International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Ray C et al. Int J Reprod Contracept Obstet Gynecol. 2017 Jul;6(7):2745-2752
www.ijrcog.org

pISSN 2320-1770 | eISSN 2320-1789

DOI: http://dx.doi.org/10.18203/2320-1770.ijrcog20172555

Original Research Article

Intrapartum cardiotocography and its correlation with umbilical cord blood pH in term pregnancies: a prospective study

Chandrima Ray*, Alokananda Ray

Department of Obstetrics and Gynecology, Tata Main Hospital, Jamshedpur, Jharkhand, India

Received: 18 May 2017
Accepted: 24 May 2017

***Correspondence:**
Dr. Chandrima Ray,
E-mail: chandrima.ray2012@gmail.com

ABSTRACT

Background: The purpose of intra-partum fetal monitoring is to identify early signs of developing hypoxia. Electronic fetal monitoring is performed using cardiotocograph, which is a paper record of the fetal heart rate (FHR) patterns plotted simultaneously in relation to uterine activity. In low resource settings umbilical cord artery blood gas analysis can provide important information about the foetuses exposed to intrapartum hypoxaemic events and can distinguish the infant at high risk for asphyxia and related sequelae. The aim of this study was to correlate intrapartum CTG findings with umbilical cord blood pH at birth in term pregnancies in labour and thus evaluate the success of CTG in predicting fetal acidosis during labour.

Methods: The present study included 301 consecutive women with term singleton pregnancies in labour. Intrapartum CTG was taken and classified into normal (category I trace), indeterminate (category II trace) and abnormal (category III trace) according to NICHD 2008 Classification (Adapted by ACOG 2013). Umbilical cord arterial blood was taken immediately after birth, in a pre-heparinised syringe and sent to the laboratory for pH study to detect acidosis. Cord blood pH <7.2 was taken as acidosis and cord blood pH ≥7.2 was taken as normal.

Results: In this study, 50.2% of the women had normal CTG, 36.5% had indeterminate CTG and 13.3% had abnormal CTG. 18.3 % of the babies had acidosis. Out of the subjects with abnormal intrapartum CTG, 52.5% had acidosis, of the subjects with normal intrapartum CTG trace, 7.3% had acidosis and of the 110 subjects with indeterminate intrapartum CTG, 22.7% had acidosis. A statistically significant association was found between intrapartum CTG and umbilical cord arterial pH ($p < 0.001$).

Conclusions: From the analysis of this study, it can be concluded that an abnormal CTG should be managed appropriately, without delay, in order to prevent acidosis in the neonate and adverse long-term sequelae. The obstetrician should be more vigilant in cases of indeterminate CTG tracings and monitor such labours closely.

Keywords: Acidosis, Cardiotocography, Cord blood pH, Intrapartum fetal monitoring

INTRODUCTION

Fetal monitoring in labour has witnessed great technological advances: this has partly been driven by increasing number of litigations, majority of them for perinatal deaths pointing to intra-partum causes. Fetal monitoring in labour can be done by intermittent auscultation, electronic fetal monitoring, along with adjuncts to electronic fetal monitoring like fetal scalp stimulation test, fetal scalp blood sampling, fetal pulse oximetry and fetal ST analysis. Electronic fetal monitoring is performed using a cardiotocograph, which is a paper record of the fetal heart rate (FHR) patterns plotted simultaneously in relation to uterine activity. This may be done continuously or intermittently depending upon the facilities available in the labour unit. The purpose of intra-partum fetal monitoring is to identify early signs of developing hypoxia. An essential criterion

to define an acute intrapartum hypoxic event is evidence of metabolic acidosis in intrapartum fetal blood, umbilical cord or very early neonatal blood samples.[1] In low resource settings umbilical cord artery blood gas analysis can provide important information about the foetuses exposed to intrapartum hypoxaemic events and can distinguish the infant at high risk for asphyxia and related sequelae. The aim of this study is to correlate intrapartum CTG findings with umbilical cord blood pH at birth in term pregnancies and thus evaluate the success of CTG in predicting fetal acidosis during labour.

