

Pattern Of Electrocardiography Findings And Correlates Among Pregnant Women At The Three Trimesters Of Pregnancy In Southwest Nigeria

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Abstract- Background: The hemodynamic and cardiovascular status of women during pregnancy changes in a pattern that completely differs from the non-pregnant state which may aggravate pre-existing asymptomatic heart disease or cause new-onset heart disease in the susceptible heart. This study assessed the electrocardiography patterns of women in each trimester of pregnancy.

Method: The study adopted a cross-sectional design carried out on ninety-six pregnant and one hundred nonpregnant women between the ages of 18 and 45 years. A self-administered questionnaire was used to obtain general data with informed consent. Each subject had a complete clinical cardiac status assessment. A resting 12-lead electrocardiogram was obtained in accordance with the recommendations of the American Heart Association specifications. Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20.0.

Results: This study demonstrated a 39% progressive increase in cardiac output. The individual contribution of stroke volume and heart rate was 23% and 16% respectively. Up to 18% had diastolic blood pressures lower than 60mmHg with significantly lower mean arterial pressure recorded by the end of the first trimester. All the participants had a background sinus rhythm but with a variable abnormality of impulse generation and conduction. The mean frontal plane QRS axis showed a leftward trend among pregnant subjects (35.4 Vs. 48.4°, $p = 0.001$).

Conclusion: The role of Electrocardiography in pregnant women is not limited to establishing cardiac dysfunctions or variations but as a cogent tool in risk assessment of preventable cardiovascular sequelae during pregnancy.

Index Terms- electrocardiography, pregnant women, ECG during pregnancy, cardiac output.

I. INTRODUCTION

The Electrocardiogram (ECG) is the Voltage–Time tracing of the electrical activity of the heart viewed from within the heart

(His-Bundle ECG) and or the surface (Scalar ECG). The ECG is usually affected by several physiologic factors among which is age, sex, height, weight, race, nutritional status and chest circumference.¹⁻³

Pregnancy is a physiological process associated with various hormonal, cellular and tissue adaptations to cope with the increased metabolic demands of the mother and the growing foetus. There are such great changes in the hemodynamic and cardiovascular status of the mother that all measurable cardiovascular variables change in a pattern that completely differs from the general population and in the non-pregnant state.³ These hemodynamics and cardiovascular changes may mimic heart disease in the apparently normal heart and may as well aggravate pre-existing asymptomatic heart disease or cause new-onset heart disease in the susceptible heart. Various reports have shown that one of the commonest causes of pregnancy-related mortality is cardiovascular disease. However, diagnostic difficulties may occur due to haemodynamic and cardiovascular changes associated with pregnancy.⁴

There is evidence from previous studies to suggest that some electrocardiographic parameters are, indeed, altered by the pregnant state⁵⁻⁸ and the known cardiovascular adaptations to pregnancy represent potential mechanisms by which these alterations may arise.

Carr and palmer⁸ reported a leftward axis deviation as a normal characteristic in pregnant Caucasian women in the third trimester while Hollander and Crawford⁴ reported a mean leftward axis deviation of 15° with some individual subjects showing left axis deviation as much as 28°. This deviation was attributed to the transverse displacement of the heart and also to its clockwise rotation around its long axis due to the effect of the gravid uterus. Zatuchi⁹, Carruth *et al.*⁸ and Wenger¹⁰ confirmed the leftward deviation of QRS axis, especially in the third trimester of pregnancy. In addition, increase in heart rate was also reported as accompanying pregnancy. Iwobi *et al.*¹¹ in a study involving 41 Nigerian pregnant women reported leftward QRS axis deviation with no significant changes in the heart rate. However, Akinwusi

et al¹² found a normal frontal-plane QRS axis, normal PR interval, significantly rare normal Negroid pattern ST elevation, significant left ventricular hypertrophy (LVH) based on Araoye RI >12 mm and a rarity of all forms of arrhythmias in a study of 69 Nigerian women.¹²

Although Electrocardiographic changes in normal pregnancy have been clearly demonstrated in Caucasians, similar studies in Negros are relatively scarce. However, there is a paucity of data on these changes in the black population especially in this study area, despite several data showing higher prevalence of pregnancy related cardiovascular death in blacks.⁵

