Original Article

Cardiovascular changes in different gestational ages of pregnancy

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Abstract

Introduction: Pregnancy is a normal physiological phenomenon causing major hemodynamic changes. **Aim:** To measure various cardiovascular changes that occurs during various trimesters of pregnancy. **Objective:** To record the pulse rate, heart rate, blood pressure (BP) and electrocardiographic (ECG) changes to see any variations in different trimesters of pregnancy. To determine if underlying physiological changes predispose the pregnant women to any cardiovascular risk. **Method:** Eighty healthy pregnant women in the age group 20-35years were included in the study. The following cardiovascular parameters were recorded pulse, BP, heart rate (HR) and ECG were thoroughly studied. In ECG- RR interval, P wave duration , PR interval, QRS complex, QT interval, QTC interval, P axis, QSR axis, Taxis, P(II)volt, S(V1)volt, R(V5)volt were noted **Results:** A significant increase in the pulse rate, heart rate was seen as the pregnancy progressed. Other changes in the BP and ECG found were positive however not statistically significant. QT interval shortened and the Taxis was altered. Dominance of the sympathetic nervous system (SNS) over the parasympathetic nervous system (PNS) has been shown to be a strong risk factor for cardiovascular disease. Sympathetic dominance is the cause of cardiovascular changes seen during pregnancy. **Conclusion:** Pregnancy has an effect on heart so as to attenuate the vagal dominance in the form of increase in the pulse and heart rate and shortening of QT interval which may be concern in patients with cardiovascular disease during pregnancy.

Key words: Normal pregnancy, cardiovascular changes, ECG.

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INTRODUCTION

During pregnancy, there are a number of important changes to cardiovascular function which are necessary for progression of a successful pregnancy. Additionally, preexisting cardiovascular conditions can be exacerbated by the adaptations that occur during gestation. These can present serious therapeutic challenges in the management of the cardiology patient during pregnancy. Significantly, the number of pregnant women at risk of cardiovascular complications is on the rise, so identification of the changes as risk factors that predict cardiac outcomes is essential to proper screening of the obstetrical patient.[1] Pregnancy is a normal physiological phenomenon causing major haemodynamic changes, including an increase in cardiac output, as well as sodium and water retention. In addition, these haemodynamic changes can mimic and/or precipitate cardiac diseases and cause diagnostic difficulties during pregnancy. There is a paucity of data relating to cardiovascular (CV) and ECG changes in healthy pregnant women. This study was therefore undertaken to describe the CV and ECG changes in normal pregnancy.[2]

MATERIAL AND METHODS

The study was conducted at Bhaskar Medical College and Hospital with collaboration of department of Physiology and Obstetrics and Gynecology during the year 2012.This study has been approved by the institutional Ethics Committee. The study population was 80 subjects in the age group of 20 to 35years attending the outpatient department of Obstetrics and gynecology for antenatal check up at Bhaskar Medical College and General

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Hospital. The study group was divided equally into four as-

GROUP-1- control (non pregnant women)
GROUP-2- 1st trimester of pregnancy
GROUP-3- 2nd trimester of pregnancy
GROUP-4- 3rd trimester of pregnancy

All the subjects were explained about the implications of the study and a written consent was taken from them to fulfill ethical guidelines.

The following parameters were studied

Pulse rate- By examining Radial pulse by usual palpation.
Blood pressure- by using sphygmomanometer
ECG- by Schillers 12 lead electrocardiogram
Location of apex beat-by palpation, percussion and auscultation.

As directed by the obstetric department pregnant mothers in the different gestational group were identified and the above parameters were studied .A 12 lead Schiller's ECG machine as shown in Fig-1 was used to record the electrocardiogram. The parameters recorded were heart rate, RR interval, P wave duration PR interval, QRS complex, QT interval, QTC interval, P axis, QSR axis, Taxis, P(II)volt, S(V1)volt, R(V5)volt as shown in Fig-2. The data was processed through SPSS software. All the participants were healthy with no history of any medical illness.