Aim of the study was to correlate intrapartum CTG findings with umbilical cord blood pH at birth in term pregnancies.

METHODS

The present study included 301 consecutive women with term singleton pregnancies in labour, who were admitted for delivery in the labour ward at Tata Main Hospital, which is a 940-bedded teaching hospital, attached to Tata Steel in Jamshedpur, Jharkhand. This hospital-based prospective observational study was conducted over a period of 12 months from 1/11/2014 to 31/10/2015.

Intrapartum CTG tracing was taken and classified into Normal (category I trace), Indeterminate (category II trace) and Abnormal (category III trace) according to NICHD 2008 Classification (Adapted by ACOG 2013).[2,3] Umbilical cord arterial blood was taken immediately after birth, in a pre-heparinised syringe and sent to the laboratory for pH study to detect acidosis. Cord blood pH < 7.2 was interpreted as acidosis and cord blood pH ≥7.2 as normal.

Sample size for the study was calculated as:

$n = Z^2 \times (p) \times (1-p)/\Delta^2$

Where n is the sample size,

Z is confidence interval i.e., 1.96 for 95%

Δ is confidence level i.e., 0.05 for ±5%

p=prevalence of the event in the population as determined by previous studies.

Taking incidence of fetal acidosis in term singleton pregnancies as per previous studies as 19.4%, sample size for my study is calculated to be:

$n = (1.96)^2 \times (0.194) \times (1-0.194)/(0.05)^2 = 240$

For a minimum of 240 women to be included in this study, 301 consecutive women with term singleton gestation in labour were studied

Inclusion criteria

Women with singleton pregnancy at term (gestational age 37-41 weeks), cephalic presentation and in active labour (cervical dilatation ≥4cm) were included in the study.

Exclusion criteria

Pregnant women with PPROM, PROM, multifetal gestation, malpresentations, congenital anomalies, diagnosed FGR, documented abnormal umbilical Doppler study, antepartum hemorrhage, placental abruption, those undergoing elective caesarean section and women refusing to participate in the study were excluded.

Baseline data recording

All consecutive women with term singleton pregnancies in labour, fulfilling the inclusion criteria, admitted to our labour ward, over a period of 1 year, were enrolled in the study, after informed consent. On admission, all patients underwent general, systemic and obstetrical examination including evaluation by ultrasound for placental localisation, assessment of liquor and to rule out any fetal compromise. Labour was monitored. Details regarding patient profile, history, gestational age at onset of labour, mode of onset of labour (spontaneous or induced), cervical dilatation on admission, membranes status, time of rupture of membranes (spontaneous or artificial), nature of liquor after rupture of membranes, CTG tracings, mode of delivery, indications for operative delivery, Apgar score at 1 and 5 minutes, nursery stay, baby details, cord blood pH values were all recorded in study proforma sheets. Intrapartum cardiotocography was done using Philips Avalon FM20 EF Monitor machine. Tracings were taken by the machine at a speed of 3 cm/min for 20 minutes. CTG tracings were taken on admission, at rupture of membranes and during active stage of labour every 2 hours till delivery. In case of abnormal fetal heart rate pattern, tracing was repeated after 1 hour for 40 minutes. Fetal heart rate patterns were interpreted using the NICHD classification (ACOG 2013) as per Table 1.[2,3]

Cord blood collection

Immediately at birth, before the baby's first breath and before delivery of the placenta, the umbilical cord was clamped at two points, 10 cm apart, with Kocher's clamps and cut. The umbilical artery was immediately identified in the cord and 2-3 ml of blood was aspirated with a pre-heparinised syringe. To prevent air contact, the syringe tip was sealed with a plastic cover. The blood was delivered to the laboratory within 5 minutes.[4] Cord blood was analysed by Radiometer ABL 800 Basic Machine used in our institute. The umbilical cord arterial blood pH was used for assessing fetal acidosis. An umbilical artery pH <7.20 was defined as fetal acidosis.[4-6]

Table 1: NICHD classification of CTG tracing (adapted ACOG 2013).

