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RESEARCH ARTICLE

RELATIONSHIP BETWEEN FETAL HEART RATE AND MATERNAL BLOOD PRESSURE AND
DIABETES MELLITUS, AND FETAL SEX

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ABSTRACT

Objective: To assess the association between aspects of maternal health (blood pressure and diabetic status) and fetal heart rate (FHR) during the third trimester, and compare FHR in male and female fetuses" or similar.

Methods: This is a descriptive cross-sectional study. The study population comprised 100 pregnant women in the third trimester. The study was conducted in Khartoum state between January and May 2012. Subjects were categorized according to blood pressure (hypertensive, hypotensive, prehypertensive and normotensive) and gestational diabetes status (diabetic vs. non-diabetic). All subjects underwent ultrasound imaging using a 3.5 MHz probe, according to the obstetric scanning protocol. Fetal heart activity was observed using the 4-chamber view and M mode was applied to measure the FHR. Data were collected on a clinical data sheet, designed to record relevant maternal clinical history (hypertension and diabetes mellitus).

Results: Fetal heart rate was weakly correlated with gestational age in the final weeks of pregnancy ($r = 0.35$). Fetal heart rate was higher in subjects with hypertension than in normotensive subjects ($p = 0.000$). Maternal hypotension had no influence on FHR ($p = 0.166$). The FHR of male and female fetuses showed no significant difference ($p = 0.456$). Maternal diabetes did not appear to influence FHR ($p = 0.166$).

Conclusion: FHR is strongly influenced by maternal hypertension. The FHR did not significantly differ between fetuses of diabetic women and non-diabetic women. There was no significant difference in the FHR between male and female Fetuses

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INTRODUCTION

Recording the fetal heart rate (FHR) using ultrasonography is an important part of routine antepartum and intrapartum care. It is well established that FHR is a vital predictor of fetal outcome. Normal FHR changes with progression of the pregnancy. Fluctuation of the FHR is also normal; in early pregnancy, the FHR fluctuates constantly, but during the third trimester, it becomes more stable. Gib and Arulkuman, (1997) and Spencer, (1990) proposed that the range of the baseline FHR should lie between 110 and 150 beats per minute (bpm), and emphasized the possibility of serious complications resulting from tachycardia (>150 bpm (beat per minute) or bradycardia (<110 bpm). The myocardium begins to contract rhythmically by 3 weeks post-conception, at which point cardiac activity is visible on the sonogram; the FHR is usually

around 100–120 bpm. This increases progressively over the subsequent 2–3 weeks, reaching approximately 150 bpm by 14 weeks. The FHR then declines to 140 bpm by 20 weeks and 130 bpm by term. Although the FHR of a healthy fetus is usually regular, a beat-to-beat variation of 5–15 bpm is considered normal (radiopedia.org, 2014). There is no consensus regarding the definition of a normal FHR. Current international guidelines recommend that normal baseline FHR is in the range of 110–150 bpm. Pilderet al. in a study of normal FHR ranges, indicated that the FHR could be expected to lie between 120 and 160 bpm (Pildner et al., 2013).

From 20 weeks gestation, both stability and variability in FHR have been demonstrated. Factors that influence baseline FHR are not well documented, although tonic maternal heart rate and sympathetic arousal induced by physical exercise have some influence. A relationship between maternal heart rate variability and FHR has been demonstrated in pregnancies complicated by pre-eclampsia, but not in uneventful,

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normotensive pregnancies (Swansburg et al., 2005). Maternal hypertension is considered one of the main influences on FHR. Brown et al., studied maternal heart rate variability and fetal behavior in hypertensive and normotensive pregnancies. These authors concluded that there was an association between maternal autonomic modulation of heart rate and FHR, such that the maternal autonomic system influences fetal cardiac function in pregnancies complicated by hypertension (Brown et al., 2008). Hypertension is attributed, at least in part, to autonomic deregulation (i.e., decreased parasympathetic and increased sympathetic cardiac modulation), which results in an increase in maternal heart rate and enhanced vasomotor sympathetic modulation, which causes vasoconstriction (Dabrowaki and Skrobowski, 1996; Guynet, 2006; Julius and Majahame, 2000; Pagani and Lucini, 2001; Souza et al., 2001).

