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Interpretation of Fetal Heart Rate Monitoring in the Clinical Context

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Abstract: Use of intrapartum fetal heart rate (FHR) monitoring has had limited success in preventing hypoxic injury to neonates. One of the most common limitations of FHR interpretation is the failure to consider chronic and acute clinical factors that may increase the risk of evolving acidemia. This manuscript reviews common clinical factors that may affect the FHR and should be considered when determining the need for early intervention based on changes in the FHR.
Key words: fetal heart rate monitoring, clinical context, fetal academia

Background

Since the introduction of intrapartum fetal heart rate monitoring (FHR) in the 1950s, it has been established as the most common way to evaluate fetal well-being during labor in the United States.[1] With the notion that FHR patterns could predict fetal acid-base status, and, indirectly fetal oxygenation, the intent was to accurately assess the fetal condition and to determine whether changes in management or expedited delivery were indicated to decrease intrapartum hypoxic-ischemic injury. Unfortunately, this goal has not been achieved. Multiple studies suggest that intrapartum FHR monitoring has, instead, been associated with increased rates of surgical delivery and increased cost to the medical system, without a recognizable effect on fetal outcome.[2–4]

Attempts to better utilize the available technology to improve outcomes has led to the development of a classification system for FHR interpretation.[5] Published in 2008, the Eunice Kennedy Shriver National Institutes of Child Health and Human Development (NICHD) instituted a system that classified FHR patterns in intrapartum patients into 3 categories. The assignment of a category, 1, 2, or 3, was solely determined by the characteristics of the FHR tracing. The goal of these definitions was to "allow the predictive value of monitoring to be assessed more meaningfully and to allow evidence-based clinical management of intrapartum fetal compromise." It also allowed standardization of a system that, to that point, had not been organized in an expanded comprehensive way.[5]

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Although this standardization was an improvement for more accurate evaluation of the fetus in labor, it is limited to use of the FHR and the differences and changes seen during labor and does not account for specific antepartum and intrapartum risk factors present that, when used in conjunction with the specific patterns of the FHR, can lead to improved interpretation. Perhaps because placental function and maternal-fetal oxygen transport are assumed to be a final step in the association between most antepartum factors and fetal-neonatal compromise, using clinical findings to consider a pregnancy's *a priori* risk for placental insufficiency and neonatal hypoxia and acidosis may allow for FHR patterns to be interpreted with consideration of the overall picture.

Adequate Interpretation

Adequate interpretation of the FHR pattern during labor requires an interplay of several critical steps. First, one must consider the clinical context for which the FHR testing has been instituted. Clinical factors such as gestational age, the presence of labor, or infection may result in physiological changes that affect the characteristics of the FHR in the absence of acidemia and thus may alter interpretation. Next, there must be an acceptable technical recording and documentation of the FHR to allow for any kind of interpretation. Last, the providers reviewing the documented FHR need to correctly evaluate the different components involved; specifically, the FHR baseline, presence and degree of variability, the presence of accelerations, and the presence, type, frequency, severity, and recurrent nature of decelerations. The providers also must be able to evaluate these components as they change over time.[6]

Clinical Context

An understanding of the clinical context is important in the interpretation of the FHR tracing. Clinical factors, such as gestational age, may alter the individual characteristics of the FHR that are used to identify evolving acidemia. An understanding of these physiological changes is important to avoid misinterpretation. Pre-existing chronic medical conditions may alter placental function resulting in a chronic state of fetal hypoxia and a more rapid fetal decline during times of stress. Presence of these conditions increase the pretest probability of finding metabolic acidemia and changes suggesting fetal compromise in these pregnancies must be considered within this context. Acute clinical conditions such as infection or abruption also convey an increased rate of fetal deterioration and must be considered as part of the interpretation of fetal status suing the FHR.

EFFECT OF CLINICAL FACTORS ON SPECIFIC FHR CHARACTERISTICS

Baseline

Without question, the FHR baseline is of critical importance in the clinical interpretation of the FHR tracing. Normal tracings are characterized by a stable baseline FHR of 110 to 160 beats per minute (BPM), for a 10-minute segment and duration $\geq$ 2 minutes. It excludes periodic and episodic changes, marked variability, and segments differing by $\geq$ 25 BPM.[5] Any evaluation of the FHR tracing demands an initial assessment of the FHR baseline. First, it allows the provider to determine if this baseline is in a normal range and second, it is the measure by which the other components of interpretation are evaluated. For example, if the baseline is not accurately assigned, then a deceleration may be inaccurately interpreted as an acceleration and vise-versa.

A change in FHR baseline is present when the change persists for 10 minutes or longer. A baseline of <110 BPM is defined as bradycardia.[5] Mild bradycardia (100 to 110 BPM) can be normal and has been associated with postterm infants and occipitoposterior position.[6] Although uncommon, abnormal

rates of <100 BPM may be seen in fetuses with congenital heart disease or myocardial conduction defects such as fetal heart block.[7] A baseline >160 BPM is defined as fetal tachycardia and this finding has been associated with certain maternal and fetal conditions, such as chorioamnionitis, maternal fever and dehydration, as well as tachyarrhythmias. Any drastic change in the FHR baseline outside of the "normal" parameters of 110 to 160 BPM necessitates evaluation by a clinician, as this may not be indicative of an abnormal fetal acid-base status, but is, again, not within the normal range.

