Role of Elaborate Cardiotocography (CTG) in Pregnancy Management

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Abstract:

Background: Elaborate Cardiotocography (CTG) is the most commonly used test for antepartum and intrapartum fetal surveillance because it gives information via the cerebro-cardiac response of fetal cerebral activity, which is modified by the hypoxia. **Objective:** This study was designed to compare the perinatal outcomes among the normal and abnormal CTG groups. **Method:** It was a prospective observational study carried out in the Department of obstetrics, BSMMU during the period July 2006 to July 2008. Hundred consecutive normal and hundred consecutive abnormal CTC tracings were collected from patients who were advised to perform CTG after admission. Both labour and non-labour patients were included. Interpretation of CTG was done based on FlGO recommendation (1987). Pregnancy and neonatal data were obtained and the findings were correlated with the FHR tracing. Statistical analysis was carried out by student's unpaired t-test, X^2 and Z-test. Level of significance was set at P value < 0.05. **Results:** Out of 100 abnormal CTG, 30% had tachycardia, 42% had deceleration, 38% was non reactive, 4% had absence beat-to-beat variability and 4% had fetal bradycardia. There was significantly higher caesarean delivery, lower apgar score, higher requirement of neonatal resuscitation and admission at neonatal unit and higher perinatal death among the abnormal CTG group. The abnormal fetal outcome was found highest in heart rate deceleration group. **Conclusion:** CTG can be continued as a good screening test of fetal surveillance but it is not the sole criteria to influence the management of high-risk pregnancies. Abnormal CTG should be supplemented with other test before intervention.

Key words: CTG, Perinatal outcome.

Introduction:

Cardiotocography (CTG) is the graphic presentation of fetal heart activity and the uterine contraction to detect the fetal hypoxia[1]. It is the most commonly used test for antepartum and intrapartum fetal surveillance in the majority hospitals of developed countries[1]. This technology was first developed in 1950 and became commercially available in 1960[1]. The goal of antepartum fetal surveillance is to predict, diagnose and timely intervene the pregnancies those are complicated with fetal asphyxia and might lead to fetal and newborn morbidity and death[2].

The electronic FHR in all fetuses with asphyxia is expected to exhibit one or more heart rate pattern abnormalities. False negative rate of reactive Contraction stress test (CTG) is low, 3 per 1000, which is similar to that of BPP[3]. The negative predictive value is similar to that of BPP (99.5% for NST, 98.5% for BPP) but positive predictive value of an abnormal non stress test (NST) (37%) is better than other screening tests [3]. Sensitivity of NST is 97%[3]. In addition, electronic FHR monitoring is significantly better in detecting all types of acidemia, metabolic (95.5%), mixed (95%) and respiratory (100%)[3].

Contraction stress test (CST) is one of the forms of fetal surveillance; it is still the only form that tries to use the principle of induced stress to reveal marginal placental insufficiency. But the procedure is risky due to chance of premature labour [4].

Great efforts have been made to increase the reliability of the antepartum non stress test (NST) in order to detect fetal deterioration early [5]. Yet visual interpretation of FHR records is associated with a high inter and intra observer variability that greatly reduces its accuracy [5]. Classification system for electronic FHR monitoring tracing is capable of discriminating both healthy fetuses and those at risk for acidemia at birth and subsequent neonatal complication [6].

From 70 to almost 90 percent fetal death occur before the onset of labour[7]. A large number of antepartum fetal deaths are caused by uteroplacental insufficiency[8]. Antepartum fetal testing by identifying the uteroplacental insufficiency and timely intervention has prevented many fetal deaths[8]. Similarly intrapartum fetal monitoring by detecting fetal distress in early stages has saved many fetuses from demise [9].

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NST is less expensive, noninvasive, technically simpler and less time consuming and has a higher false positive and possibly lower false negative rate than BPP or CST [7-10]. It has surpassed oxytocin stress test due to its safety, convenience to the patient and the physician, non invasiveness, easy interpretation, can be repeated without risk [7-10].

This study was designed to compare the pregnancy outcomes and early neonatal outcomes among the normal and abnormal CTG groups and to find out the association between abnormal findings of CTG with that of pregnancy outcomes and early neonatal outcomes.

Methods:

It was a prospective observational study carried out in the Department of Obstetrics and Gynaecology, BSMMU during the period, July 2006 to December 2007. Only indoor patients were included here.

