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Diagnostic Accuracy of Fetal Heart Rate Monitoring in the Identification of Neonatal Encephalopathy

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Abstract

Objective—To estimate the diagnostic accuracy of electronic fetal heart rate abnormalities in the identification of neonates with encephalopathy treated with whole-body hypothermia.

Methods—Between January 1, 2007, and July 1, 2013, there were 39 neonates born at two hospitals within our system treated with whole-body hypothermia within 6 hours of birth. Neurologically normal controls were matched to each case by gestational age and mode of delivery in a 2:1 fashion. The last hour of electronic fetal heart rate monitoring before delivery was evaluated by three obstetricians blinded to outcome.

Results—The differences in tracing category were not significantly different (cases 10.3% I, 76.9% II, 12.8% III; controls 9.0% I, 89.7% II, 1.3% III, $p=0.18$). Bivariate analysis showed cases had significantly increased late decelerations, total deceleration area 30 (debt 30) and 60 minutes (debt 60) prior to delivery and were more likely to be nonreactive. Multivariable logistic regression showed cases had a significant decrease in early decelerations ($P=0.03$) and a significant increase in debt 30 (0.01) and debt 60 ($P=0.005$). The area under the ROC curve, sensitivity and specificity were 0.72, 23.1%, 94.9% for early decelerations; 0.66, 33.3% and 87.2% for debt 30 and 0.68, 35.9% and 89.7% for debt 60.

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Conclusion—Abnormalities during the last hour of fetal heart rate monitoring before delivery are poorly predictive of neonatal hypoxic-ischemic encephalopathy qualifying for whole-body hypothermia treatment within 6 hours of birth.

Introduction

The Perinatal Quality Foundation has created an examination containing both knowledge-based and judgment questions in order to optimize and standardize the interpretation of electronic fetal monitoring (EFM).⁽¹⁾ Recognition of the importance of EFM interpretation led to this requirement in 2005 that all caregivers in the hospitals insured by the Medical Center Insurance Company (MCIC) pass this EFM credentialing examination before being allowed to work on any of the labor and delivery floors in their network and undergo recertification every 3 years.⁽¹⁾ It was hoped that having labor and delivery staff more rapidly identify abnormalities indicative of metabolic acidemia on intrapartum EFM tracings and communicate these findings among team members would decrease the incidence of hypoxic-ischemic encephalopathy (HIE) and related litigation. The 2012 ACOG survey on professional liability found that neurologically impaired infant claims were the most common primary allegation of an obstetric claim occurring in 28.8%.⁽²⁾

Meta-analysis of randomized controlled trials comparing EFM to intermittent auscultation has failed to show that EFM decreases neurologic morbidity or mortality⁽³⁾; however, the combined sample size of 12 randomized controlled trials has been criticized as insufficient to evaluate whether EFM can significantly lower neonatal morbidity and mortality.⁽⁴⁾

Although some investigators have concluded that because of the low prevalence of the target conditions and mediocre validity (ability to distinguish between those diseased and well) that the positive predictive value of EFM for fetal death in labor or cerebral palsy is near zero,⁽⁵⁾ researchers using the U.S. 2004 linked birth and infant death data found that 89% of singleton pregnancies had EFM, and that the use of EFM was associated with a substantial decrease in early neonatal mortality and morbidity that lowered infant mortality.⁽⁴⁾ EFM is premised on the assumption that abnormalities indicative of severe metabolic acidosis leading to HIE should be present in the tracing prior to delivery. Our objective in this study is to estimate the diagnostic accuracy of human assessment of electronic fetal heart rate tracings during the hour prior to delivery to identify abnormalities associated HIE) qualifying for whole-body hypothermia treatment.