II. METHODOLOGY

Study area

The study was carried out at the Ladoke Akintola University of Technology (LAUTECH) Teaching Hospital (LTH) Ogbomoso, located in Ogbomoso town, Oyo state Nigeria. It is a tertiary institution that serves as a referral Centre for the entire community as well as surrounding cities and states. Ogbomoso lies on the plateau of Yorubaland (elevation 1,200 feet) in an area of savannah and farmland at the intersection of roads from Oyo, Ilorin, Osogbo and Ikoyi which bound the city in the south, north southeast and southwest respectively. Ogbomoso is estimated to have a population of about 1.2 Million people majority of whom are of the Yoruba ethnic group. Ogbomoso is the second largest city in Oyo state after the capital city of Ibadan.^{13,14}

Study design

The study was a cross-sectional study

Sample size

The sample size was determined using the formula¹⁵

$$n = \frac{r+1}{r} \frac{(\bar{p})(1-\bar{p})(Z_{\beta} + Z_{\alpha/2})^2}{(p_1 - p_2)^2}$$

r = Ratio of subjects to controls

Z_{β} = Represents the desired power (typically 0.84 for 80% power).

$Z_{\alpha/2}$ = Represents the desired level of statistical significance (typically 1.96).

P_1 = Proportion of various abnormalities on ECG in the subjects=0.102

P_2 = Proportion of various abnormalities on ECG in the control=0.000

$$\bar{P} = \text{Average proportion} = \frac{P_1 + P_2}{2}$$

$$n = \frac{2(0.051)(0.949)(0.84+1.96)^2}{0.010404}$$

=72.943. Therefore n=146

The proportion of various abnormalities on ECG from a previous study¹² in Nigeria in the subjects and control was 10.2% and 0.0% representing p_1 and p_2 respectively Therefore 96 cases and 100 (hundred) controls were recruited.

III. STUDY POPULATION

Ninety-six consecutive pregnant women in all trimesters of pregnancy (stratified as follows: 24 pregnant women in first trimester, 39 in second trimester and 33 in the third trimester) and one hundred non pregnant women between the ages 18 and 45 years.

Sampling Technique

Consecutive pregnant women in all trimesters who satisfied the inclusion and the exclusion criteria after administration of questionnaire were serially recruited for the study until the sample size was met.

The controls were age-matched female subjects who were not pregnant. The control participants were selected from the members of staff and undergraduates of various departments of the Ladoke Akintola University of Technology and the members of staff of LAUTECH Teaching Hospital (LTH). All selected controls were free of cardiac disease.

Verbal and written informed consent were obtained after careful explanation of the study to them and the study questionnaire was administered to each participant to obtain biodata and other relevant information.

Procedure

Each subject had a complete clinical cardiac status assessment. A resting 12-lead electrocardiogram was obtained in accordance with the recommendations of American Heart Association specification¹⁶ using the Schiller machine CS-200 model. Recording was commenced after participants had rested for at least 15 minutes and ECG tracing ran at a speed of 25mm/second. For the purpose of this study ECG tracing were evaluated using the abnormalities below:

- (a) Sinus bradycardia = ECG heart rate < 60 beats per min in sinus rhythm
- (b) Sinus tachycardia = ECG heart rate > 100 beats per min in sinus rhythm
- (c) Left ventricular hypertrophy(LVH)
 - (i) $SV_1 + RV_5 \geq 35mV$ (Sokolow – Lyon criteria)⁵
 - (ii) $SV_1 + RV_6 \geq 35mV$
 - (iii) $SV_2 + RV_6 > 3.5mV$ (Araoye's criteria)³
 - (iv) $SV_2 + RV_5 > 3.5mV$
 - (v) Flat or inverted Twaves (strain pattern) in V_5 or V_6 ,
 - (vi) R_1 amplitude > 1.2mV.
 - (vii) $RaVL + SV_3 \geq 20$ Cornell's criteria
- (d) Right ventricular hypertrophy(RVH) = $RV_1/SV_1 \geq 1$ or $RV_1 \geq 7mm$
- (e) Axis deviation
 - (i) Normal axis = QRS axis between 0 to +90°
 - (ii) Left axis deviation = QRS axis less than 0 to -90° (depending on the age of the subject)
 - (iii) Right axis deviation = QRS axis > +90°
- (f) Atrioventricular(A – V) block
 - (i) 1st degree = P – R interval > 0.20 sec¹⁷
 - (ii) 2nd degree (morbitz type 1 or type 2) Morbitz type 1 {characterized by progressive prolongation of the PR interval before a nonconducted P wave} Morbitz type 2 {characterized by a nonconducted P wave without preceding PR prolongation}

