# Second stage fetal heart rate patterns and neonatal acid-base status

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**Background:** Fetal surveillance in labour is performed mostly to identify fetuses at risk of hypoxia in order to reduce neonatal morbidity and mortality by initiating timely intervention. While normal and abnormal fetal heart rate (FHR) patterns have been well recognised and characterized for the first stage of labour, FHR patterns during the second stage of labour commonly showed some forms of abnormalities leading to problems in interpretation, particularly in predicting fetal hypoxia and acidosis. This study aims to identify patterns of FHR tracing during the second stage of labour associated with neonatal acidosis.

**Methods:** A prospective cross sectional study was conducted in the Labour Ward of a state referral hospital. The study population were patients with low-risk singleton pregnancies between 37 to 42 weeks gestation who had normal cardiotocograph (CTG) tracing in the first stage of labour. CTG was recorded during the second stage of labour and neonatal umbilical cord blood was obtained for acid-base analysis immediately after birth prior to the delivery of placenta. FHR patterns were grouped according to modified Melchior and Barnard's classification and matched with neonatal acid-base status. Patients with normal FHR pattern in the second stage acted as control.

**Results:** A total of 111 matched pairs were analysed. Ninety nine (89.2%) second stage FHR tracings showed abnormal features when compared to control. There were significantly more neonatal acidosis and hypercapnia in type 1b, type 2a, type 2b and type 3 CTG patterns compared to control, in increasing order of severity. In addition, types 2b and 3 showed significant difference in the base excess.

**Conclusion:** Certain second stage fetal heart rate patterns were found to be associated with neonatal acidosis.

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*Key words: fetal heart rate patterns; second stage of labour; neonatal acidosis*

## Introduction

Fetal surveillance in labour is performed mostly to identify fetuses at risk of hypoxia. The aim is to reduce neonatal morbidity and mortality by initiating timely intervention. Since its introduction in 1960s, electronic fetal monitoring (EFM) has been widely used for fetal surveillance during labour. For the first stage of labour, the normal and abnormal FHR patterns have been well recognised and characterized.[1-3] On the other hand, FHR patterns during the second stage of labour commonly showed some forms of abnormalities leading to problems in interpretation, particularly in predicting fetal hypoxia and acidosis.

There has been many previous works in an attempt to determine the characteristics of an abnormal second stage CTG and its relation to neonatal status. Fetal bradycardia, either persistent or progressive has been shown to be strongly suggestive of fetal hypoxemia and Apgar score of less than 7 at 5 minutes, lower mean umbilical artery pH and neonatal acidosis.[4,5-8] Similarly, fetal tachycardia has been shown to be associated with low Apgar score at five minutes and with neonatal acidemia in up to 20 percent.[4,6,7]

Absent variability has been shown to be associated with an increased risk of neonatal acidosis, even in the presence of an otherwise normal CTG.[6,9] The presence of bradycardia preceded by decreased baseline variability strongly predicts the development of pathologic neonatal acidemia and indicating the need for urgent delivery.[6]

Accelerations were noted to be not commonly present in the second stage. Decelerations were more common, and were seen in more than 70 percent of second stage heart rate traces. Early decelerations did not appear to increase the risk of low 5 minute Apgar score and

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should be viewed as benign, regardless of baseline heart rate.[4] Variable decelerations were much common and were seen in approximately half of second stage CTG. If the baseline heart rate was normal, mild variable decelerations appeared to have little influence on the incidence of low Apgar scores.[4] However, variable decelerations with a drop in fetal rate of >70 beats per minute have been shown to be associated with an increased risk of metabolic acidosis.[9] Late decelerations, although relatively uncommon in the second stage of labour, markedly increased the risk of a low of 5 minute Apgar scores, regardless of baseline heart rate.[4]

To evaluate the neonatal outcome, APGAR score alone is a poor indicator of neonatal asphyxia and/or acidosis. The fetal acid-base status was the most reliable index of fetal oxygenation.[10] For this purpose, the umbilical artery is an easily accessible route to investigate fetal oxygenation after delivery. The acid-base status of the cord blood represents an objective parameter of the neonatal condition. The umbilical arterial sample was preferred to the venous sample as this will provide the acid base status of fetal blood returning to the placenta. However, the degree of acidaemia which led to a significant increase of neonatal morbidity is still controversial. Although fetal acidemia had been classically defined as an umbilical artery pH of <7.20, significant or pathologic fetal acidemia had more recently been defined as an umbilical artery pH of <7.00.[11,12] Even though neonatal seizures, other morbidities and deaths are significantly more common in neonates with a pH <7.0, the majority have no significant short- or long-term morbidity.[11, 13-15]

With the above background, we aim to identify the specific FHR patterns during second stage of labour in our patients and evaluate the association between the specific FHR patterns and the neonatal acid-base status.