Category	Features	Interpretation
I-Normal	Baseline FHR 110 to 160 bpm	No evidence of fetal hypoxia, normal acid-base status
	Baseline variability moderate (6-25 bpm)	
	Late or variable decelerations absent	
	Early decelerations absent/present	
	Accelerations: present or absent	
II-Indeterminate	Includes all FHR tracings not categorised as I and III:	Uncertain fetal status
	Baseline rate	
	Bradycardia (FHR baseline <110 bpm) not accompanied by absent baseline variability	
	Tachycardia (FHR baseline >160 bpm)	
	Baseline FHR variability	
	Minimal baseline variability (less than or equal to 5 bpm)	
	Absent variability (amplitude range undetectable) without recurrent decelerations	
	Marked variability (>25bpm)	
	Accelerations	
	Absence of induced accelerations fetal stimulation	
	Periodic or episodic decelerations	
	Recurrent variable decelerations with minimal or moderate variability	
	Prolonged deceleration ≥2 minutes but ≤10 minutes	
	Recurrent late decelerations with moderate variability	
	Variable decelerations with slow return to baseline, overshoots or shoulders	
III-Abnormal	**Absent variability and any of the following**	Abnormal acid-base status
	Recurrent late decelerations	
	Recurrent variable decelerations	
	Bradycardia	
	Sinusoidal pattern	

Table 2: Interpretation of correlation.

Correlation coefficient (r)	Interpretation
0 - 0.3	Positive weak correlation
0.3-0.6	Positive moderate correlation
0.6-1.0	Positive strong correlation
0 to (-0.3)	Negative weak correlation
(-0.3) to (-0.6)	Negative moderate correlation
(-0.6) to (-1)	Negative strong correlation

Statistical analysis

Data was entered into Microsoft excel data sheet and analysed using SPSS 22 version software. Categorical data was represented in the form of frequencies and proportions. Chi-square was used as test of significance. Relative Risk or Risk Ratio was calculated to know the risk of abnormal CTG findings with respect to Cord blood pH. P value <0.05 was considered statistically significant.

RESULTS

On performing intrapartum CTG it was observed that 8% had bradycardia, 1.3% had tachycardia, 45.8% had abnormal beat to beat variability. Of the patients who had abnormal beat-to-beat variability, 10.3% had absent beat-to-beat variability, 34.6% had minimal beat-to-beat variability (<5 bpm) and 1% had increased (>25bpm) variability, 54.2% had normal beat to beat variability (6 to 25 bpm). 45.2% of subjects had no accelerations and 17.9% of the subjects had decelerations. Of them, 2.7% had early decelerations, 4% had late decelerations, 2% had prolonged decelerations, 9.3% had variable decelerations. Summarizing, 36.5% of subjects had Indeterminate (category II) CTG and 13.3% had Abnormal (category III) CTG findings. Remaining 50.2% had Normal (category I) CTG. The findings are depicted in Table 3.

Table 3: Baseline Intrapartum CTG Findings.

Individual features of CTG		Frequency	Percent
Intrapartum FHR	FHR 110 to 160 Normal	273	90.7
	< 110 Bradycardia	24	8.0
	> 160 Tachycardia	4	1.3
Beat to beat variability	Absent	31	10.3
	Less than 5 (Min)	104	34.6
	6 to 25 (Moderate)	163	54.2
	>25 (Marked)	3	1
Acceleration	No	136	45.2
	Yes	165	54.8
Deceleration type	None	247	82.1
	Early	8	2.7
	Late	12	4.0
	Prolonged	6	2.0
	Variable	28	9.3
CTG	Normal (Category I)	151	50.2
	Indeterminate (Category II)	110	36.5
	Abnormal (Category III)	40	13.3

In the study 18.3% of neonates had acidosis (defined as cord blood pH <7.2). The mean cord blood pH was 7.253±0.07 (Table 4).