- (iii) 3rd degree/Complete A – V block (in which none of the Orthograde impulse reaches the ventricles)
- (g) QT interval corrected for heart rate using the Bazett’s formular, $QTc = \frac{QT}{\sqrt{R-R}}$ Prolonged when = QTc interval > 0.47sec¹⁷
- (h) ST segment, T wave repolarization changes (a) J point > 1mm above (ST segment elevation) or below the base line (ST segment depression) (b) Inverted T-waves.¹⁷
- (i) Left atrial enlargement by various indices: Morris index (Biphasic P wave in V1 with terminal force $\geq 1mm$), Macruz index (Ratio of P duration to PR segment >1:1.6 (Macruz index), Duration between peaks of p wave notch >0.04s or Maximal p wave duration >0.11s¹⁷
- (j) OTHERS – any other notable ECG abnormalities (Right atrial enlargement-RAE, arrhythmias).

Data Analysis

Data was analyzed using the Statistical Package for Social Sciences (SPSS) version 20.0 for windows (SPSS Inc; Chic; Ill). Continuous data were presented as mean \pm standard deviation and categorical variables were presented as percentages. The chi-square was used to compare the relationship between qualitative variable, T-test and analysis of variance (ANOVA) to assess for significant associations between group means in quantitative variables. Statistical significance was taken as $p < 0.05$, and the confidence level of 95%.

Ethical clearance

Approval by the Ethical Review Committee of Ladoke Akintola University Teaching Hospital (LTH), Ogbomosho was obtained prior to commencement of the study.

IV. RESULTS

The study included 196 subjects consisting of 96 pregnant women and 100 controls. The mean gestational age of subjects in the first trimester was 10 ± 2 weeks, second trimester was 24 ± 3 weeks and third trimester was 34 ± 3 weeks. The mean age of both pregnant women and the controls were similar (28.1 ± 5.1 vs. 26.4 ± 5.3 years, $p=0.10$). Thirty-six percent ($n= 35$) of the pregnant women were primigravidae. Table I.

The weight of the pregnant women was significantly higher than that of the control (64.9 ± 11.8 vs. 59.7 ± 12.0 kg, $p=0.01$) whereas the height was higher in control than the pregnant women (1.58 ± 0.06 vs. 1.61 ± 0.06 m, $p= 0.17$). The body mass index was significantly higher in the pregnant women than in the controls (26.0 ± 4.1 vs. 23.2 ± 4.4 , $p < 0.001$).

Table I: Socio-demographic and Anthropometric Parameters of subjects and Controls

VARIABLE	SUBJECTS Mean \pm SD (n = 96)	CONTROLS Mean \pm SD (n = 100)	P VALUE
Age (years)	28.1 \pm 5.1	26.4 \pm 5.3	0.10
Weight (kg)	64.9 \pm 11.8	59.7 \pm 12.0	0.010*
Height (m)	1.58 \pm 0.06	1.61 \pm 0.06	0.003*
BMI (g/m ²)	26.0 \pm 4.1	23.2 \pm 4.4	< 0.001*

BSA (m²)	1.68 \pm 0.17	1.63 \pm 0.17	0.093
Average income (Naira)	14,940	\pm 13,401 \pm 8,248	0.566
Parity (N)			
0	35 (36.5%)	84 (84.0%)	
1	29 (20.2%)	6 (6%)	
2	14 (14.6%)	2 (2%)	
3	10 (10.4%)	5 (5%)	
4	7 (7.3%)	2 (2%)	
5	1 (1.0%)	1 (1%)	

Key: **SD**= standard deviation, **BMI**= Body mass index, **SBP**= systolic blood pressure, **DBP**= diastolic blood pressure, **BSA**= Body surface area, **kg**= kilograms, **m**= metres * statistically significant