SUBJECTS AND METHODS

This study was conducted at Al zaiemAlazhari University Clinic and Al mutakamil Medical Center, Khartoum State, Sudan between May 2012 and December 2012. The sample comprised 100 pregnant women (third trimester, singleton pregnancy) referred to the ultrasound department for routine antenatal fetal assessment. Subjects with a history of fetal anomaly were excluded from the study. A clinical data sheet was designed for recording personal data and clinical history, such as the presence of diabetes and hypertension.

Maternal blood pressure

Blood Pressure (BP) was measured using a standard mercury sphygmomanometer. Antenatal phase 1 and phase 5 Korotkoff sounds were considered representative of the systolic and diastolic pressure, respectively. Study subjects were grouped into 4 categories on the basis of maternal BP, in accordance with the categories outlined by the National High Blood Pressure Education Program:

- 1- Normotensive: maternal systolic BP ≤ 120 mmHg, maternal diastolic BP ≤ 80 mmHg
- 2- Prehypertensive: maternal systolic BP 120–139 mmHg, maternal diastolic BP 80–89 mmHg
- 3- Hypertensive: maternal systolic BP ≥ 160 mmHg. Clinical history includes a diagnosis of hypertension.
- 4- Hypotensive: maternal systolic BP ≤ 90 mmHg. This hypotension is transient, as reported in the patient's file, and was attributed to the pregnancy.

Subjects were also categorized according to the presence of diabetes, diagnosed and reported in the medical records (diabetes, $n = 14$; non-diabetic, $n = 86$).

Fetal heart rate

The FHR was estimated using an ultrasound scan during the third trimester (26–42 weeks gestation). Subjects were scanned in accordance with the International Protocol Guidelines of Fetal Scanning in Obstetric Ultrasonography.

Sonographic technique

Cross-sectional gray scale ultrasound imaging is the basis for assessing fetal cardiac activity. Longitudinal, transverse, and oblique sections were performed through the fetal chest to

localize the heart, and to obtain a 4-chamber view. Once the heart was visualized, M mode was set to enable calculation of the FHR. The procedure was performed using a convex transducer (3.5 MHz) known to be suitable for use during pregnancy. Ultrasound was performed by 2 expert sonographers and obstetricians. The fetal pelvis was also scanned to determine sex, with reference to the external genitalia (male, scrotum; female, (labia majora). Informed consent was obtained from all patients for being included in the study.'

Statistical Analysis

The data had been analyzed using software program SPSS (Statistical Package for Social Sciences) version 16. Variable statistical tests had been used to analyze the data of the study among which were Anova, Independent T-test and linear Correlation. Anova test was used to compare means within and between the groups such as comparing the HR of the fetuses among hypertensive and normotensive pregnancies. Correlation test was used to find the association between fetal HR and gestational age. The independent T-test was used to compare the means among each paired groups such as comparing the mean FHR between diabetes and non-diabetes and comparing the mean FHR between male fetuses and female fetuses. The study considers 5% as significance value, so $p\text{-value} > 5\%$ was considered to be insignificant and $p\text{-values} < 5\%$ was considered to be significant results.

RESULTS

As shown earlier in statistical analysis, there were important statistical tests had been used. Table 1 showed the general descriptive statistics of the study variables and the mean of the FHR and GA had been estimated.

Table 1. Descriptive statistics for fetal heart rate, gestational age

	N	Minimum	Maximum	Mean	SD
Fetal heart rate	100	120	166	141.73	10.318
Gestational age	100	37	42	38.37	1.284
total	100				

Abbreviations: SD, standard deviation; N, total number

Table 2. Correlation between fetal heart rate and gestational age

		Fetal heart rate	Gestational age
Fetal heart rate	Pearson correlation	1	-.353**
	Sig. (2-tailed)		.000
	N	100	100
Gestational age	Pearson correlation	-.353**	1
	Sig. (2-tailed)	.000	
	N	100	100

** Correlation is significant at the 0.01 level (2-tailed).

Table 3. Relationship between mean fetal heart rate and maternal blood pressure

ANOVA test					
Fetal heart rate	Sum of squares	Df	Mean square	F	p-value
Between groups	2754.475	3	918.158	11.322	0.000
Within groups	7785.235	96	81.096		
Total	10539.710	99			