Table 4: Cord blood distribution of neonates.

		Frequency	Percent
Cord blood pH	< 7.2	55	18.3
	≥7.2	246	81.7
	Total	301	100.0
Mean Cord Blood pH		7.253±0.07	
Range		0.61	
Minimum		6.80	
Maximum		7.41	

Forty-four babies (18.3%) had cord blood pH <7.2 and 257 babies (81.7%) had cord blood pH ≥7.2. Out of the 151 mothers with normal CTG trace intrapartum, 11 (7.3%) had acidosis.

Out of the 110 mothers with indeterminate intrapartum CTG, 25 cases (22.7%) had acidosis while 85 (77.3%) had normal umbilical artery blood pH.

Of the 40 mothers with abnormal CTG, 21 (52.5%) had acidosis and 19 (47.5%) babies had normal umbilical artery blood pH. Significant association was found between the type of CTG and umbilical cord artery blood pH ($p<0.001$) as shown in Figure 1.

An abnormal (category III) CTG had a sensitivity of 47.5% and a specificity of 92.72% in detecting acidosis. An abnormal CTG had a positive predictive value of 63.33% and negative predictive value of 86.96%. The overall diagnostic accuracy of an abnormal (category III) CTG in diagnosing fetal acidosis was 83.25%.

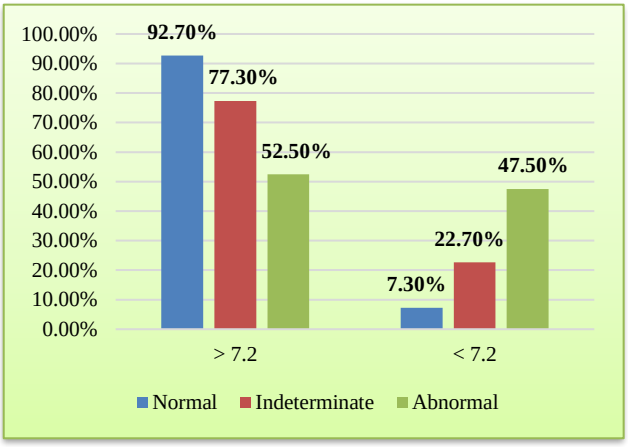

Figure 1: Association between type of CTG and umbilical artery cord blood pH.

Indeterminate (category II) CTG had a sensitivity of 22.73%, specificity of 92.72%, positive predictive value of 69.44%, negative predictive value of 62.22% and overall diagnostic accuracy of 63.22%. When abnormal (category III) and indeterminate (category II) CTGs were considered together for diagnosis of acidosis, it had a higher sensitivity of 80%, specificity of 56.91%, positive predictive value and negative predictive value of 29.33% and 92.72% respectively.

A normal CTG had a sensitivity, specificity, positive predictive value and negative predictive value of 56.91%, 80%, 92.72% and 29.33% respectively (Table 5). When individual features of CTG were studied, it was found that abnormal FHR had 2.86 times higher risk for abnormal cord blood pH and bradycardia had 3 times higher risk for abnormal cord blood pH.

Table 5: Validity of abnormal CTG (category III CTG), indeterminate (category II) CTG and abnormal+indeterminate CTG in diagnosing abnormal cord blood pH (acidosis) and validity of Normal CTG (category III) in diagnosing normal cord blood pH.

Parameter	Abnormal (category III) CTG	Indeterminate (category II) CTG	Abnormal + indeterminate (category III+category II) CTG	Normal (category I) CTG for Normal cord blood pH
Sensitivity	47.5%	22.73%	80%	56.91%
Specificity	92.72%	92.72%	56.91%	80%
Positive predictive value	63.33%	69.44%	29.33%	92.72%
Negative predictive value	86.96%	62.22%	92.72%	29.33%
Diagnostic accuracy	83.25%	63.22%	61.13%	61.13%

Table 6: Association between abnormal cord blood PH and individual components of intrapartum CTG findings.