The mean systolic and mean diastolic blood pressures were lower in pregnant women compared with the controls. However, up to 18% of pregnant women had diastolic blood pressures lower than 60mmHg as against 3% of the controls. (OR= 0.240, CI = 0.067-0.852, $p = 0.018$). None of the pregnant subjects and controls had systolic BP lower than 90mmHg. There was a significantly lower mean arterial pressure (MAP) in pregnancy compared with the controls (83.7 ± 9.9 vs. 90.0 ± 8.8 mmHg, $p < 0.001$). This was already noticeable by the end of first trimester with a further decline to the nadir in the second trimester and a subsequent rise towards term. (Table II)

Table II: Haemodynamic variables of subjects and control

Variables	Subjects (N= 96) Mean \pm SD	Controls (N=100) Mean \pm SD	P-value
Heart Rate (bpm)	87.9 \pm 10.6	75.8 \pm 9.8	<0.001*
SBP (mmHg)	114.2 \pm 14.0	119.1 \pm 13.6	0.047*
DBP (mmHg)	68.5 \pm 9.2	75.0 \pm 10.0	<0.001*
PP (mmHg)	45.7 \pm 10.1	44.1 \pm 15.0	0.444
MAP (mmHg)	83.7 \pm 9.9	90.0 \pm 8.8	0.001*
SV (ml)	68.5 \pm 13.7	57.3 \pm 12.8	<0.001*
CO (L/min)	6.03 \pm 1.30	4.34 \pm 1.11	<0.001*
CI	3.57 \pm 0.68	2.48 \pm 0.67	<0.001*
SVR (dyn x sec/cm⁵)	1106.5 \pm 267.5	1462.7 \pm 304.7	<0.001*

Key: **SD**= standard deviation, **SBP**= systolic blood pressure, **DBP**= diastolic blood pressure, **PP**= pulse pressure, **MAP**= mean arterial pressure, **SV**= stroke volume, **CO**= cardiac output, **CI**= cardiac index, **SVR**= systemic vascular resistance, * statistical significance between all pregnant women and all non-pregnant women.

There was relative higher mean heart rate in pregnancy which was progressive from first trimester through to third trimester. The cardiac output was significantly higher in pregnancy progressively (6.03 ± 1.3 vs, 4.34 ± 1.1 L/min, $p < 0.001$). The maximum increase of heart rate and stroke volume was observed in the third trimester with about 38.7% increase in

cardiac output contributed by 16% increase in heart rate and 20% increase in stroke volume.

The mean systemic vascular resistance was significantly lower in pregnancy which represented a progressive decline from the first through third trimester as shown in Table III.

Table III: ANOVA Table Comparing the Haemodynamic variables among subjects in each trimester and controls

Variables	T1 (N=24)	T2 (N= 39)	T3 (N= 33)	Controls(C)		Post Hoc Test [§]
	Mean \pm SD	Mean \pm SD	Mean \pm SD	(N=100)	p-value	
				Mean \pm SD		
Heart Rate (bpm)	85.3 \pm 10.4	88.7 \pm 11.5	88.7 \pm 10.2	75.8 \pm 9.8	<0.001*	T1>C T2>C T3>C
SBP (mmHg)	115.1 \pm 16.1	110.9 \pm 12.5	117.1 \pm 13.8	119.1 \pm 13.6	0.048*	T2<C T2<T3
DBP (mmHg)	68.1 \pm 9.0	66.0 \pm 8.5	71.7 \pm 9.5	75.0 \pm 10.0	<0.001*	T1 < C T2 < C T2 < T3
PP (mmHg)	47.1 \pm 12.1	44.9 \pm 10.5	45.4 \pm 8.6	44.1 \pm 15.0	0.893	-
MAP (mmHg)	83.7 \pm 10.4	81.0 \pm 8.7	86.8 \pm 10.3	90.0 \pm 8.8	<0.001*	T2 < C
SV (ml)	62.5 \pm 10.9	66.8 \pm 9.5	72.6 \pm 15.9	57.3 \pm 12.8	<0.001*	T2 > C T3 > C T3 > T1
CO (L/min)	5.32 \pm 1.05	5.95 \pm 1.27	6.42 \pm 1.32	4.34 \pm 1.11	<0.001*	T2 > C T3 > C T3 > T1
CI	3.23 \pm 0.68	3.64 \pm 0.69	3.65 \pm 0.65	2.48 \pm 0.67	<0.001*	T2 > C T3 > C T3 > T1
SVR (dyn x sec/cm⁵)	1229.4 \pm 320.3	1095.6 \pm 259.4	1067.5 \pm 238.9	1462.7 \pm 304.7	<0.001*	T2 > C T3 > C