Figure 1

Figure 2

OBSERVATION AND RESULTS

Table 1

Parameter	Group	N	Mean	SD	Minimum	Maximum	P-value
Pulse Rate	Control	20	73.8	4.96	60	80	*<0.0001*
	First trimester	20	77.0	7.95	64	96	
	Second trimester	20	88.7	8.49	78	102	
	Third trimester	20	86.0	14.73	60	120	
Systolic BP	Control	20	115.0	12.77	80	130	0.434
	First trimester	20	118.3	8.97	100	136	
	Second trimester	20	113.5	10.40	100	130	
	Third trimester	20	111.9	17.13	80	148	
Diastolic BP	Control	20	74.0	11.88	40	90	0.306
	First trimester	20	73.0	11.29	50	90	
	Second trimester	20	71.5	10.89	60	90	
	Third trimester	20	67.5	12.09	40	80	
Pulse Pressure	Control	20	41.0	10.21	20	60	0.543
	First trimester	20	45.3	11.39	20	76	
	Second trimester	20	42.0	8.94	20	60	
	Third trimester	20	44.4	11.60	30	78	
Mean arterial BP	Control	20	89.1	13.31	53	113	0.101
	First trimester	20	89.0	10.60	70	113	
	Second trimester	20	86.0	11.01	73	113	
	Third trimester	20	79.3	19.37	17	100	

Table 2

Parameter	Group	N	Mean	SD	Minimum	Maximum	P-value
HR (per min)	Control	20	79.3	7.20	68	97	***<0.0001***
	First trimester	20	82.8	8.73	67	97	
	Second trimester	20	97.1	15.73	74	140	
	Third trimester	20	90.8	16.98	61	120	
RR (ms)	Control	20	741.3	88.54	555	877	***0.005***
	First trimester	20	733.0	79.79	621	902	
	Second trimester	20	633.6	98.62	430	815	
	Third trimester	20	686.5	133.93	500	990	

Table 3

Parameter	Group	N	Mean	SD	Minimum	Maximum	P-value
QT(ms)	Control	20	364.8	25.20	328	416	***0.033***
	First trimester	20	368.3	30.68	316	432	
	Second trimester	20	341.5	27.71	296	406	
	Third trimester	20	363.1	38.66	306	442	
T(degree)	Control	20	12.6	44.80	-152	61	***0.013***
	First trimester	20	44.3	42.03	12	172	
	Second trimester	20	8.3	45.82	-155	52	
	Third trimester	20	-8.6	64.84	-154	51	

As shown in table -1 and chart-1 there is an increase in the mean pulse rate in all the trimesters and is observed more in the 2nd trimester. The blood pressure did not show any significant change but the diastolic blood pressure showed decrease in all the trimesters as seen in chart-2. Mean heart rate also showed an increase in all the trimesters and more in the 2nd trimester seen in chart-3.Table -2 shows a significant decrease of Mean RR interval in the 2nd and 3rd trimester. QT interval changes were significant. T axis showed significant decrease in 1st and 3rd trimesters as seen in the Table-3
Only statistically significant data has been represented in the tables