Materials and Methods

This is a case control study of all neonates born at 2 hospitals within our system with suspected HIE treated with whole-body hypothermia within 6 hours of birth during the 6.5 year period from January 1, 2007 to July 1, 2013. Controls were matched to each case in a 2:1 fashion using the subsequent 2 deliveries in the same hospital matched by gestational age within 1 week and mode of delivery. This study was conducted using the standards for reporting of diagnostic accuracy.⁽⁶⁾ It was approved by the institutional review board of the Johns Hopkins School of Medicine. Neonates were eligible for treatment with whole-body hypothermia if moderate-severe encephalopathy⁽⁷⁾ was present at birth (manifested as

lethargy, stupor, coma, decreased or no activity, distal flexion, complete extension, decerebrate posture, hypotonia or flaccidity, abnormal primitive reflexes, bradycardia, periodic breathing, apnea or seizures) and had a cord gas or early neonatal gas at < 1 hour with pH < 7.0 or base deficit > 16 mM. They were also eligible if the cord or early neonatal gas at < 1 hour showed pH 7.01–7.15 and base deficit 10–15.9 mM if moderate-severe encephalopathy was present with evidence of an acute sentinel event (8), 10 min Apgar < 5 or there was need for assisted ventilation initiated at birth with continuation for at least 10 minutes. It is the policy within the two hospitals to obtain umbilical artery cord gases at all deliveries, and the number of neonates with cord pH < 7.0 or base deficit > 12 mM were recorded. Exclusion criteria for whole-body hypothermia treatment included greater than 6 hours of life, gestational age < 35 weeks, severe growth restriction (birth weight < 1800 grams), major congenital anomaly, severe persistent pulmonary hypertension with anticipated need for extracorporeal membrane oxygenation, coagulopathy with active bleeding, and suspected sepsis with severe hemodynamic compromise requiring large doses of pressors.

Infant and maternal medical records were reviewed to identify relevant clinical data. Intrauterine growth restriction was defined as an estimated fetal weight less than the 10th percentile.(9) Oligohydramnios was defined as an amniotic fluid index (AFI) < 5.0 cm with intact membranes at the time of the admission in which delivery occurred. The clinical diagnosis of chorioamnionitis was made in the presence of maternal fever, with at least one other finding of fetal tachycardia, uterine tenderness, or purulent vaginal discharge.

The two hospitals employ universal continuous EFM during labor which are stored electronically. The primary exposure of the study was the noncomputer-assisted interpretation of the last hour of tracing prior to delivery which was reviewed independently by 3 obstetricians blinded to outcome using the National Institute of Child Health and Human Development (NICHD) and the American College of Obstetricians and Gynecologists (ACOG) 3-tiered category system and definitions.(10) The reviewers were an obstetric resident (RA) and 2 maternal-fetal medicine attendings (FW, EG), all of whom had passed our required EFM course. Each reviewer assessed the last hour of tracing and assigned category based on the most nonreassuring portion of the tracing, and the final category was assigned based on consensus among the reviewers. Each reviewer recorded the baseline fetal heart rate (FHR), time with FHR > 160 beats/minutes (bpm) (tachycardia) or < 110 bpm (bradycardia), number of accelerations, reactivity, total number of decelerations, and number of late, variable, or early decelerations.(10) Reactivity was defined as the presence of at least 2 FHR accelerations that peak (but do not necessarily remain) at least 15 bpm above the baseline and last 15 seconds within a 20 minute period that occurred any time during the last hour prior to delivery. Variability was classified as absent (undetectable), minimal (amplitude range < 5 bpm), moderate (amplitude range from 6 to 25 bpm), and marked (amplitude range > 25 bpm).(10) Absent and minimal were considered decreased variability. The number of prolonged decelerations lasting 2–10 minutes was recorded, as well as the nadir and length of the most severe prolonged deceleration. Since some have hypothesized that non-NICHD measures of fetal heart rate decelerations immediately prior to delivery, which account for properties such as depth, duration and frequency, would have a greater predictive ability for acidemia compared with the NICHD

category system(11) we performed a review of the literature to identify other clinician determined fetal heart rate parameters. Human fetal acidosis has been shown to correlate with severe variable decelerations, though not with mild or moderate variables.(12) Severe variable decelerations were those with a drop to < 70 bpm or lasting > 60 seconds.(12) The number of contractions in the last hour prior to delivery were counted, and the ratio of late decelerations/contractions and variable decelerations/contractions were expressed as a percentage. Total deceleration area was calculated as the sum of the area within all decelerations in the final 30 minutes (debt 30) and final 60 minutes (debt 60) of the tracing as a measure of both quantity and severity.(11) The area within each deceleration was approximated as $\frac{1}{2}$ (width in seconds x depth in beats/minute).(11) Other non-NICHD fetal heart rate tracing characteristics such as shoulders, slow return and variability within the deceleration during the 30 minutes prior to delivery have not been shown to be associated with acidemia or neonatal depression and were not included.(13).