Individual components of intrapartum CTG findings	Abnormal cord blood PH	RR	95% CI; P value
Baseline FHR			
Abnormal	10/28	2.167	1.233 to 3.809 0.01*
Normal	45/273		
Baseline FHR			
Tachycardia	1/4	1.517	0.2721 to 8.454 NS
Normal	45/273		
Baseline FHR			
Bradycardia	9/24	3.011	1.645 to 5.51 <0.001*
Normal	45/273		
Beat to beat variability			
<5 beats	26/104	0.4844	0.30 to 0.78 0.004*
Normal	16/ 31		
Beat to beat variability			
6 to 25 beats (moderate)	13/ 163	0.154	0.082 to 0.288 <0.001*
Normal	16/31		
Beat to beat variability			
>25 (marked)	0/3	0.276	0.02 to 3.68 0.185
Normal	16/31		
Acceleration			
Present	8/165	0.1403	0.06868, 0.2866 <0.001*
Absent	47/136		
Early decelerations			
Present	1/8	0.908	0.141 to 5.83 0.918
Normal	34/247		
Late decelerations			
Present	3/12	1.816	0.649 to 5.079 0.278
Normal	34/247		
Prolonged decelerations			
Present	2/6	2.42	0.748 to 7.83 0.175
Normal	34/247		
Variable decelerations			
Present	15 / 28	2.534	1.516 to 4.237 <0.001*
Normal	34/247		
CTG			
Indeterminate	25/ 110	3.12	1.604 to 6.068 0.0003*
Normal	11/151		
CTG			
Abnormal	19/ 40	6.52	3.385 to 12.56 <0.001*
Normal	11/151		

Prolonged and variable decelerations had 3.58 and 2.53 times higher risk for abnormal cord blood PH respectively. On the other hand, a normal FHR variability (6 to 25 bpm) had lower risk of abnormal cord blood pH (RR was 0.023). As also presence of accelerations had a lower risk for abnormal cord blood pH (RR=0.1403). Indeterminate CTG and abnormal CTG findings had significantly higher risk for abnormal cord blood PH. These observations were statistically significant (Table 6).

DISCUSSION

In this study 50.2% of the subjects had category I (normal) CTG tracing, 36.5% had category II (indeterminate) CTG tracing and 13.3% had category III (abnormal) intrapartum CTG tracing. 90.7% had normal baseline fetal heart rate, 8.0% had bradycardia and 1.3% had tachycardia. This was comparable to the study by Aboulghar W et al, in this study 82% had normal baseline fetal heart rate.[5] However, the incidence of fetal bradycardia- 8.0% and that of tachycardia-1.3% was different from the study by Aboulghar W which had a higher incidence of fetal tachycardia (17%).[5] This difference can be explained by the fact that the study by Aboulghar W had included cases with PROM, which might have been the cause of fetal tachycardia.[5] In present study, cases with PROM were excluded.

In the present study, 45.8% had abnormal beat-to-beat variability (34.6% cases had minimal variability, 10.3% had absent variability and 1% had increased variability). This value of abnormal FHR variability on CTG is slightly less than that in a study by W Aboulghar[5] et al, where abnormal FHR variability was found to be 55%.[5] In the present study, accelerations on CTG were present in 54.8% cases whereas in the study by W Aboulghar[5] et al, accelerations were present in 6% cases.[5] Decelerations on CTG were present in 17.9% cases in the present study, which is comparable to the study by Modarressnejad V, who studied prospectively 400 consecutive term gestations, where 14.25% of the subjects had decelerations.[7] In the study by Aboulghar W et al, decelerations were present in 53% cases.[5] This difference could be due to the fact that in the present study consecutive women in labour have been included, whereas Aboulghar W et al studied 100 women who underwent caesarean section for pathological or suspicious CTG.[5]