One hundred consecutive normal and one hundred consecutive abnormal CTC tracings were collected from patients who were advised to perform CTG after admission to exclude fetal asphyxia. Both labour and non-labour patients were included. Patient having multiple pregnancy and pregnancy less than 32 weeks were excluded from the study. Inform consent was taken from the patients. All CTG was performed from the same CTC machine by the same obstetrician. Initially the monitoring was done for 20 minutes and if there was no fetal movement or heart rate acceleration within those 20 minutes then the monitoring was continued for another 20 minutes.

Interpretation of CTG was based on FlGO recommendation (1987). CTG was considered normal when the basal heart rate was 110-150 bpm, beat to beat variability was 5-25 bpm and at least two accelerations of the fetal heart rate were present during a 20 minutes period, each at least 15 beats above the baseline rate and lasting at least 15 seconds. Abnormal CTG included fetal tachycardia (> 150 bpm), fetal bradycardia (FHR< 110 bpm), reduced or absent beat-to-beat variability, late decelerations and extreme variable decelerations.

Pregnancy outcomes and neonatal data were obtained from delivery note and neonatal chart review and the findings were correlated with the FHR tracing.

Measures of pregnancy outcomes were mode of delivery, indications of caesarean section, percentage requiring caesarean section for fetal distress, olygohydramnios, meconium stained liquor, small placenta, and cord around the neck or the fetus.

Measures of neonatal outcome were Apgar score at 1 and 5 minutes after birth, birth weight, requirement of admission into neonatal intensive care unit (NICU) for reasons other than pre-maturity, duration of stay in NICU, complications, perinatal mortality.

Statistical analysis was carried out by unpaired students t-test, Chi-square test and Z-test. Level of significance was considered significant when P value was 0.05 or less.

Results

The mean age of the patients were 26.7±4.9 years ranging from 18 to 36. The mean age, height, weight, socio economical status and occupation were almost similar among the patients of both normal and abnormal CTG group (Table I).

Table-I
Distribution of the patients by socio demographical characteristics

Variables	Abnormal CTG (n=100) Mean ± SD	Normal CTG (n=100) Mean ± SD	P value
Age in years	26.7±4.9	26.7±4.9	0.15[NS]
Height (cm)	152.6± 5.2	153.1± 3.6	0.13 [NS]
Weight (kg)	63.5± 5.4	62.8± 7.3	0.583 [NS]
Occupation			
Housewife	72%	80%	
Teacher	12%	6%	
Service holder	10%	14%	
Student	6%	0	
Economical status			
Poor	52%	48%	
Middle class	38%	62%	

NS = P > 0.05

Previous history of stillbirth and abortion were more among the pregnant women with abnormal CTG but the difference was not statistically significant.

Haemodynamic parameters and haemoglobin were similar in both normal and abnormal CTG group (Table II) however; blood sugar was higher among the patient with abnormal CTG (p<0.05).

Figure 1 shows the different indications of CTG. Highest number was less fetal movement (23.0%), followed by pre-eclampsia (18.0%), oligohydramnios (18.0%), prolonged 1st stage of labour (12.0%), postdated

pregnancy (10.0%), IUGR (9.0%), GDM (8.0%), PROM (7%), chronic hypertension (7.0%) and others were Rh-ve, HbsAg +ve, placenta previae, thalassemia, fibroid uterus, meconeum stained liquor, polyhydramnios, CPD, bad obstetric history, rubella infection, and congenital heart disease.

Table-II

Haemodynamics, hematocrit and blood sugar levels of the study patients (n=100)

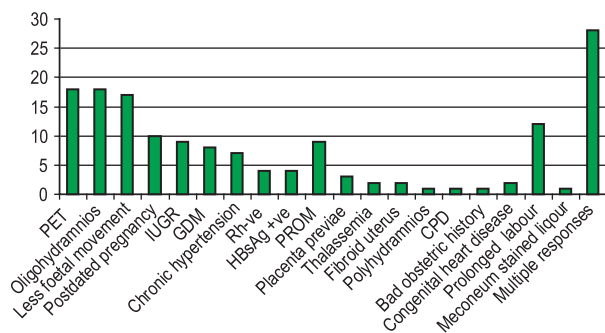| Parameters	Abnormal CTG	Normal CTG	p value
Pulse/minute	84.5±7.5	81.4±5.9	0.023*
Systolic Blood Pressure (mmHg)	123.9±18.3	122.9±15.9	0.771 NS
Diastolic Blood Pressure (mmHg)	81.8±15.2	79.4±12.4	0.389 NS
Haemoglobin (gm %)	10.0± 1.7	10.2± 1.3	0.503 NS
Blood Sugar (mmol /l)	6.9± 1.8	6.2± 1.0	0.021*