Categorical EFM tracing parameters were determined by consensus among the 3 reviewers, and continuous parameters were averaged. We calculated kappa statistics to assess the inter-observer reliability in classifying the categorical EFM parameters reactivity, variability and category. Predefined criteria for agreement were: poor (kappa 0.0–0.20), fair (0.21–0.40), moderate (0.41–0.60), substantial (0.61–0.80) and excellent (0.81–1.0).(14) We calculated the Pearson correlation coefficient to assess the inter-observer reliability in classifying continuous EFM parameters such as fetal heart rate baseline, accelerations, decelerations and debt time.

Bivariate analyses were performed using conditional logistic regression to account for matching for both continuous and categorical variables. Categorical variables with multiple components such as race were compared using chi square. Multivariable logistic regression models were used to determine the diagnostic accuracy of EFM parameters in the identification of neonates with encephalopathy treated with whole-body hypothermia. Variables significant at a P value of less than 0.10 in bivariate analyses were utilized in the multivariable regression. Final variable selection in the multivariable regression was determined by statistical significance as defined by a confidence interval that did not include 1.0 and a P value less than 0.05. For each significant final variable in the multivariable analysis, receiver operator characteristic (ROC) curves were produced and sensitivity and specificity calculated. Stata version 10 (StataCorp LP, College Station, TX) was used for statistical analysis.

Results

During the 6.5 year study period, 39 infants were treated with whole-body cooling ((1 case/521 deliveries 35 weeks). There were no meaningful differences in maternal demographics between the cases and controls.(Table 1) Sentinel events occurred in 13 (33.3%) of the cases and none of the controls (<0.001). There was no difference in intrapartum oxytocin exposure. The cases had a significantly higher incidence of clinical chorioamnionitis, but there was no difference in histologic chorioamnionitis or funisitis(Table 1).

Cases were significantly more likely to have 1 and 5 minute Apgars < 7, and significantly lower cord pH and higher base deficit (Table 2). Cases were significantly more likely to have respiratory distress, seizures and a longer length of stay, as well as a significantly higher incidence of positive blood cultures in the neonatal period (Table 2).

The kappa coefficient for the 3 reviewers for categorical variables ranged from 0.48 for reactivity to 0.37 for short term variability, indicating moderate to fair reproducibility (Table 3). The Pearson correlation coefficient for the 3 reviewers for continuous variables ranged from 0.94 for fetal heart rate baseline to 0.28 for severe variables. (Table 3) The Pearson correlation coefficient for total number of decelerations between the two MFM attendings was 0.67 and between the resident and each attending was 0.82 and 0.81. For late decelerations, the Pearson correlation coefficient between the two MFM attendings was 0.67 and between the resident and each attending was 0.76 and 0.64. There was no statistically significant difference in tracing category between the cases and controls with 77% of cases and 90% of controls being category II (Table 4). The cases were significantly more likely to be nonreactive during the hour prior to delivery. Of the 16 cases (41.0%) that were reactive during the hour prior to delivery, 8 had sentinel events. There was no difference in the number of accelerations or total decelerations, but the cases had a significantly increased number of late decelerations during the hour prior to delivery. There was no difference in variable decelerations/contraction or late decelerations/contraction. The measurement of total deceleration area debt 30 and debt 60 were significantly increased in the cases (Table 4).

Multivariable logistic regression was performed for all fetal heart rate tracing parameters with $P < 0.10$ on bivariate analysis, adjusting for clinical chorioamnionitis which was significantly increased in the cases. There was a significantly increased odds ratio for decreased early decelerations and increased debt 30 and debt 60 in cases (Table 5). For the detection of cases using significant variables from the multivariable logistic regression, the area under the ROC curve ranged from 0.66–0.72 and the sensitivity from 23.1–35.9% (Table 6).