In the present study, mean cord blood pH was 7.253 ± 0.07. This is comparable to several studies.[4-7] Kaban et al used the cut off value for defining acidosis as cord blood pH <7.2 in their study.[4] Modarressnejad V defined fetal acidosis as cord blood pH <7.1.[7] In the present study, an umbilical arterial cord blood pH of <7.2 at birth is taken as the cut-off value to define acidosis in the neonate. 18.3% of the neonates had acidosis which is comparable to the studies by Kaban[4] et al (13.26%) and Modarressnejad V (20.25%).[4,7] In the study by Aboulghar W et al, incidence of acidosis was higher, with 34% of babies having abnormal cord blood pH. This higher value of acidosis in the neonates could be explained by the fact that their study included only those women who had undergone caesarean section for pathological and suspicious CTG, while in the other studies mentioned above and in our study, consecutive term labouring women were included.

Jackson et al studied the intrapartum fetal heart rate (FHR) characteristics of over 48,000 patients with a singleton, non-anomalous fetus in term labor at 10 hospitals.[8] In their study, considering all of labor, the FHR pattern was category I in 77.9 percent of the time, category II 22.1 percent of the time and category III 0.004 percent of the time. In the two hours before delivery, category I tracings were less commonly observed (60.9%) and both category II and category III tracings became more common (39.1% and 0.006%, respectively). They concluded that category I and II fetal heart rate patterns are more common in labour than category III. Results from the present study are comparable with this finding.

In this study, 52.5% of the cases with category III (abnormal) CTG had acidosis and 22.7% cases with category II (indeterminate) CTG had acidosis (Figure 2). Only 7.3% of the subjects with category I (normal) CTG had acidosis. A significant association was found between the type of CTG and cord blood pH. Also, there was an increased risk of having abnormal cord blood pH with an abnormal CTG. These values are comparable with the study by Aboulghar W et al, where 50% of cases with pathological CTG had acidosis and 19.2% of cases with suspicious CTG had acidosis.[5] They also found that pathological (rather than suspicious) CTG significantly increased the risk of abnormal cord blood pH. Findings of the present study are also comparable to study by Kaban et al who studied 101 term pregnant women admitted for delivery.[4] In their study 85 neonates had normal cord arterial pH and 13 had fetal acidosis as diagnosed by cord arterial pH values <7.2. Of the 13 neonates with acidosis, 5 had non-reactive CTG tracings intrapartum. All the 85 neonates without acidosis had reactive CTG tracings.

In the present study, it was found that abnormal FHR had 2.86 times higher risk for abnormal cord blood pH and bradycardia had 3.011 times higher risk for abnormal cord blood pH. There was significantly lower risk of having an abnormal cord blood pH in cases where CTG showed normal beat-to-beat variability (between 6-25 bpm). Prolonged and variable decelerations had higher risks for abnormal cord blood pH. Indeterminate and abnormal CTG had significantly higher risk for abnormal cord blood pH (Table 6). In the study by Aboulghar W et al, analysis of different features of CTG as predictor of abnormal cord blood pH (<7.2) showed that bradycardia significantly increased the risk for abnormal cord blood pH by almost 3 times, which is similar to this study.[5] They also found that abnormal beat-to beat variability,

late deceleration and variable deceleration significantly increased the risk of abnormal cord blood pH. They concluded that pathological (rather than suspicious) CTG significantly increased the risk of abnormal cord blood pH. Findings of this study are comparable to theirs.

