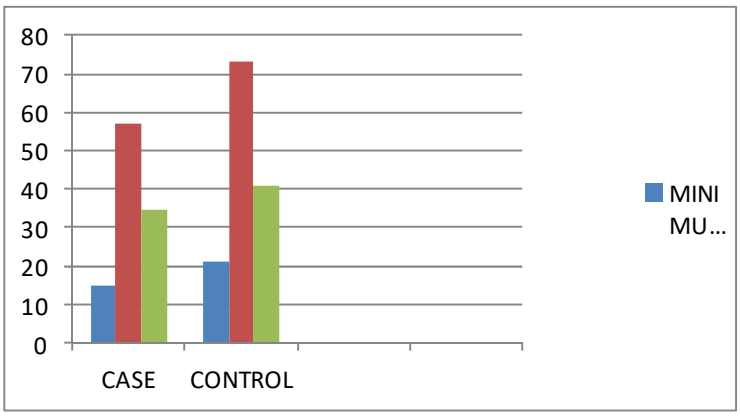


ST segment was depressed (STD) in 24 subjects out of 200 in the Case group and ST elevation (STE) was seen in only 4 out of 200 whereas STD in only 4 cases out of 200 and STE was seen in 12 control subjects out of 200. Chi-square value is 0.004 when compared between two groups with respect to ST depression.

2) A-V Nodal delay (in ms) (represented by PR segment duration)

	CASE	CONTROL
Mean	34.9	40.86
Min	15	21
Max	57	73
SD	9.98	11.61

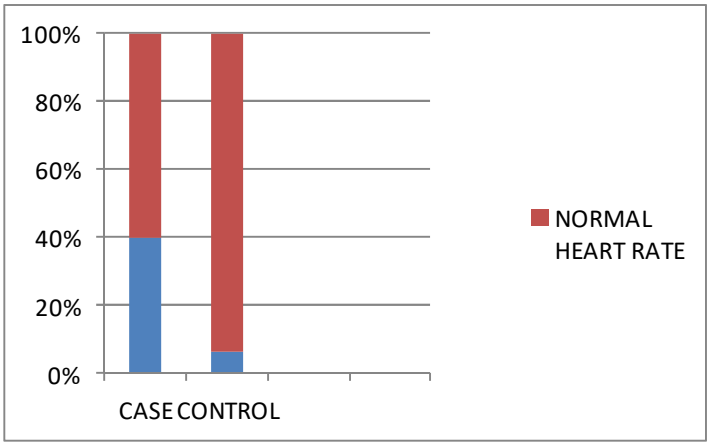
Figure 2: Showing pattern of AVN delay



AV Nodal delay is less in the study group indicating that pregnant women are more prone to arrhythmia.

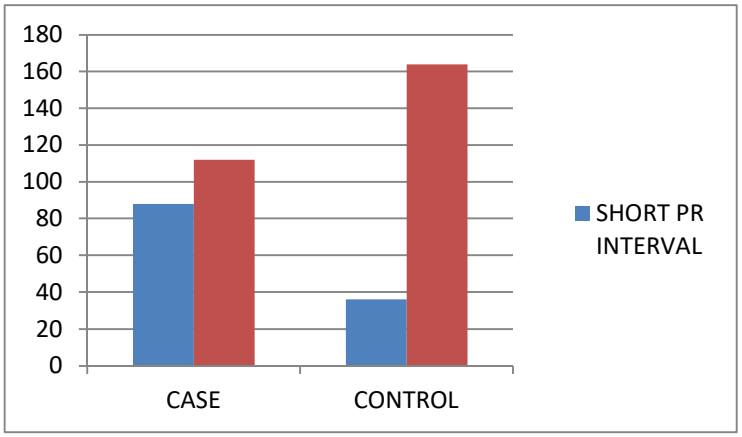
www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

Figure 3: Bar diagram showing the prevalence of tachycardia in the study group.