Discussion

Although we had hoped to be able to identify specific FHR abnormalities associated with neonatal HIE and focus on these in our team training in order to decrease the incidence of this catastrophic complication, we were unable to identify such changes. Our study found a significantly increased rate of nonreactive fetal heart rate tracings and late decelerations among fetuses diagnosed with HIE as infants on bivariate analysis, but these differences were not significant in multivariable analysis. The measure of total deceleration area in the 30 minutes (debt 30) and 60 minutes (debt 60) prior to delivery were significantly increased in encephalopathic fetuses on bivariate and multivariable analysis, but due to the high incidence of decelerations in normal babies their sensitivity and specificity in detecting neurologic injury was too low to be clinically useful. Multivariable analysis also showed a significant decrease in early decelerations in encephalopathic neonates, but this change also had a sensitivity and specificity too low to be useful in detecting injury in the general obstetric population.

Retrospective observational studies on the effects of EFM on decreasing HIE are limited since tracing abnormalities may prompt intervention prior to the deterioration to metabolic acidosis and result in a metabolically normal newborn. (15) This “treatment paradox effect” can occur where an outcome (neonatal HIE) with a known association with the test predictor (EFM abnormalities) can be ameliorated or avoided by an intervention,(16) but if early intervention based on EFM abnormalities leads to a decreased incidence of HIE we should be able to describe and quantitate these abnormalities when comparing encephalopathic neonates to neurologically normal controls.

Our finding that only 12.8% of these encephalopathic neonates had category III tracings in the hour prior to delivery is similar to the rarity of severely abnormal tracings in other studies. Computerized software using the 5-tier color coded levels comparing the last 3 hours prior to delivery of 60 fetuses that developed encephalopathy to 280 with metabolic acidosis but without encephalopathy and 2132 fetuses with normal cord gases found that only 8.3% of babies born with severe metabolic acidosis and encephalopathy ever reached the red level.(15) Even when the red level EFM tracing occurred, its average cumulative duration was similar and very short ranging from 4.7–6.3 minutes in the 3 study groups. The area under the curve for red level EFM to identify encephalopathy was 0.53 which is no better than chance. High sensitivity for the encephalopathic babies was achieved at the expense of a high false-positive rate in the normal babies. EFM patterns that detected around 75% of the encephalopathic babies were also present in 29% of the normal babies.(15) In a study of 154 fetal heart rate tracings evaluated by 3 maternal-fetal medicine specialists using the NICHD 3-tier system, category III was rare occurring in only 1.9%, and interobserver reliability for category III was poor (kappa 0.0) under idealized study conditions mainly due to lack of agreement regarding absent versus minimal variability.(14) Review of the entire intrapartum EFM tracing for 48,444 patients found that category III was identified in only 0.1% of patients (1 in every 897).(17) Category II tracings were very common occurring in 84% of labors which was very similar to our finding of 77% of encephalopathic babies and 90% of controls.

A 1997 confidential inquiry into all cases of neonates with grade II and III encephalopathy born in Trent, UK concluded that there was evidence of a peripartum insult in 88%.(18) The investigators found a major episode of suboptimal care in 64% of all cases of neonatal encephalopathy and in 75% of the deaths, and concluded that incorrect EFM interpretation led the list of causes.(18) If it is true that there is now reassuring evidence for the use of EFM in that its use is linked with long-term improvement, such as a significant decrease in neonatal and infant morbidity and mortality, (4) then there should be identifiable fetal heart rate patterns associated with neonatal encephalopathy which the obstetrician can quantitate. However, studies have shown that the false positive rate of EFM for predicting cerebral palsy is high at greater than 99%,(19) and that the use of EFM has not resulted in a reduction of cerebral palsy.(20)

When investigators have found an association between category II EFM in the last 2 hours of labor and short-term neonatal morbidity they were not able to describe the association of specific components of category II features and morbidity.(17) The overall EFM pattern during the last 30 minutes before delivery is universally overall category II,(11) and more

than 98% of term fetuses with terminal decelerations deliver with normal umbilical cord gas pH levels.(21)