Abbreviation: Df, degrees of freedom

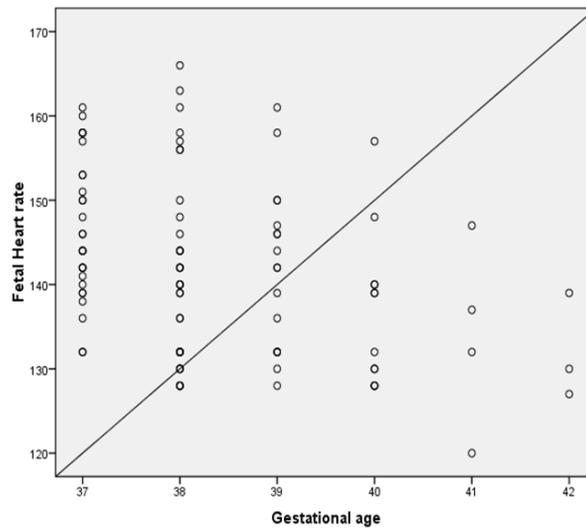


Figure 1. Correlation between fetal heart rate and gestational age

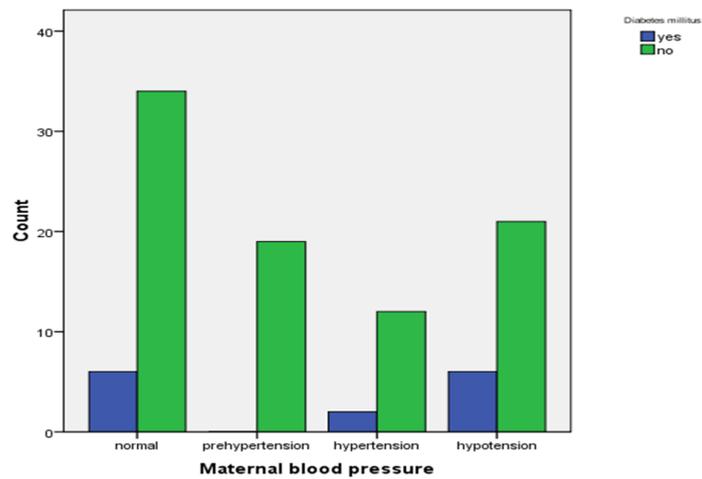


Figure 2. The classification of maternal blood pressure in the study population

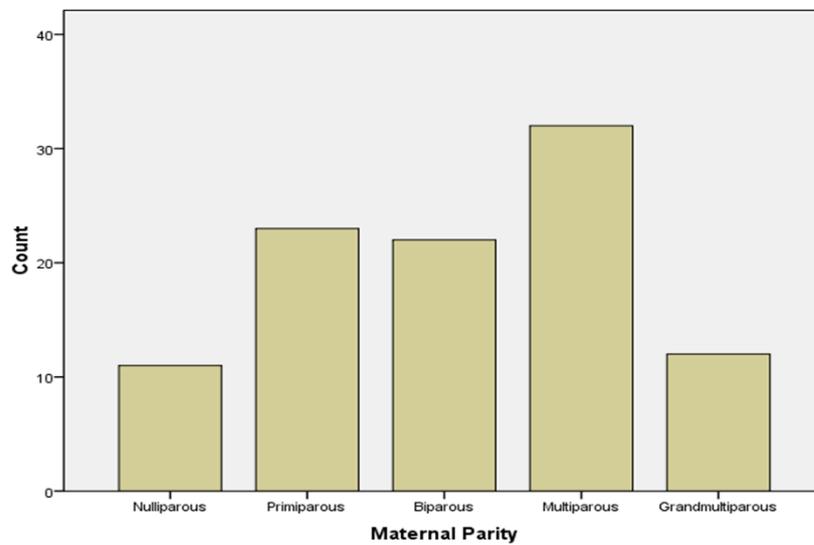


Figure 3. Status of parity of the study population

Table 2 and Figure 1 showed plot diagram demonstrating the correlation between FHR and GA and it was observed that was no significant correlation. Figure 2 showed the classification maternal blood pressure versus maternal gestational diabetes, Figure 3 revealed the maternal parity and it was noted that multiparous mothers were dominant among the study population. In Table 3 and Table 4, the Anova test was used to compare the mean FHR with the maternal hypertension as general, it was observed that there was significant change (p -value $<5\%$) which means maternal blood pressure could influence FHR.