Tachycardia is seen around 40% of study/case population whereas in control group it is seen in <10% of the population.

Figure 4: Bar diagram comparing the duration of PR interval between the two groups



Shortening of PR interval, which is the basis of tachycardia in pregnancy, is seen in almost double number of women in the study group than in the control group.

DISCUSSION:

Brijler et all (2) said that more than one-fifth of maternal deaths in Illinois over a period of nine years were attributed to cardiovascular disease such as Cardiomyopathy(27.9%) followed by stroke (22.9%), hypertensive disorders (12.9%), arrhythmias (10.7%) and coronary disease (9.3%). Nearly 75% of cardiac deaths were related to pregnancy as compared to 35.3% of non-cardiac deaths. More than one fourth (28.1%) were potentially preventable by proper antenatal care and intensive postpartum monitoring.

Aslan MM & Atici A (3) studied electrocardiographic parameters on 280 pregnant women and risk of arrhythmia in advanced-age pregnancy was evaluated by P-wave duration, QT interval, T Peak-to-end interval (Tp-e) and Tp-e/QT ratio. They found positive correlation of repolarization parameters like increased dispersion (i.e the difference between maximal and minimal duration of the wave) of QTc and P wave with maternal age indicating routine electrocardiography in pregnancy especially in advanced age.

Shotan A et all (4) assessed relation between symptoms and cardiac arrhythmias in 110 consecutive pregnant patients without evidence of heart disease but who were referred for evaluation of palpitations, dizziness and syncope (study group) and 52 consecutive patients referred for evaluation of asymptomatic precordial murmur (control group). Both

groups had a high incidence of arrhythmias on Holter monitoring with atrial premature complexes (APCs) in 56% of the study and 58% in the control group and isolated ventricular premature beats (VPCs) in 59% and 50% respectively. There was no correlation between the incidence of both VPCs and APCs and the symptoms ; 10% of the symptomatic episodes were accompanied by arrhythmias.

Tanindi A et al (5) studied P-wave duration to determine atrial arrhythmia and QT interval, Tp-e (peak to end) interval and Tp-e/QT ratio as indices of ventricular repolarisation in pregnancy with respect to trimesters and found that P-wave duration was prolonged in the second trimester which resumed plateau thereafter and maximum QTc, Tp-e and Tp-e/QT all increased in late pregnancy albeit within the normal range.

Elming H, Holm E et al (6) studied the prognostic value of QT interval and QT interval dispersion in all-cause and cardiac mortality and morbidity in Danish citizens and concluded that prolongation of the QT interval and QT interval dispersion independently affected the prognosis of cardiovascular mortality and cardiac fatal and non-fatal morbidity in a general population.

Malik M and Batchvarov VN (7) measured the QT dispersion in health and disease and found that QT dispersion varied widely in normal subjects but increase in dispersion of QT had more prognostic value in cardiac patients. They advised new methods of assessment and quantification of repolarization such as T loop descriptors, T wave morphology and wavefront direction descriptors.

In our study, we found significant increase in the HR in the study group [Ref: Table A, Fig:3] which can be explained by the decrease in vagal baroreflex as well as decrease in parasympathetic tone (8). Increase in HR compensated for the fall in stroke volume resulting from cardiac compression (9). Similar findings are seen in other studies too (8, 10, 11, 12). Increase in HR is due to decrease in PR interval during pregnancy which can be explained by shortening of AV conduction (13) as seen in the case group [Ref: Table C(2), Fig:2]. The decrease in the PR segment in the study group indicates that AVN is less refractory, hence pregnant women are more prone to arrhythmia compared to non-pregnant women which is corroborated by other studies.