This study began as a safety project by resident and attending obstetricians who after passing the required course on EFM would use this knowledge to identify and quantitate EFM abnormalities in the last hour of labor in encephalopathic neonates. If such changes could be identified we hoped to decrease the incidence of HIE in our unit by focusing on these changes. Unfortunately, although we found some EFM abnormalities that were significantly different when compared to neurologically normal controls, the high incidence of these abnormalities in normal neonates and the low incidence of HIE shows they would not be able to identify encephalopathic neonates. EFM is not a precision technology, and EFM generates many patterns that are not easily fitted into defined categories by obstetricians even after specific training. Although we are unable to measure predictive values in a case control study, our findings of low sensitivity for any of the EFM abnormalities are consistent with the study by Grimes and Peipert who reported that the positive predictive value of EFM for detecting HIE is near zero.(5) The high frequency of EFM abnormalities in normal babies, which coupled with the low prevalence of HIE of 1.0–3.8/1000 term births,(22–24) do not allow us to quantify specific abnormalities during the last hour before delivery that have precision in identifying neonatal encephalopathy qualifying for treatment with hypothermia within 6 hours of birth.

References

1. Berkowitz RL, D'Alton ME, Goldberg JD, O'Keeffe DF, Spitz J, Depp R, et al. The case for an electronic fetal heart rate monitoring credentialing examination. *Am J Obstet Gynecol.* 2014; 210:204–7. [PubMed: 24113255]
2. Klagholz, J.; Strunk, AL. Overview of the 2012 ACOG survey on professional liability. Washington D.C: The American Congress of Obstetricians and Gynecologists; 2012. [cited 5/14/2014]. Available from: <http://www.acog.org/~media/Departments/Professional%20Liability/2012PLSurveyNational.pdf>
3. Alfievic Z, Devane D, Gyte GM. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. *Cochrane Database Syst Rev.* 2013; 5:CD006066. [PubMed: 23728657]
4. Chen HY, Chauhan SP, Ananth CV, Vintzileos AM, Abuhamad AZ. Electronic fetal heart rate monitoring and its relationship to neonatal and infant mortality in the United States. *Am J Obstet Gynecol.* 2011; 204:491, e1,491.10. [PubMed: 21752753]
5. Grimes DA, Peipert JF. Electronic fetal monitoring as a public health screening program: the arithmetic of failure. *Obstet Gynecol.* 2010; 116:1397–400. [PubMed: 21099609]
6. Bossuyt PM, Reitsma JB, Bruns DE, Gatsonis CA, Glasziou PP, Irwig LM, et al. The STARD statement for reporting studies of diagnostic accuracy: explanation and elaboration. The Standards for Reporting of Diagnostic Accuracy Group. *Croat Med J.* 2003; 44:639–50. [PubMed: 14515429]
7. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. *Arch Neurol.* 1976; 33:696–705. [PubMed: 987769]
8. American College of Obstetricians and Gynecologists, American Academy of Pediatrics. Neonatal encephalopathy and neurologic outcome. 2. Washington DC: American College of Obstetricians and Gynecologists; 2014.
9. Hadlock FP, Harrist RB, Martinez-Poyer J. In utero analysis of fetal growth: a sonographic weight standard. *Radiology.* 1991; 181:129–33. [PubMed: 1887021]