Key: **SD**= standard deviation, **SBP**= systolic blood pressure, **DBP**= diastolic blood pressure, **MAP**= mean arterial pressure, **SV**= stroke volume, **CO**= cardiac output, **CI**= cardiac index, **SVR**= systemic vascular resistance, **T1**= first trimester, **T2**= second trimester, **T3**= third trimester. * Statistical significance among pregnant women in each trimester and controls. [§] Post hoc test with Bonferroni's correction

The pregnant women showed significantly higher mean heart rate than non-pregnant women (87.3 ± 12.2 vs. 75.1 ± 11.4 bpm, $p = <0.001$). Sinus tachycardia was found to be significantly more in pregnant women than in non-pregnant women (11% vs. 0%, $p=0.008$), whereas bradycardia was rare and similar in both groups (2% vs. 0%, $p=0.117$) (Table IV).

All the participants had a background sinus rhythm. Atrial and ventricular premature complexes were rare in pregnancy. Left

bundle branch blocks occurred significantly more in pregnant than non-pregnant women (22% vs. 1%, $p < 0.001$). Non-specific intraventricular conduction block was found in 57 (59%) of the pregnant group in the form of Rsr', mostly in lead III, against 14 (14%) in the control group. Similarly, Rsr' pattern was found in lead avF in 36 (38%) of the pregnant group, against 5 (5%) of the control (OR = 6.324, CI = 2.299–17.4393, $p = 0.0105$). Table IV

Table IV: Electrocardiographic parameters of Subjects and Controls

	Subjects N = 96 (%)	Controls N= 100 (%)	OR (95% Confidence interval)	p-value
Number	96 (100%)	100 (100%)		
Bradycardia	1 (1%)	3 (3%)	0.192 (0.020-1.889)	0.117

Tachycardia	11 (11%)	1 (1%)	0.597 (0.524-0.681)	0.008*
APC	1 (1%)	1 (1%)	0.596 (0.037-9.708)	0.713
VPC	1 (1%)	0	1.606 (1.423-1.813)	0.437
LBBB	22 (23%)	1 (1%)	7.0 (2.223-12.032)	<0.001*
Rsr' in lead III	57 (59%)	14 (14%)	4.853 (2.301-10.236)	<0.001*
Rsr' in lead aVF	36 (38%)	5 (5%)	6.324 (2.299-17.4393)	<0.001*
Prolonged PRI	0	2 (2%)	0.367 (0.299-0.450)	0.066
Shortened PRI	0	0	-	-
Prolonged QTc	11 (11%)	4 (4%)	0.524 (0.161-1.705)	0.276
Shortened QTc	2 (2%)	0	0.620 (0.549-0.701)	0.270

Key: **SD**= standard deviation, **APC**= atrial premature complex, **VPC**= ventricular premature complex **LBBB**= left bundle branch block, **PRI**= PR interval, **QTc**= corrected QT interval,* Statistical significance between all Subjects and controls.

The mean frontal plane QRS axis was within the limit of normal in all study participants but there was a leftward trend in the pregnant women as opposed to the non-pregnant control (35.4 Vs. 48.4°, $p = 0.001$). There was also a progressive leftward trend from the first to the third trimester as shown in Table V.

Table V: ANOVA table comparing the Electrocardiographic parameters among subjects in each trimester and Controls

Variables	T1 (N=24)	T2 (N= 39)	T3 (N= 33)	Controls (C) (N=100)		Post Hoc Test [§]
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	p-value	
ECG HR (bpm)	85.5 ± 10.5	86.7 ± 11.9	89.4 ± 14.0	75.1 ± 11.4	<0.001*	T1 > C T2 > C T3 > C
QRS axis (°)	38.3 ± 24.5	36.6 ± 22.0	30.6 ± 26.3	48.4 ± 18.7	<0.001*	T3 < C
p-wave axis (°)	40.5 ± 9.0	38.7 ± 19.1	34.0 ± 15.7	47.4 ± 19.7	0.028*	T3 < C
T-wave axis (°)	26.7 ± 15.7	31.7 ± 12.3	28.1 ± 11.7	30.4 ± 11.5	0.938	-
PR interval (ms)	155.7 ± 22.7	150.3 ± 22.4	149.0 ± 22.4	163.9 ± 25.6	0.020*	T3 < C
QTc (ms)	399.4 ± 80.7	401.5 ± 25.5	398.1 ± 62.7	396.0 ± 30.1	0.018*	T3 < C