The axis of the QRS wave is significantly deviated towards the left in the study group though within normal range - the mean values are 48+/-21.72 deg and 66.32+/-18.77 deg in the case and control group respectively [Ref: Table A]; the range of deviation being 1 degree to 108 degree in the study group [Ref: Table: B] and this can be explained by the raising of the diaphragm (14). The direction of Cardiac Vector was measured in Lead II and closely matched the axis of QRS wave as expected and it was grossly deviated towards left; values were 45.22+/-21.79 deg in the case and 63.64+/-16.83 deg in the control group [Ref: Table A].

The axis of T wave is significantly deviated towards left indicating that ventricular repolarisation and Slow Ejection Phase (SEP) were affected the most. Apart from this, the duration of both atrial depolarisation (P wave) and ventricular depolarisation (QRS) decreased significantly in the study group [Ref: Table A]. Changes in QT dispersion and P wave is corroborated by Aslan MM & Atici A(3).

We have seen more number of ST segment depression in the study group compared to elevation [Ref: Fig:1]. T-wave inversion in the Lead III and V2 is attributable to the increased overload on the heart due to temporary increase in blood volume during pregnancy which may cause temporary ischaemia (15).

QTc (corrected QT interval) increased in the study group having the mean value of 417.24+/-14.81ms compared to 406.28+/-15.23 ms in the control group which may be due to increase in the HR. This could be linked to changes in the ventricular depolarisation and repolarisation pattern during pregnancy. This must be considered as a complex consequence of the changes in the various regulatory mechanisms occurring during pregnancy (16). These changes were attributed to the changed spatial arrangements of chest organs during pregnancy (17). The changes in the

electrical properties of myocardium is due to the changed sympathetic and hormonal modulation (by Epinephrine, Progesterone etc) during pregnancy. Lechmanova M et al (16) and Sharad Kole et al (18) opined that prolonged QT and QTc intervals should be interpreted simply as 'an unspecific sign of the changed course of repolarisation'.

The change in ventricular repolarisation can be explained by the change in sum of the amplitudes of R wave in V5 and S wave in V1 (RV5+SV1) which represents the left ventricular (LV) muscle mass (19). In 3rd trimester of pregnancy, there is hyperdynamic circulation along with volume overload. Our study showed a decrease in total potential at (RV5+SV1) in pregnant women beyond 32 weeks of pregnancy; mean value being 1988.32=/-464.26 mV compared to 2579.84+/-965.78 mV in the control [Ref: Table A] which indicates that there is a decrease in LV muscle mass probably due to thinning by volume overload which eventually leads to the dilatation of the ventricles making it prone to failure.

CONCLUSION:

Pregnancy is the nature's stress test and cardio-vascular disease in pregnancy is the leading cause of death in North America (1). Electrocardiography should be a routine test in antenatal check up to prevent potential cardiac arrhythmias and sudden maternal death. We observed that the changes in ECG parameters occurred over a wide range; hence the interpretation of electrocardiography in pregnancy should be done with caution. The study variables, which showed statistically significant changes, if found to be beyond the two extremes, should be investigated further to rule out pathology.

PROS AND CONS OF OUR STUDY:

Pros are that we get the range of normal variation of each parameter of the Electrocardiography in advanced pregnancy. Cons are that the study is done in a small group of subjects and follow up could not be done during the post-partum period to determine when those changed parameters reverted to normal in postpartum period.

ACKNOWLEDGEMENT:

We would like to thank Prof. Dr. Picklu Chowdhury, HOD, Department of Gynaecology and Obstetrics, Rampurhat Medical College for the concept and continuous guidance. Also we are thankful to Prof. Dr. Ananda Kumar Mukhopadhyay, HOD, Department of Physiology, Nil Ratan Sircar Medical College and Hospital, Kolkata for the logistic support.

REFERENCES:

[1] K. Park. Preventive medicine in obstetrics, paeditrics and geriatrics. In: Park's Textbook of Preventive and Social Medicine 24th Ed. M/s Banarsidas Bhanot publishers, India 2017:555.

[2] Briller J, Koch AR, Geller SE. Maternal Cardiovascular Mortality in Illinois, 2002-2011. Obstet Gynecol. 2017;129(5):819–826. doi:10.1097/AOG.0000000000001981.