10. American College of Obstetricians & Gynecologists. Intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. ACOG Practice Bulletin No 106. *Obstet Gynecol.* 2009; 114:192–202. [PubMed: 19546798]
11. Cahill AG, Roehl KA, Odibo AO, Macones GA. Association and prediction of neonatal acidemia. *Am J Obstet Gynecol.* 2012; 207:206, e1,206.e8. [PubMed: 22939728]
12. Kubli FW, Hon EH, Khazin AF, Takemura H. Observations on heart rate and pH in the human fetus during labor. *Am J Obstet Gynecol.* 1969; 104:1190–206. [PubMed: 5807980]
13. Cahill AG, Roehl KA, Odibo AO, Macones GA. Association of atypical decelerations with acidemia. *Obstet Gynecol.* 2012; 120:1387–93. [PubMed: 23168764]
14. Blackwell SC, Grobman WA, Antoniewicz L, Hutchinson M, Gyamfi Bannerman C. Interobserver and intraobserver reliability of the NICHD 3-Tier Fetal Heart Rate Interpretation System. *Am J Obstet Gynecol.* 2011; 205:378, e1,378.e5. [PubMed: 21864826]
15. Elliott C, Warrick PA, Graham E, Hamilton EF. Graded classification of fetal heart rate tracings: association with neonatal metabolic acidosis and neurologic morbidity. *Am J Obstet Gynecol.* 2010; 202:258, e1,258.e8. [PubMed: 19716539]
16. Maso G, Businelli C, Piccoli M, Montico M, De Seta F, Sartore A, et al. The clinical interpretation and significance of electronic fetal heart rate patterns 2 h before delivery: an institutional observational study. *Arch Gynecol Obstet.* 2012; 286:1153–9. [PubMed: 22791414]
17. Jackson M, Holmgren CM, Esplin MS, Henry E, Varner MW. Frequency of fetal heart rate categories and short-term neonatal outcome. *Obstet Gynecol.* 2011; 118:803–8. [PubMed: 21897312]
18. Draper ES, Kurinczuk JJ, Lamming CR, Clarke M, James D, Field D. A confidential enquiry into cases of neonatal encephalopathy. *Arch Dis Child Fetal Neonatal Ed.* 2002; 87:F176–80. [PubMed: 12390986]
19. Nelson KB, Dambrosia JM, Ting TY, Grether JK. Uncertain value of electronic fetal monitoring in predicting cerebral palsy. *N Engl J Med.* 1996; 334:613–8. [PubMed: 8592523]
20. Clark SL, Hankins GD. Temporal and demographic trends in cerebral palsy--fact and fiction. *Am J Obstet Gynecol.* 2003; 188:628–33. [PubMed: 12634632]
21. Cahill AG, Caughey AB, Roehl KA, Odibo AO, Macones GA. Terminal fetal heart decelerations and neonatal outcomes. *Obstet Gynecol.* 2013; 122:1070–6. [PubMed: 24104779]
22. Low JA, Lindsay BG, Derrick EJ. Threshold of metabolic acidosis associated with newborn complications. *Am J Obstet Gynecol.* 1997; 177:1391–4. [PubMed: 9423740]
23. Graham EM, Ruis KA, Hartman AL, Northington FJ, Fox HE. A systematic review of the role of intrapartum hypoxia-ischemia in the causation of neonatal encephalopathy. *Am J Obstet Gynecol.* 2008; 199:587–95. [PubMed: 19084096]
24. Badawi N, Kurinczuk JJ, Keogh JM, Alessandri LM, O'Sullivan F, Burton PR, et al. Intrapartum risk factors for newborn encephalopathy: the Western Australian case-control study. *BMJ.* 1998; 317:1554–8. [PubMed: 9836653]

Table 1

Maternal variables.

	Cases n=39	Controls n=78	P value
Maternal Age, years [†]	27.2±7.7	27.4±6.2	0.88
Nulliparous	24 (61.5%)	39 (50%)	0.24
Race			0.20
Caucasian	11 (28.2%)	18 (23.1%)	
African-American	23 (59.0%)	38 (48.7%)	
Hispanic	5 (12.8%)	16 (20.5%)	
Other	0	6 (7.7%)	
Oxytocin	23 (59.0%)	45 (57.7%)	0.89
Preeclampsia	5 (12.8%)	7 (9.0%)	0.56
Intrauterine growth restriction	3 (7.7%)	2 (2.6%)	0.24
Oligohydramnios	2 (5.1%)	8 (10.3%)	0.39
Clinical chorioamnionitis	10 (25.6%)	5 (6.4%)	0.01*
Abruption	1 (2.6%)	2 (2.6%)	1.0
Nonreassuring fetal heart rate	21 (53.8%)	26 (33.3%)	0.025*
Meconium	16 (41.0%)	13 (16.7%)	0.01*
Histologic chorioamnionitis	11/32 (34.4%)	9/35 (25.7%)	0.67
Histologic funisitis	6/32 (18.8%)	4/35 (11.4%)	0.90
Histologic placental infarcts	6/32 (18.8%)	2/35 (5.7%)	0.09

* indicates P < 0.05

[†] (mean ± standard deviation)