NS = P > 0.05
*   = P < 0.05

Fig. 1: *Indications of CTG*

Table-III shows the CTG abnormalities of the studied women. Out of 100 abnormal CTG 30% had fetal tachycardia, 38% had deceleration, 38% was non reactive CTG, 4% had fetal bradycardia and 4% had absence beat-to-beat variability. 18 patients had more than one abnormal findings.

Pregnancy outcome with abnormal CTG is shown in Table-IV. It was found that out of 100 pregnant women, two fetuses were stillbirth and two died at 2nd and two at 3rd day of birth (perinatal death), all these six had deceleration in CTG (Table VII). Other abnormal outcomes were low apgar score at 1 minute and 5 minute, the babies needed immediate resuscitation and admission at neonatal care unit. All these parameters were higher in the abnormal

Table-III

Frequency distribution of major CTG abnormality of the patients (n=100)

Types of abnormal CTG	Frequency	Percent
Tachycardia	30	30.0
Bradycardia	4	4.0
Absent/reduced beat to beat variability	4	4.0
Deceleration	38	38.0
Non reactive	38	38.0

Table-IV

Distribution of the patients by pregnancy outcome in abnormal CTG group (n=100)

Types of abnormal CTG	Normal outcome	Abnormal outcome
Tachycardia (n=30)	26 (86.7%)	4 (13.3%)
Bradycardia (n=4)	4 (100%)	0 (0.0)
Absent beat to beat variability (n=4)	2 (50.0%)	2(50.0%)
Deceleration (n=38)	6 (15.8)	32 (84.2%)
Non reactive (n=38)	10 (52.6%)	9 (47.3%)
Total (n=100)	46 (46%)	54 (54%)

Table-V

Distribution of the patients by delivery related variables

Variables	Study subjects		p value
	Abnormal CTG (n=100) Frequency	Normal CTG (n=100) Frequency	
Mode of delivery			
LUCS	82	22	0.001**
NVD	18	78	
Liquor amount			
Almost nil	12	0	0.091 NS
Less amount	30	26	
Adequate	56	74	
Polyhydramnios	2	0	
Liquor colour			
Normal	60	86	0.003**
Meconeum stained	40	14	
Umbilical cord			
Normal	94	100	0.242 NS
Compressed	6	0	
Placenta			
Normal	84	96	0.046*
Big	6	0	
Small	10	4	

** = P < 0.01
NS = P > 0.05
*   = P < 0.05

CTG group. The abnormal fetal outcome was found to be highest in heart rate deceleration group followed by absence beat to beat variability and then non reactive CTG and lastly tachycardia group, the percentage were 76%, 50%, 47% and 13% respectively. Pregnancy outcome was abnormal in only 4% of normal CTG group.

Caesarian delivery and meconium staining liquor was significantly higher in abnormal CTG group (p < 0.001). It was also revealed that the proportion of adequate amount of liquor was higher among the women with normal CTG (74.0.0%) compared to abnormal CTG (56.0%), but the difference was not statistically significant (p>0.05). Similarly the proportion of normal placenta was significantly high among the women with normal CTG

(96.0%) compared to abnormal CTG (84.0%). However, a statistically significant difference was found (p<0.05) in colour of liquor indicating the proportion of meconeum stained liquor was higher among the women with abnormal CTG (40.0%) compared to normal CTG (14.0%). No statistically significant difference was found in umbilical cord compression (p>0.05) though the compressed umbilical cord was found to be high among the women with abnormal CTG (Table V).

Fetal birth weight and apgar score at 1 minute and 5 minute was significantly lower in abnormal CTG group. The babies needed immediate resuscitation and admission at neonatal care unit was higher in this group (Table-VI). The fetal death rate was high among the women with abnormal CTG.