## Methodology

This prospective cross sectional study was conducted from 1st March 2009 until 1st September 2009 at the labour ward in Tuanku Jaafar Hospital, Seremban. One hundred and fifteen women with normal pregnancy, at term, singleton and vertex presentation were recruited and followed up until the stage of labour.

Data collected include background demographic and obstetric characteristics, labour and delivery characteristic, fetal heart rate pattern, Apgar scores, acid-base status and early neonatal morbidity (need for resuscitation and NICU admission).

An external cardiotocograph was used to record the fetal heart rate and tocodynamometry was used to monitor the contractions. A paper speed of 1cm/min was used. The fetal heart rate tracings during the last hour of first stage and during second stage of labour were independently interpreted and classified by investigators. Interpretation of CTGs was based on modified Melchior and Barnard's classification.[5,16,17] However, for the type 1 pattern, we followed the subdivision of type 1 in two groups introduced by Cardoso et al (1995).

| Type of second stage CTG | Criteria |
|---|---|
| Type 0 | Stable fetal heart rate during the entire second stage |
| Type 1a | Mild variable decelerations with each contraction, normal fetal heart rate between contractions |
| Type 1b | Moderate to severe variable decelerations or late decelerations with each contraction, fetal heart rate returning to the normal level between contractions |
| Type 2a | Baseline fetal heart rate between 90 and 120beats/min with decelerations with contractions |
| Type 2b | Baseline fetal heart rate below 90beats/min with decreased baseline variability |
| Type 3 | Baseline fetal heart rate below 90beats/min, decreased variability, with giant accelerations with contractions. |
| Type 4 | Baseline fetal heart rate below 90beats/min during final moments of second stage only |

Because of complete normality of FHR patterns, type 0 was considered as the control group.

At delivery, blood was taken from cord umbilical artery using pre-heparinised syringes. Samples were drawn according to that described by Riley.[18] Immediately after delivery of the baby and before delivery of placenta, double clamps were applied as far as possible from the baby, enclosing a 5 cm segment of the umbilical cord before division. A cord blood in a double clamped cord is stable for up to an hour at room temperature.[19] Measurements of pH and base deficit were performed using Bayer Rapidlab Blood Analyser M248, Bayer Healthcare, USA located in the labour ward. A cord blood sample in a syringe flushed with heparin is stable for 30 to 60 minutes at room temperature.[20-22] Samples were kept in icepacks and processed within 30 minutes of collection. Acidemia was diagnosed when pH levels were more than one standard deviation below the mean level obtained in the control group.

**Statistics**

All data collected were analysed using SPSS for Windows version 15. Descriptive statistics (mean, standards deviation, percentage) and Independent *t* tests to compare means were used in this study. $P < 0.05$ was considered statistically significant.

**Results**

A total of one hundred and eleven matched pairs were included in the final analysis after exclusion of four pairs due to incomplete data. The fetal heart rate patterns on the cardiotocogram were evaluated and categorised according to the modified Melchior and Barnard's classification into 5 types with two subtypes in 1 and 2. Type 0, which showed no abnormal features based on the first stage fetal heart rate pattern and no abnormality in the neonatal acid base balance was taken as control.

For the background data of maternal age and gestational age of pregnancy, we found no significant differences among the different types of FHR pattern. There were also no significant differences in the neonatal birth weight between the study groups and the control group.

Our data showed that normal fetal heart rate pattern was only seen in 12 out of 111 (10.8%) second stage CTG tracings. The majority (89.2%) of second stage FHR patterns showed some form of abnormality with the most frequently seen being Type 1a (40.5%) and Type 1b FHR patterns (24.3%).

On analysing the acid-base status of the different fetal heart rate pattern types, with regards to the means of umbilical artery pH, PCO2, PO2, HCO3 and BE for each type of FHR patterns, we found significantly more neonatal acidosis and hypercapnia in type 1b, type 2a, type 2b and type 3 FHR pattern compared to control, in increasing order of severity. However, the differences in the umbilical artery base excess were only seen in type 2b and type 3 patterns, indicating significant acidosis. Types 1a and 4 showed no difference in the acid-base parameters when compared to the control group.

With regards to the influence of the duration of the second stage of labour to neonatal acid-base status, we found no significant association. Although patients in Type 2a have significantly longer labour and the newborns were more acidotic compared to control, this did not achieve statistical significance.