Table 2

Neonatal variables

	Cases n=39	Controls n=78	P value
Birth weight (grams)	3303±500	3163±466	0.09
Male gender	26 (66.7%)	41 (52.6%)	0.20
1 min Apgar < 7	37 (94.9%)	15 (19.2%)	<0.001*
5 min Apgar < 7	29 (74.4%)	5 (6.4%)	<0.001*
Cord pH	6.99±0.12	7.24±0.07	<0.0001*
Cord base deficit (mM)	14.1±4.4	4.0±3.0	<0.0001*
pH<7.0 or Base deficit>12 mM	25/36 (69.4%)	0	<0.0001*
Respiratory distress	15 (38.5%)	3 (3.8%)	0.001*
+ Blood cultures	3 (7.7%)	0	0.035*
+ Cerebrospinal fluid cultures	0	0	
Seizures	8 (20.5%)	0	<0.001*
Length of stay (days)	10, 5	3, 2	0.002*
Death	1 (2.6%)	0	0.33
Abnormal brain MRI at 7 days	6/37 (16.2%)		

* indicates P < 0.05

For continuous variables, normally distributed data are presented as mean ± standard deviation and skewed data as median, interquartile range.

Table 3

Correlation among the 3 reviewers in assessing fetal heart rate parameters in the last hour of electronic fetal monitoring prior to delivery

	Kappa Correlation	Pearson Correlation
Reactivity	0.48	
Category	0.40	
Short term variability	0.37	
Fetal heart rate baseline		0.94
Tachycardia		0.90
Debt 60		0.83
Total decelerations		0.77
Debt 30		0.73
Accelerations		0.71
Late decelerations		0.69
Early decelerations		0.55
Variable decelerations		0.51
Severe variable decelerations		0.28

Table 4

Fetal heart rate characteristics in the last hour monitoring prior to delivery

	Cases n=39	Controls n=78	P value
Category			0.18
I	4 (10.3%)	7 (9.0%)	
II	30 (76.9%)	70 (89.7%)	
III	5 (12.8%)	1 (1.3%)	
Baseline (bpm)	148±18	141±14	0.054
Time baseline > 160 bpm (min)	0, 0	0, 0	0.11
Time baseline < 110 bpm (min)	0, 2	0, 0	0.22
Decreased variability	13 (33.3%)	15 (19.2%)	0.17
Reactive	16 (41.0%)	48 (61.5%)	0.047*
Accelerations	1, 3	2, 4	0.34
Total Decelerations	8, 11	6, 7	0.18
Late decelerations	1, 6	1, 2	0.01*
Variable decelerations	3, 5	3, 4	0.85
Severe variables	0, 1	0, 0	0.50
Early decelerations	0, 0	0, 1	0.06
Prolonged decelerations	1, 1	0, 1	0.11
Nadir (bpm)	62, 29	70, 35	0.60
Length (min)	4, 3	3, 3	0.17
Contractions/Hour	16.5±9.8	16.9±7.7	0.80
Lates/Contraction	0.1, 0.3	0, 0	0.21
Variables/Contraction	0.2, 0.4	0, 0.2	0.48
Debt 30 (sec*bpm)	9,458; 14,019	3,942; 6,630	0.008*
Debt 60 (sec*bpm)	13,347; 20,612	6,082; 8,871	0.003*

* indicates $P < 0.05$

For continuous variables, normally distributed data are presented as mean ± standard deviation and skewed data as median, interquartile range.

Table 5

Multivariable analysis of fetal heart rate tracing characteristics in the last hour monitoring prior to delivery adjusting for the presence of clinical chorioamnionitis.

Variable value	Odds Ratio	95%CI	P
Reactive	0.50	0.22 – 1.12	0.09
Late decelerations	1.10	1.00 – 1.21	0.06
Early decelerations	0.58	0.35 – 0.94	0.03*
Debt 30	1.00	1.00 – 1.00	0.01*
Debt 60	1.00	1.00 – 1.00	0.005*

* indicates $P < 0.05$

Table 6

Area under receiver operator characteristic (ROC) curve, sensitivity and specificity of electronic fetal heart rate monitoring during last the hour prior to delivery

	Area under ROC curve	Sensitivity	Specificity
Early decelerations	0.72 (0.64–0.80)	23.1% (11.7–39.7%)	94.9% (86.7–98.3%)
Debt 30	0.66 (0.54–0.78)	33.3% (19.6–50.3%)	87.2% (77.2–93.3%)
Debt 60	0.68 (0.56–0.79)	35.9% (21.7–52.8%)	89.7% (80.3–95.2%)