An abnormal (category III) CTG had sensitivity of 47.5% and specificity of 92.72% in detecting acidosis (Table 5), which meant that while an abnormal (category III) CTG tracing could detect 47.5% of subjects with acidosis, it had a good ability to identify those who did not have acidosis because of its high specificity. It was also found that an abnormal CTG had a positive predictive value of 63.33% and negative predictive value of 86.96%, which meant that in absence of an abnormal CTG, the chance of having acidosis was very little. The overall diagnostic accuracy of an abnormal (category III) CTG in diagnosing fetal acidosis was 83.25%. This study also found that an indeterminate (category II) CTG had sensitivity of 22.73% and specificity of 92.72% in detecting acidosis, which meant that an indeterminate CTG could diagnose only 22.72% of subjects who had acidosis. However, from its high specificity, it also had a good ability to identify those who did not have acidosis.

However, when abnormal (category III) and indeterminate (category II) CTG were taken together to diagnose acidosis, they were shown to have a higher specificity of 80% in detecting acidosis. Specificity of both abnormal and indeterminate CTG in detecting acidosis was 56.91%. Positive predictive value and negative predictive value of abnormal+indeterminate CTG were 29.33% and 92.72% respectively. It meant that in the absence of an abnormal or an indeterminate CTG, there was very less chance of having a fetus with acidosis. The present study also found that a normal CTG had a sensitivity, specificity, positive predictive value and negative predictive value of 56.91%, 80%, 92.72% and 29.33% respectively, for diagnosing normal cord blood pH. This meant that a normal CTG can accurately detect babies without acidosis, that is, in presence of a normal CTG, there was minor possibility of fetus having acidosis. These findings are comparable with those found by Parveen who concluded that a normal CTG trace correlates highly with absence of fetal acidosis.[9] Parveen studied 122 cord blood samples were using umbilical cord arterial base excess (>12mmol/l) at birth to diagnose fetal acidemia.[9] From their study they found that cardiotocography has a sensitivity of 15.38%, specificity of 86%, positive predictive value 11.76% and a negative predictive value 89%. This difference may be because of the fact that they took umbilical artery base excess at birth to diagnose fetal acidosis, while in the present study, umbilical cord arterial pH value of <7.2 was taken to diagnose fetal acidosis.

Visser et al who studied normal and abnormal patterns of CTG tracings and correlated CTG findings with fetal distress after birth by umbilical cord blood gas analysis and found sensitivity, specificity, positive predictive value and negative predictive value of CTG to be 79%, 85%, 68% and 91% respectively.[10] Steer PJ et al conducted a prospective study to find correlation among FHR patterns, MSL, umbilical cord arterial blood pH and Apgar score in 698 cases and found that sensitivity of an abnormal CTG at any time for fetal acidosis (cord arterial pH < 7.17) was 80% and for severe acidosis (pH<7.08) was 83%.[11] However positive predictive value was low: 32% foetuses had abnormal CTG but no acidosis. If only CTG abnormality in 1st stage labour was considered sensitivity was 47% for acidosis & 67% for severe acidosis and false positive was 14%. In a study by Tasnim et al, positive predictive value of CTG was 18% for fetal hypoxia, 21% for fetal hypercarbia, 26% for fetal acidosis and 37% for base excess.[12] Predictive value of suspicious trace for similar blood indices was 13%, 13%, 17% and 35% respectively. For pathological trace, predictive value was 50%, 83 %, 100% and 66% and respectively. They concluded that suspicious CTG trace has low predictive value in terms of fetal acid base status at birth and needs to be complemented with other diagnostic modalities before undertaking any operative intervention. Pathological CTG on the other hand is highly predictive of fetal acidosis at birth warranting immediate intervention.

Thus, from the findings of the present study, although the sensitivity of CTG was found to be low, its high specificity, low cost and ease of carrying out the monitoring supported its use in intrapartum fetal monitoring and in alerting the obstetrician regarding an intra-uterine hypoxic event.