With regards to the Apgar scores of the neonates, from 111 studied, only one had a low Apgar score of less than 7 at 5 minutes. This was a newborn with a type 3 FHR pattern, which also showed the most severe form of fetal acidosis among the groups.

On analysing the delivery pattern, we found no significant differences with regards to the incidence of operative deliveries in different FHR patterns when compared to control.

**Discussion**

Melchior and Barnard originally described five fetal heart rate patterns during the active second stage of labour which are type 0, type 1, type 2, type 3 and type 4

with different prognostics concerning newborn acid base balance. In this study we used the modified Melchior and Barnard's classification as introduced by Cardoso[5] which are type 0, type 1a, type 1b, type 2a, type 2b, type 3 and type 4. Types 1 and 2 were further divided to indicate the increasing severity of the abnormality. In the sample selection, we only included the low risk pregnancy at 37 to 42 weeks with normal fetal heart rate tracings during first stage of labour to control for the effects of maternal illness and first stage problems on the outcome of the study.

Type 0 FHR pattern in this study was used as control because of complete normality of fetal heart pattern and all the acid base parameters in this FHR pattern were similar to normal cord blood parameters. We recorded a 10.8% normal second stage FHR pattern in our study population. Other authors have reported percentages ranging from 9 to 35%.[4,5,9]

The most common FHR patterns seen were Types 1a and 1b, which showed varying degrees of variable and late decelerations. This can be explained by the effects of uterine contractions and maternal bearing down effort which increase the intrauterine pressure leading to reduced feto-placenta perfusion and produces intermittent hypoxia to the fetus. The consequence would be decreasing umbilical cord pH and neonatal acidosis.[23] Despite the abnormal FHR pattern, we have shown that the final outcome was good and neonatal acidosis was rare and mild with no changes in the base excess.

The more sinister finding would be when the FHR patterns were showing fetal bradycardia (Types 2a, 2b and 3). In these instances, there would be neonatal acidosis, particularly when the baseline variability is also reduced. Similar findings have been reported by other authors.[5-7,9] However, when the fetal bradycardia occurred only during the final moments of the second stage, no fetal morbidity is observed. This suggests a need for a prompt action when fetal bradycardia is detected in the second stage of labour when birth is not imminent.

From this study we were able to identify the specific FHR patterns associated with neonatal acidosis. Based on the cord pH <7.2 and base excess >12, Types 2b and 3 were identified to be significantly associated with neonatal acidosis. Although Types 1b, 2a, 2b and 3 all had neonatal acidosis with cord blood pH <7.2, the base excess were normal in Types 1b and 2a. Given that most fetuses were shown to tolerate acidemia during labour with a pH cut-off of 7.0 without incurring neurological impairment, the clinical significance of isolated acidemia without concomitant rise in base excess is questionable. We were also able to show that the fetuses in Types 1b and 2a had good Apgar scores (>7 at 5 minutes) and if neonatal intensive care unit (NICU) admissions were required it was mainly for observation due to post-instrumentation and grunting, hence further supporting the fact that Types 1b and 2a FHR pattern are not associated with significant neonatal morbidity. The fetus in Type 2b, even with a pH of 7.14, admission to the NICU was not needed, putting further question on the clinical significance when the cord pH is >7.0. In contrast, the one fetus in Type 3 with a cord pH of 6.9 had significant neonatal morbidity requiring NICU admission and diagnosed with hypoxic ischemic encephalopathy (HIE) grade 2.

With regards to operative delivery, previous studies had shown that patients with abnormal fetal heart rate patterns had significantly higher rates of interventions9. Our study showed that patients with abnormal FHR patterns indeed had higher rates of operative deliveries. However, the difference did not reach statistical significance. In fact, fetuses in Types 2b and 3 were all delivered normally. This highlights the difficulty in evaluating the types of fetal heart rate pattern associated with acidemia, thereby influencing the decision for assisted delivery. By identifying specific fetal heart rate patterns for acidotic fetuses, a more informed decision can be made, thus avoiding both unnecessary interventions as well as delayed interventions.

Apgar score is being used widely to evaluate the status of the newborns and the need for resuscitation, despite

the controversy regarding its reliability as a measure of morbidity in a newborn. In our study, out of 111 newborns, only one had an Apgar score <7 at 5 minutes. This is despite the fact that 27 newborns had a cord pH<7.2 including 2 newborns who were confirmed to have neonatal acidosis (pH<7.2, base excess <12). Our finding adds to the evidence that Apgar score is a poor indicator of neonatal acid base status.