Table-VI

Distribution of the patients by fetal outcome

Variables	Study subjects		p value
	Abnormal CTG (n=100)	Normal CTG (n=100)	
Birth weight (kg)	2.4±0.8(1.2-4.0)	2.7±0.6(1.4-4.8)	0.036*
Apgar score (1 minute)	6.2 ±1.5(0- 9)	7.0 ±0.9(5- 10)	0.003**
Apgar score (5 minute)	8.6 ±1.6(0- 10)	9.3 ± 1.2(2- 10)	0.014*
Immediate resuscitation needed	76%	54%	0.021*
Admission into neonatal care unit	42%	10%	0.001**

Data expressed as mean ± SD and percent. Figures in parenthesis indicate range.
** = P < 0.01

Table-VII

Evaluation of perinatal death in different cases (medical audit)

Sl. No.	Outcome	Ges. age	Maternal risk factors	CTG	Mode of delivery	Cause of death
1	Stillbirth	36	Severe jaundice	Non reactive Deceleration present	Vaginal delivery	Birth weight 2.5 kg Birth asphyxia
2	Stillbirth	34	Oligohydramnios with severe preeclampsia	Non reactive Deceleration present	LUCS	Birth weight 1.8 kgAntepartum asphyxia
3	Neonatal death at 2nd day	33	Pre eclampsia with GDM with IUGR	Non reactive Deceleration present	LUCS	Birth weight 1.4 kg, SGA with prematurity
4	Neonatal death at 2nd day	33	Oligohydramnios, IUGR	Non reactive Deceleration present	LUCS	Birth weight 1.2 kg, SGA with prematurity
5	Neonatal death at 3rd day	34	Pre eclampsia with DM	Non reactive Reduced bear to beat variability Deceleration present	LUCS	Birth weight 4 kg, RDS.
6	Neonatal death at 3rd day	34	Oligohydramnios, IUGR	Non reactive Deceleration present	LUCS	Birth weight 1.1 kg, SGA with prematurity

Discussion:

The goal of the obstetrician and neonatology is not only to prevent fetal death by taking care of pregnent women but also to detect fetal compromise and to monitor timely delivery of such infant .[7] Seventy to ninety percent of fetal deaths occurred before the onset of labour.[7] A number of intra partum fetal deaths occur among compromised fetus which can not tolerate the stress of the labour and succumb.

Although technology has made great advances in the field of antepartum fetal surveillance and intrapartum monitoring, obstetricians should be aware of the limitation of these methods.[11] The diagnosis of fetal distress during labour cannot be assessed by any single clinical or laboratory measurement. CTG is the most commonly used test for antepartum and intrapartum fetal surveillance in majority hospitals of developed countries although the clinical impact of cardiotocography on neonatal outcome remains controversial[1]. CTG provides direct information of fetal condition in contrast to other technique. The rational behind this test is that it gives an indication via the cerebro-cardiac response of fetal cerebral activity that is modified in the presence of hypoxia. Acceleration of FHR is due to intact responsive CNS mechanism. The loss of FHR variability or decelerations reflects depression of this CNS mechanism. It is not only the result of fetal hypoxia and acidosis; it can be due to fetal sleep, fetal anomalies, sedative and narcotics to mother, which explain healthy outcome of non-reactive CTG. In spite of lack of specificity cardiotocography is a useful procedure for antepartum fetal surveillance and it remains the mainstay of intrapartum fetal assessment [11]. The purpose of this study was to test the ability of a CTG to predict pregnancy outcomes and early neonatal outcomes.

CTG was taken from patients at > 32 weeks gestation because the likelihood of a non-reactive test is substantially increased early in third trimester. Both Lavin et al[12] and Druzin et al[13] have reported that 15% of NST remain non reactive between 28 and 32 weeks. After 32 weeks, the incidences of reactive and non-reactive tests are comparable to those seen at term and eliminate concern regarding the immature nervous system[7]. Before 27 weeks gestation, the fetal heart response to fetal movement may in fact be a bradycardia[7].

There was no significant difference between the two CTG groups regarding the maternal age, parity, gravidity and gestational age, which was similar to many published studies such as by Dellinger et al[6]. Both the groups included patients who were relatively elderly and have low parity. Gravidity was higher due to induced abortion or MR and history of repeated pregnancy loss.

Though the mean gestational age of the two CTG groups showed no significant difference, but in abnormal CTG group the frequently observing gestational age was lower than the normal CTG group because of early intervention was taken by observing the abnormal fetal heart rate pattern.