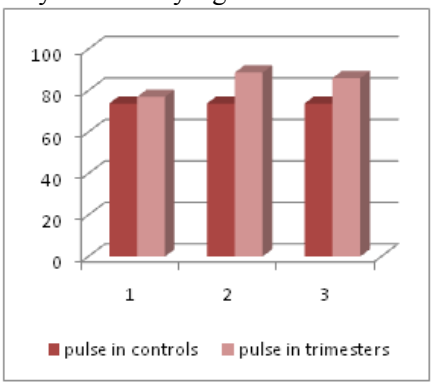

Figure 1: pulse across the 3 trimesters

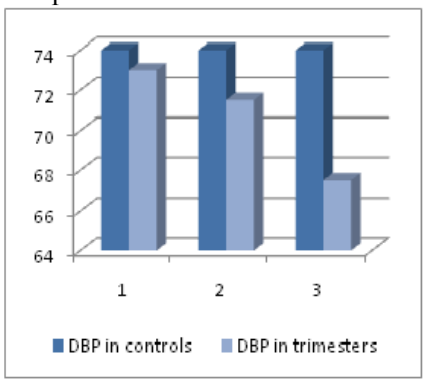

Figure 2: DBP across the 3 trimesters

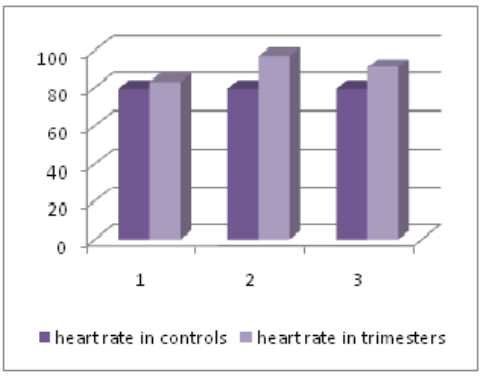

Figure 3: heart rate across the 3 trimesters

DISCUSSION

Among the various changes that occur during the pregnancy is the changes in the blood pressure. The low DBP was expected, because pregnancy reduces systemic vascular resistance and after load, as a result of peripheral vasodilatation and the low resistance, high flow circulation of the uterus and placenta similar were the findings of Grindheim etal [3] and Capless *et al*[4] Moodly [5] found out in his study that the average decrease in systolic blood pressure is 5–10 mmHg and the decrease in diastolic is 10–15 mmHg. If this decrease fails to occur, he reported that such women are more likely to develop hypertension in the third trimester of pregnancy. Hunter[6] explained the haemodynamic changes during normal singleton pregnancy in longitudinal studies. Cardiac output increases by five weeks after the last menstrual period and the increase continued to 24 weeks when it is 45% above the non-pregnant level. Thereafter no further significant change is normally found. Both heart rate and stroke volume contributed to this increase: the heart rate increases by five weeks' gestation and continues till 32 weeks as also is seen in this study. The stroke volume increase occurs a little later at eight weeks and reaches its maximum at about 20 weeks. Cardiac output reaches a maximum at the end of the second trimester. Venous return is bound to be increased during pregnancy probably because of the 40% increase in blood volume. Despite this increase in volume the pressures within the

lung vessels and the central venous pressures do not increase during pregnancy possibly because of ventricular dilatation and reduction in vascular resistance. The first haemodynamic change during pregnancy seems to be a rise in heart rate. Starting between two and five weeks this continues well into the third trimester. Stroke volume increases slightly later than the heart rate and continues throughout the second trimester after an augmentation of venous return and a fall of systemic vascular resistance and after load. Myocardial contractility is probably slightly increased. During the third trimester there is relatively little change in these cardiac indices. Structural changes within the heart reflect the volume loading of pregnancy and include dilatation of the valve ring and increase in myocardial thickness.

According to Richard *et al*[7] pregnancy is a condition of primary peripheral arterial vasodilation, which initiates a series of haemodynamic and hormonal events resulting in high cardiac output and plasma volume expansion. The hyperdynamic circulation of pregnancy develops in early gestation, and the increase goes on to the end of the second trimester.

Hall *et al* 1 gave the evidence that nitric oxide (NO) production is elevated in normal pregnancy and that these increases appear to play an important role in the vasodilation of pregnancy. Inhibition of NO synthesis in animal models of pregnancy attenuates the decreases in total peripheral resistance and increases in cardiac output associated with pregnancy. Hormonal factors such as estrogen and relaxin are thought to be important in stimulating the production of NO during pregnancy. Relaxin, which is primarily produced by the corpus luteum, has been shown to chronically reduce total peripheral resistance and increase cardiac output and systemic arterial compliance. In addition, neutralization of endogenous circulating relaxin by antibodies during early gestation markedly attenuate the changes in cardiac output, systemic vascular resistance, and arterial compliance during pregnancy. These effects of relaxin are thought to be mediated by interactions between endothelin type B receptors and NO. Thus, relaxin appears to play an important role in many of the cardiovascular adaptations of pregnancy via NO-dependent mechanisms.

Speranza *et al*[8] in their study found changes in pregnancy as a shift of autonomic balance as the consequence of attenuation of baroreflexes.

Halphen [9] showed pregnancy affects the ECG at some time point and there is restoration of these pregnancy-induced changes late in pregnancy or following delivery.HR increases progressively throughout the pregnancy, reaching a peak during the third trimester as was seen in our study. This increase in HR seems related to hormonal factors in early stages of pregnancy and later to increased left atrial diameter and sympathetic activation (sinus-node remodeling. Halphen also described that gestational age also impacts T-waves, promoting a leftward axis shift as pregnancy progresses as also is seen in our study. In particular, a leftward deviation of the mean QRS axis during the second and third trimesters of pregnancy and then rightward before delivery is observed in the majority of women. PR interval exhibits a significant reduction in the mean values during pregnancy, while the QRS amplitude generally increases slightly in the late pregnancy (but without a clear evidence of left ventricular hypertrophy).

Moertl *et al* concluded that the non-invasive determination of cardiovascular and autonomous parameters throughout pregnancy is possible and the results of their pilot study can serve as basic parameters for classifying and assessing cardiovascular and autonomous changes in pathological conditions in pregnancy such as hypertensive disorders.

So our study too is a modest effort to add to the scientific information to this topic, the major limitation in this study was the small sample size.Further a longitudinal study in this area will highlight more facts about the cardiovascular changes in the pregnancy.

CONCLUSIONS

This study has provided data on the cardiovascular and ECG findings in healthy pregnant women in Ranga Reddy District of Telangana. The most common findings on physical examination were low diastolic blood pressure increase in pulse rate as was reflected in heart rate and RR interval. There were also some distinctive ECG features as QT interval shortening and T axis changes can be attributed to the dominance of SNS and should be kept in mind dealing with patients of pregnancy with cardiovascular disease.

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