Overall, the fetal outcome in our study population was good with only 2 (1.8%) newborns confirmed to have neonatal acidosis. This could be attributed to our patient selection criteria which only included the low risk pregnancies with an apparent normal fetal status in the first stage of labour. For high risk pregnancies, we expect the incidence of neonatal acidosis to be higher.

The main limitation of our study is the small sample size and the small number of certain types of abnormal FHR patterns. Nevertheless, we have been able to identify specific fetal heart rate patterns associated with significant neonatal acidosis which would aid decision making during the second stage of labour.

## Conclusion

In conclusion, certain fetal heart rate patterns during second stage of labour were identified to be associated with neonatal acidosis. Second stage electronic fetal heart monitoring is thus a useful tool to identify fetuses that are potentially acidotic and the information can be used to aid decision making during the second stage of labour.

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Table I: Neonatal outcomes and the second stage fetal heart rate patterns

| | Type of Second stage Fetal Heart Rate Patterns (n=111) | | | | | | |
|---|---|---|---|---|---|---|---|
| | **Type 0 n=12** | **Type 1a n=45** | **Type 1b n=27** | **Type 2a n=9** | **Type 2b n=1** | **Type 3 n=1** | **Type 4 n=16** |
| **(%)** | 10.8 | 40.5 | 24.3 | 8.1 | 0.9 | 0.9 | 14.4 |
| **Age[a] (years)** | 26.83 (4.89) | 27.89 (4.74) | 28.22 (4.73) | 25.60 (2.50) | 26.0 (0) | 32.0 (0) | 28.75 (3.77) |
| | | NS | NS | NS | NS | NS | NS |
| **Gestational age[a] (weeks)** | 39.17 (1.53) | 39.00 (1.19) | 39.41 (1.31) | 38.56 (1.01) | 41.00 (0) | 38.00 (0) | 38.81 (0.91) |
| | | NS | NS | NS | NS | NS | NS |
| **Birth weight[a] (gram)** | 2970 (338) | 3128 (345) | 3081 (372) | 2883 (398) | 3300 (0) | 3020 (0) | 2987 (377) |
| | | NS | NS | NS | NS | NS | NS |
| **Second stage[a] (minute)** | 16.25 (11.6) | 17.67 (17.5) | 25.22 (16.6) | 35.89 (27.28) | 25.00 (0) | 27.00 (0) | 6.81 (6.8) |
| | | NS | NS | **p<0.05** | NS | NS | **p<0.05** |
| **Neonatal acid-base status** | | | | | | | |
| PH[a] | 7.29 SD± 0.05 | 7.28 SD ±0.06 | 7.17 SD ±0.10 | 7.18 SD ±0.09 | 7.14 SD ±0 | 6.9 SD ±0 | 7.30 SD± 0.08 |
| | | **NS** | **p<0.05** | **p<0.05** | **p<0.05** | **p<0.05** | **NS** |
| PCO2[a] | 44.92 SD± 5.07 | 47.89 SD± 8.56 | 59.38 SD±14.14 | 61.16 SD±12.97 | 56.70 SD ±0 | 83.5 SD ±0 | 43.8 SD± 8.48 |
| | | **NS** | **p<0.05** | **p<0.05** | **p<0.05** | **p<0.05** | **NS** |
| HCO3[a] | 20.20 SD ±2.61 | 21.63 SD ±2.73 | 21.65 SD± 2.98 | 21.45 SD ±2.11 | 15.70 SD ±0 | 17.9 SD ±0 | 20.84 SD ±2.76 |
| | | **NS** | **NS** | **NS** | **NS** | **NS** | **NS** |
| BE[a] | -5.67 SD±2.48 | -4.85 SD±2.69 | -8.01 SD±4.42 | -7.35 SD±3.33 | -13.3 SD ±0 | -15.9 SD ±0 | -5.47 SD± 2.91 |
| | | **NS** | **NS** | **NS** | **p<0.05** | **p<0.05** | **NS** |
| **Apgar Score** | | | | | | | |
| Less than 7 at 5" | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| **Mode of Delivery (%)** | | | | | | | |
| **SVD** | 11 (91.7%) | 40 (88.8%) | 19 (70.4%) | 6 (66.7%) | 1 (100%) | 1 (100%) | 15 (93.8%) |
| **Operative delivery** | 1 (8.3%) | 5 (11.2%) | 8 (29.6%) | 3 (33.3%) | 0 | 0 | 1 (6.2%) |
| | | **NS** | **NS** | **NS** | **NS** | **NS** | **NS** |

*Mean[a], SD = standard deviation, NS =Not significant (P>0.05)*