Table 4. The comparison of heart rate of fetuses of hypertensive and normotensive pregnancies

Mother Blood Pressure	N	Mean	SD	p value
Fetal heart rate	40	141.05	7.348	0.00
Normal				
Hypertension	14	154.36	7.281	

Abbreviations: SD, standard deviation; N, total number

Table 5. The comparison of fetal heart between maternal hypotension and normotensive groups

Mother Blood Pressure	N	Mean	SD	p-value
Fetal Heart rate	40	141.05	7.348	0.166
Normal				
Hypotension	27	137.96	10.700	

Abbreviations: SD, standard deviation; N, total number

Table 6. Fetal heart rate according to fetal sex

Fetal sex	N	Mean FHR (bpm)	SD	pvalue
Male	46	140.89	9.675	0.456
Female	54	142.44	10.874	

Abbreviations: FHR, fetal heart rate; SD, standard deviation; N, total number

Table 7. Relationship between fetal heart rate and maternal diabetic status

Diabetes mellitus	N	Mean FHR (bpm)	SD	p value
Yes	14	145.29	10.593	0.166
No	86	141.15	10.218	

Abbreviations: FHR, fetal heart rate; SD, standard deviation; N, total number

In Table 5, Table 6 and Table 7, the independent Test was used to compare the mean FHR with maternal hypotension, fetus sex and maternal diabetes. It was observed that maternal hypotension had no influence on FHR (p -value $<5\%$) as shown on Table 5. On Table 6, the FHR showed no significant difference between male and female fetuses (p -value $>5\%$) and Table (6) showed the mean FHR in maternal diabetes versus non-diabetes. It was observed that there was no significant difference (p -value $>5\%$), this suggests that maternal diabetes could not influence the FHR.

DISCUSSION

FHR is routinely measured with ultrasound as part of antenatal care. This study assessed the relationship between FHR, measured using ultrasound in the third trimester, and aspects of maternal health (BP, diabetes status). The relationship between FHR and fetal sex was also investigated. Descriptive statistics

relating to the study population are presented in Table (1). The minimum FHR in the study population was 120 bpm (1 case), which lies within the normal range. The maximum FHR in the study was 166 bpm, and the mean \pm standard deviation (SD) was 141.73 ± 10.31 bpm. Pildner et al. concluded that the normal range for FHR is 120–160 bpm, which seems to be safe in daily practice. The measurement of 166 bpm was higher than the normal range and may be attributed to maternal hypertension (Pildner et al. 2013).

The mean FHR in this study lies within the normal range and is consistent with the international value. Subjects were categorized into 4 groups, as shown in Table (2). There was weak negative correlation ($r = 0.35$) between fetal gestational age (GA) and FHR in the study subjects, who were assessed during the final weeks of the third trimester. This indicates that the FHR is unchanged in the last weeks of the third trimester. Figure (1) demonstrates the correlation between the FHR and GA.

The relationship between FHR and maternal BP has been studied by many researchers. In this study, the mean FHR was significantly associated with the maternal BP (p -value = 0.000), as shown in Table (4). This indicates that FHR is influenced with maternal blood pressure. This relationship is clearly evident when the FHR of the normotensive group is compared with that of the hypertensive group; the difference was significant ($p = 0.000$). Therefore, maternal hypertension increases FHR. This result is consistent with that of a study by Brown et al., which suggested that maternal hypertension influences FHR. This is attributed to an association between autonomic modulation of heart rate and FHR. Therefore, the maternal autonomic system influences fetal cardiac function in pregnancies complicated by hypertension (Brown et al., 2008). As shown in Table (6) there was no significant association between FHR and hypotension ($p = 0.166$); this indicates that FHR remains stable in hypotensive pregnancies. On the other hand, female fetuses showed a mild increase in FHR (142.4 bpm) compared with male fetuses (140.9 bpm), although this difference was not significant ($p = 0.456$). Thus, FHR is not an indicator of fetal sex, as previously reported by Terry (Terry et al., 1989).

Table (7) compares the FHR of fetuses of diabetic mothers and non-diabetic mothers. The FHR did not significantly differ between the 2 groups (p value = 0.166). Therefore, FHR might not be influenced by maternal diabetes mellitus. A study performed by Weinar et al. (1996) showed that FHR was different in fetuses of well-controlled diabetic mothers and fetuses of non-diabetic mothers (Weinaret al., 1996). A study conducted by Bjorklund et al. (1996) studied the effect of hypoglycemia on fetal heart rate activity and umbilical artery Doppler velocity waveforms in pregnant women with insulin-dependent diabetes mellitus. The result of his study concluded that hypoglycemia was associated with an increase in frequency and amplitude of fetal heart rate acceleration. This result is contradicted with our result, since we studied the maternal diabetes on time of scanning regardless of hypo or hyperglycemia.

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Conflicts of interest: None.

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