CONCLUSION

The fetus undergoes physiological stress during labour. Fetal morbidity and mortality may occur as a consequence of labour even in low risk patients. Cardiotocography is a simple, noninvasive recordable method of intrapartum fetal monitoring which can be used as a tool to detect hypoxaemic event in the fetus-in-utero during labour, enabling initiation of appropriate management. We studied the correlation between intrapartum CTG in term labouring women with cord blood pH in their neonates in order to predict acidosis in the fetus. From the findings of this study, we can conclude that in presence of an abnormal or indeterminate CTG, there is higher possibility of intrapartum fetal acidosis while presence of a normal CTG indicates a minor possibility of intrapartum fetal acidosis. There is high risk of acidosis when findings of bradycardia, loss of beat-to-beat variability, prolonged and variable decelerations were present in CTG during labour. Presence of a normal beat-to-beat variability (between 6-25 bpm) is reassuring.

Thus, CTG is a simple test, easy to perform and can alert the obstetrician for necessary interventions in case of an abnormal CTG. It can detect fetal distress in labour, thus helping to reduce neonatal morbidity by early

intervention in cases of abnormal tracings. From the analysis of this study, it can be concluded that an abnormal CTG should be managed appropriately, without any delay, in order to prevent acidosis in the neonate and adverse long-term sequelae. The obstetrician should be more vigilant in cases of indeterminate CTG tracings and monitor such labours closely.

ACKNOWLEDGMENTS

Authors would like to thank Dr. Manas Kabiraj, Senior Specialist and Head of the Department and Dr Alokananda Ray, Senior Consultant, Department of Obstetrics and Gynaecology, Tata Main Hospital, Jamshedpur for their constant guidance and constructive advice during this work.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. MacLennan A. A template for defining a causal relationship between acute intrapartum events and cerebral palsy. In: Bonnar J, ed. By. Recent advances in Obstet Gynaecol. Churchill Livivngstone; 2001(21):86-93.
2. Macones G, Hankins G, Spong C, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation and research guidelines. Obstet Gynecol. 2008;112(3):661.
3. Macones GA. Intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. Obstet Gynecol. 2009;114(1):192-202.
4. Kaban A, Cengiz H, Kaban I, Özcan A, Karakaş S. The success of cardiotocography in predicting perinatal outcome. J Clini Experiment Investigations. 2012;3(2):168-171.
5. Aboulghar WM, Ibrahim MA, Allam IS, Hosny W, Otify M. Validity of cardiotocography in the diagnosis of acute fetal hypoxia in low resources settings. Internet J Gynecol Obstet. 2013;17(1):1-8.
6. Yeh P, Emary K, Impey L. The relationship between umbilical cord arterial pH and serious adverse neonatal outcome: analysis of 51519 consecutive validated samples. BJOG. 2012;119:824-831
7. Modarressnejad V. Umbilical cord blood pH and risk factors for acidaemia in neonates. Eastern Mediterranean Health J. 2005;11(1/2):96-101.
8. Jackson M, Holmgren CM, Esplin MS, Henry E, Varner MW. Frequency of fetal heart rate categories and short term neonatal outcome. Obstet Gynecol. 2011;118(4):803-8.
9. Parveen S. Umbilical cord arterial blood base excess as gold standard for fetal wellbeing screening test at term delivery. JMPA. 2010;60(5):347-350.
10. Visser GHA, Sadovsky G, Nicolaides KH. Antepartum fetal heart rate patterns in small for gestational age third trimester foetuses: correlations with blood gas values obtained at cordocentesis. Am J Obstet Gynecol. 1990;162(3):698-703.
11. Steer PJ, Eigbe F, Lissauer TJ, Beard RW. Interrelationships among abnormal cardiotocograms in labor, meconium staining of the amniotic fluid, arterial cord blood pH and Apgar scores. Obstet Gynecol. 1989;74(5):715-21.
12. Tasnim N, Mahmud G, Akram S. Predictive accuracy of intrapartum cardiotocography in terms of fetal acid base status at birth. J Coll Physicians Surg Pak. 2001;19(10):632-5.

Cite this article as: Ray C, Ray A. Intrapartum cardiotocography and its correlation with umbilical cord blood pH in term pregnancies: a prospective study. Int J Reprod Contracept Obstet Gynecol 2017;6:2745-52.