[3] Aslan MM1, Atici A2.Assessment of increased risk of arrhythmia in advanced age pregnancies. Pak J Med Sci. 2018 May-Jun;34(3):687-690. doi: 10.12669/pjms.343.14313.

[4] Shotan A, Ostrzega E, Mehra A, Johnson JV, Elkayam U. Incidence of arrhythmias in normal pregnancy and relation to palpitations, dizziness, and syncope. Am J Cardiol. 1997;79(8):1061–1064.

[5] Tanindi A, Akgun N, Pabuccu EG, Gursoy AY, Yuce E, Tore HF, et al. Electrocardiographic P-Wave Duration, QT Interval, T Peak to End Interval and Tp-e/QT Ratio in Pregnancy with Respect to Trimesters. Ann Noninvasive Electrocardiol. 2016;21(2):169–174. doi:10.1111/anec.12285.

[6] Elming H, Holm E, Jun L, Torp-Pedersen C, Kober L, Kircshoff M, et al. The prognostic

value of the QT interval and QT interval dispersion in all-cause and cardiac mortality and morbidity in a population of Danish citizens. Eur Heart J. 1998;19:1391–1400.

[7] Malik M, Batchvarov VN. Measurement, interpretation and clinical potential of QT dispersion. J Am Coll Cardiol. 2000;36:1749–1766.

[8] Voss A, Malberg H, Schumann A, Wessel N, Walther T, Stepan H, Faber R. Baroreflex sensitivity, heart rate and blood pressure variability in normal pregnancy. Am J Hypertens. 2000;13(11):1218-25.

[9] Julian DG, Wenger NK. Heart disease and heart surgery during pregnancy. In: women and heart disease. United Kingdom: Martin Dunitz; 2000s.

[10] Katz R, Karliner JS, Resnik R. Effects of a natural volume overload state (pregnancy) on left ventricular performance in normal human subjects. Circulation. 1978;58:434-41.

[11] Burwell CS. Circulatory adjustments to pregnancy. Bull Johns Hopkins Hosp. 1954;95(3):115-29.

[12] Stein PK, Hagley MT, Cole PL, Domitrovicn PP, Kieigner RE, Rottman JN. Changes in 24 hours heart rate variability during normal pregnancy. Am J ObstetGynecol. 1999;180(4):978-85.

[13] Sharad Kole, Jnaneshwara Shenoy, Shivakumar J, Suvarna P, Prasannakumar J. Variations in electrical activity of the heart as the pregnancy progresses. National Journal of Physiology, Pharmacy & Pharmacology. 2014;4(3):187-90.

[14] Singh AD, Devi L, Singh L, Devi R, Singh J. Electrocardiographic findings at term, labour and immediate postpartum. J Obstet & Gynecol of India. 1986;36:316-9.

[15] Misra J, Dutta B, Ganguly D. Electrocardiographic study in pregnant women in normal and toxemia of pregnancy. J ObstetGynecol India. 1986;36:635-38.

[16] Lechmanova M, Kittnar O, Mleck M, Slavicek J, DohnalovaA, Havranek S, et al. QT Dispersion and T-Loop Morphology in Late Pregnancy and After Delivery. Physiol Res. 2002;51:121-9.

[17] Carruth JE, Mirvis SB, Brogan DR, Wenger NK. The electrocardiography in normal pregnancy. Am Heart J. 1981;102:1075-8.

[18] Sharad Kole, Jnaneshwara Shenoy, Shivakumar J, Suvarna P, Prasannakumar J. Variations in electrical activity of the heart as the pregnancy progresses. National Journal of Physiology, Pharmacy & Pharmacology. 2014;4(3):187-90.

[19] Debasis P. Electrocardiogram In: Principles of Physiology. Jaypee Brothers Medical Publishers (P) Ltd. N.Delhi 2015: 327-8.