Key: **SD**= standard deviation, **HR**= heart rate, **QTc** = corrected QT interval * statistically significant. T1= first trimester, T2= second trimester, T3= third trimester. * Statistical significance among pregnant women in each trimester and controls. § Post hoc test with Bonferroni's correction

V. DISCUSSION

There are fewer women in the first trimester due to delayed antenatal care visits common in this environment. The mean age was similar between the pregnant women and controls. The mean weight and body mass index (BMI) of pregnant women were significantly higher than those of the controls. The decrease in the systolic, diastolic and mean arterial pressures observed in the pregnant women was due to a decline in the systemic vascular resistance associated with pregnancy as shown in this study.

Compared with the age-matched control, this study demonstrated a 39% increase in cardiac output which represented a progressive increase from the first trimester through to the third trimester. The individual contribution of stroke volume and heart rate was 23% and 16% respectively. This is similar to studies by Desai *et al.*¹⁸ and Mabie *et al.*¹⁹

In the meta-analysis of cross-sectional studies, Van oppen *et al.*²⁰ showed a trend to a lower cardiac output in the third trimester compared with the second, the authors observed large ranges in cardiac output among the different studies that did not allow for any firm conclusions. However, in the 6 longitudinal studies evaluated, Van Oppen found that cardiac output between the second and third trimesters plateaued, decreased, or increased. Of these, the 4 studies with comparable techniques still showed striking differences in the course of cardiac output in the third trimester, with Duvekot *et al.*²¹ showing a decrease of 11.5%, no change by Robson *et al.*²² and increases of 9.3% by Mabie *et al.*¹⁹ and 16.4% by Thomsen *et al.*²³ Although design differences and measurement techniques among studies can explain some of the reported differences in maternal hemodynamics in normal pregnancy, most researchers concur that patient factors rather than measurement error are largely responsible for discrepancies in reported studies.

Also, as shown in this study and others,²⁴ parity correlates with cardiac output, so over-representation of women with higher

parity in the third trimester could also result in higher cardiac output relative to the second trimester.

The discrepancies observed in cardiac output in the third trimester could also be a result of maternal position during the examination as lying in the supine position is expected to decrease stroke volume by up to 5%.²⁵⁻²⁷ This is due to the compression of the inferior vena cava by the uterus especially in the third trimester when the weight of the uterus is increased.²⁸

Though the mean heart rate in pregnancy is within the limits of normal, pregnancy was found to cause an increase of 12 beats per minute with up to 11% having tachycardia (OR= 0.597, confidence interval= 0.524-0.681). No documentation of bradycardia in pregnancy. This finding is similar to the earlier reports that found a marginal increase in heart rate by about 10-20 beats per minute^{29,30} and that of Akinwusi *et al.*¹² who found tachycardia in about 9% of pregnant women against 3% of control in a study of 69 pregnant women.

The occurrence of APC and VPC is not significantly higher in pregnancy, both occurring in 5% of pregnant women against 3% and 1% of control. Left bundle branch block and nonspecific intraventricular blocks in form of Rsr pattern in aVF and/or lead III are significantly more common in the pregnant women than the controls. This report is similar to the finding of Akinwusi *et al.*¹² However, Shotan *et al.*³¹ showed a prevalence of isolated APC and VPC in a population of normal pregnant women with complaints of palpitation, dizziness, or syncope to be up to 56% and 59% respectively while multifocal VPCs occurred in 12%.

This study also suggests that there is a leftward displacement of QRS axis with advancing pregnancy. There is also a negative correlation between gestational age and the QRS axis.

This finding is similar to the report of Iwobi *et al.*¹¹

This finding of leftward shift could be explained by the left ventricular hypertrophy which is progressive from the first to the third trimester. Also, the pressure effect of the enlarging uterus on the diaphragm and consequent on the apex of the heart which would then be shifted leftwards

The return of the QRS axis to its original pre-pregnancy direction thought to occur in late pregnancy³², or after delivery³³, is thought to be a consequence of lightening – the process whereby the uterus assumes a lower position, thereby releasing the pressure it exerted earlier in the pregnancy on the diaphragm³⁴

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