The gestational age of the pregnancy is important, as FHR patterns are different in the preterm compared with the term gestation. Physiological control of FHR and the features observed in the preterm fetus can make interpretation difficult.[7] The preterm fetus often has a FHR with a higher baseline and there is a normal, incremental decline in the FHR baseline as the fetal parasympathetic nervous system matures with advancing gestational age.[8] Thus, relative FHR tachycardia may be normal in a less mature fetus.

Variability

Baseline variability, defined as fluctuations in the FHR of >2 cycles per minute, with grades of fluctuation based on amplitude range (peak to trough), is crucial, as it reflects a normal fetal nervous system, chemoreceptors, baroreceptors and cardiac responsiveness. Persistently minimal or absent FHR variability appears to be the most significant intrapartum sign of fetal compromise.[9] Fetal metabolic acidosis is one concerning cause for decreased variability but other etiologies include central nervous system depressants such as maternal narcotic use, fetal sleep cycles, congenital anomalies, prematurity, fetal tachycardia, administration of betamethasone and preexisting fetal neurologic abnormality.[10–13] Interpretation of decreased baseline variability should consider the presence of benign causes for this change.

Variable Decelerations

Establishing the type of deceleration is critical, as these "drops" in the baseline FHR are caused by different conditions. The definitions of various deceleration types including early, late and variable have previously been described.[5] Variable decelerations vary in terms of shape, depth, and timing in relationship to uterine contractions. Overall, variable decelerations are usually benign changes caused by cord compression, with subsequent changes in peripheral vascular resistance or oxygenation[14] and they are very common in the second stage of labor. However, the presence of vaginal bleeding or intractable pain may suggest the possibility of uterine rupture or abruption which may not be amenable to normal interventions and may result in rapid fetal deterioration. Variable decelerations in patients with a previous cesarean section or risk factors for abruption must be interpreted with caution.

Late Deceleration

Since recurrent late FHR decelerations are indications of utero-placental insufficiency and possible fetal hypoxia, attempts by the clinician to improve circulation to the placental unit and the fetus is imperative. This is seen in the case of maternal hypotension, which may be the result of regional anesthesia, maternal factors, such as dehydration or shock or with medication use. Attempts to improve the maternal blood pressure with medication, such as ephedrine or volume expansion can improve maternal circulation and perfusion of the uterus and improve the FHR tracing.[15] Administration of a tocolytic agent, such as terbutaline is often employed to transiently stop contractions, with the understanding that in studies evaluating this administration, improvement in FHR tracings in treated groups compared with control groups did not translate into an improvement in neonatal outcomes

(administration of a tocolytic agent improved FHR tracings compared with untreated control groups, but there were no improvements in neonatal outcomes.[16]

REVIEW OF CHRONIC CLINICAL FACTORS AFFECTING FHR

It has long been recognized that maternal antepartum factors play an important role in fetal-placental development and, therefore, in maternal and newborn outcomes. Conditions commonly associated with nonreassuring fetal status and abnormal FHR tracings include maternal cardiovascular disease, anemia, diabetes, hypertension, infection, placental abruption, abnormal presentation of the fetus, intrauterine growth restriction and umbilical cord compression, among other obstetric, maternal or fetal conditions. These elements need to be considered with interpretation of the FHR tracing in the clinical setting.

Maternal Age

Advanced maternal age (AMA) is increasingly prevalent in developed countries[17] and maternal age has been established as a risk factor for specific adverse pregnancy outcomes and should be considered as a chronic condition that may alter the interpretation of the FHR, both antepartum and while in labor.

A retrospective evaluation of the patients from a large, multicenter trial demonstrated that increasing maternal age was significantly associated with multiple pregnancy complications including miscarriage, chromosomal abnormalities, congenital anomalies, gestational diabetes, placenta previa, and cesarean delivery.[18] Increased risk for abruption, preterm delivery, low birth weight, and perinatal mortality was noted in women aged 40 years and older. Canterino et al[19] demonstrated increasing rates of fetal death at >24 and at >32 weeks in patients with increasing maternal age. In their cohort, the relative risk for fetal death at >24 and at >32 weeks among women 35 to 39 years were 1.21 and 1.31, respectively, while the relative risks were 1.62 and 1.67 among women aged 40 to 44 years. Performing a population-based cohort study, Kenny et al[20] compared pregnancy outcomes in women aged 30 to 34, 35 to 39 and 40 years and above with women aged 20 to 29 years using log-linear binomial regression and found that women greater than 40 years of age were at increased risk for stillbirth, preterm and very preterm birth, and macrosomia. The risk for pre-eclampsia also increases with AMA. Women aged 40 or older have a 2-fold higher rate of pre-eclampsia compared with the general population.[21] Collectively, these increased risks from AMA can likely be linked to placental dysfunction. In fact, Lean et al[22] suggested that placental dysfunction underlies the increased risk for fetal growth restriction and stillbirth in AMA patients.