CTG showing only tachycardia had 13.3% abnormal out comes. It is the early sign of fetal distress[7]. As interventions were taken early outcomes were good. Bradycardia was seen in 4% case outcome was normal. Druzin et al[14], Dashow et al[15] in their study observed bradycardia in 1-2% of all CTG. Druzin et al[14] defined such bradycardia, a fetal heart rate of 90 beats per minute or a fall in the fetal heart rate of 40 beats per minute below the base line for one minute or longer. Druzin et al[13] in his study found that bradycardia was associated with increased morbidity and mortality; the causes were cord compression, IUGR and fetal malformation.

In the current study 50% tracings with absent beat-to-beat variability showed abnormal outcomes. Studies done by Shields et al[16] and Langer et al[17] demonstrated that the fundamental component of ominous fetal heart rate pattern is absent or markedly decreased FHR variability.

In this study non-reactive CTG were associated with 47.3% abnormal outcomes which is similar to the studies done by Kubli et al[18], Schifrin et al[19] Keogen et al[20], Flynn et al[21].

CTG showing decelerations were associated with 84.2% abnormal outcomes. In this study deceleration included spontaneous decelerations in antepartum CTG, moderate to severe variable decelerations and late decelerations. In most cases associated with reduced baseline variability and basal fetal heart rate abnormality.

When the risk factors are more the over all outcomes are more among the abnormal CTG group[17]. In this study it was seen that when diabetic patients developed preeclampsia, intrauterine growth retardation, oligohydramnios, abnormal outcomes were more and risk factors are interrelated, one predispose to others.

In respect to mode of delivery, there was a high incidence of caesarean section in this study. The reason for high incidence of caesarean section in this study in spite of normal test result was due to obstetrical indications, like history of previous caesarean section, cephalopelvic disproportion, severe preeclampsia and severe intrauterine growth retardation. Here caesarean delivery for fetal distress was significantly higher in abnormal CTG group, this finding is similar to the observation of Dellinger et al[6].

Reduced liquor volume before labour is considered an indication of placental insufficiency and reduced liquor volume during labour is associated with an increased incidence of FHR decelerations and may reduce the volume of the intervillous space and may predispose to umbilical cord occlusion, which increase the risk of fetal hypoxemia. In the past, the presence of meconium in the amniotic fluid was considered to be a sign of fetal hypoxia.

In the study Apgar score was < 7 at 1 min among the babies of abnormal CTG group than normal CTG that was similar to the study done by Dellinger et al[6]. The difference in number of the babies with Apgar score < 7 at 5 min was very insignificant in normal CTG group than abnormal CTG group which was similar to many other studies.

Apgar scoring has been the conventional means of evaluating the status of the infant at birth. It is usually assumed that this score reflects the degree of neonatal asphyxia. However recent studies [17] using cord blood analysis and fetal scalp blood pH have cast serious doubts on the reliability of Apgar scoring for asphyxia. An analysis of several published works on the subject gives the following approximate indexes of accuracy for the Apgar score in the prediction of hypoxemia: sensitivity 47%, specificity 89%, positive predictive value 56%, negative predictive value 86%[22].

In the present study no perinatal death was observed in normal CTG group. In abnormal CTG group rate was 60 per 1000 total birth, which was similar to some other studies. Perinatal mortality (PNM) among total study population (n=200) was 30 per 1000.

The PNM rate in the present study was significantly lower than the PNM in other teaching centers of Bangladesh. Timely intervention according to CTG results may be the reasons for such variation.

The study did not attempt to demonstrate an ability to decrease caesarean delivery rates, nor did it attempt to link electronic fetal monitoring with long term neurologic function and cerebral palsy. It only attempted to show the pregnancy outcome and early neonatal outcomes in case of normal and abnormal CTG cases.

Conclusion:
The present study showed that the mean Apgar score was low among the babies with mothers having abnormal than normal CTG. The proportion of immediate resuscitation and admission of the babies at neonatal care unit was higher among this group. Perinatal death was also significantly higher in this group. Thus overall outcome was better in

normal CTG group. On the other hand the perinatal mortality (PNM) in the present study was significantly lower than the PNM of Bangladesh. Timely intervention according to CTG results may be the reasons for such variation. Many neonates in abnormal CTG group were not asphyxiated at birth. So CTG can be continued as a good screening test of fetal surveillance but it is not the sole criteria to influence the management of high-risk pregnancies. CTG should be supplemented with other test like biophysical profile and fetal scalp pH before intervention.

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