It is not surprising that AMA has been associated with and increased risk for abnormal FHR tracings and fetal acidemia. In 2013, maternal age above 35 was 1 of 10 antepartum factors found to be significantly associated with poor neonatal outcome when found in association with category II FHR tracings among $>51{,}000$ patients evaluated.[23] In addition, a prospective cohort study of 8580 women with specific electronic fetal monitoring patterns associated with and predictive of acidemia found that these patterns were more common in women designated as AMA.[24]

In short, there is evidence to suggest that AMA is a known risk factor for adverse obstetric outcome, likely related in part to placental dysfunction, and demonstrated by abnormal FHR patterns in labor. AMA should be considered a risk factor for adverse neonatal outcome in the clinical context of FHR interpretation.

Obesity

Obesity is an important consideration in the clinical evaluation of the FHR as it has been identified as a risk factor for adverse pregnancy outcome.[25] Obesity often makes

it difficult to obtain a readable, and thus interpretable, FHR tracing. In fact, the inferiority of external Doppler ultrasound and toco to internal modalities is well established and is more notable in the obese patient.[26] FHR monitoring is of particular importance in the obese patient because high body mass index is associated with a number of comorbidities, such as diabetes mellitus and hypertensive disorders. These comorbidities are associated with abnormal FHR tracings and adverse pregnancy outcomes, such as stillbirth.[27]

Insulin Dependent Diabetes

Insulin dependent diabetes can have a significant impact on the FHR tracing, both from an acute standpoint and as a more chronic influence. This is to be expected given the convincing evidence of the diabetes and the effects on the structure and function of both the fetal heart and the maternal/fetal placenta. Acutely, maternal hyperglycemia has been associated with an elevated FHR. Costa and colleagues prospectively evaluated pregnant women with pregestational diabetes mellitus in the third trimester and found a significant positive correlation (Pearson test, $P = 0.0001$, $r = 0.57$) between basal FHR and mean glycemia. A significant negative correlation was observed between short-term variation and mean glycemia (Pearson test, $P = 0.003$, $r = -0.47$).[28] Tincello and colleagues also found significant differences between the FHR tracings of those patients with type I diabetes and control patients including reduced FHR variability and frequency of accelerations observed in third trimester fetuses of diabetic mothers. These differences were felt to represent a more immature form of FHR than that which would be expected based on gestational age.[29]

Chronically, maternal diabetes and hyperglycemia influences placental development during the embryonic phase resulting in changes to fetal metabolic status. It has been shown that with maternal diabetes, the fetus likely increases oxidative metabolism, placing the fetus at risk for hypoxemia in situations of increased oxygen demand such as labor.

Intrauterine Growth Restriction and Oligohydramnios

Fetal growth restriction and oligohydramnios are well established risk factors for abnormal FHR patterns and adverse obstetric outcome. Intrauterine fetal growth restriction (IUGR) refers to a fetus that has failed to achieve its genetically determined growth potential and affects up to 5% to 10% of pregnancies.[30] It is associated with an increase in perinatal mortality and morbidity, with a resultant high risk for intrauterine fetal demise, and intrapartum fetal morbidity. At delivery, growth restricted infants (< 3rd percentile) have nearly twice the incidence of low Apgar scores and umbilical pH <7.0. Since normal placental development is required for adequate fetal growth, many of the cases of IUGR are the result of placental dysfunction and this may predispose the fetus to abnormal FHR patterns in labor. Epplin et al[31] performed a 5-year retrospective cohort study of singleton term laboring patients comparing IUGR infants with non-IUGR infants and found that IUGR at term confers an increased risk of late decelerations and that the patterns in these patients may require different interpretations based on *a priori* risk and clinical factors.

Late decelerations would be expected in the patient with an IUGR fetus given the relationship of those findings to the etiology of uteroplacental insufficiency. However, the IUGR fetus is also at risk for variable decelerations. Pregnancies affected by IUGR often have attendant oligohydramnios[32] and are thus at risk for intermittent umbilical cord compression. Despite this, understanding of the acid/base responses rates of IUGR fetuses to variable FHR decelerations as might occur during human labor, is somewhat

limited. Amaya and colleagues evaluated the increase in base deficit among chronically hypoxic as compared with normoxic ovine fetuses in response to different simulated variable FHR decelerations. The researchers found that repetitive umbilical cord occlusion resulted in the development of acidosis ($pH < 7.0$) in both groups, but that in comparison to the normoxic fetuses, hypoxic fetuses progressed more rapidly to significant metabolic acidosis in response to moderate FHR variable decelerations. These hypoxic fetuses were also slower to recover with in utero resuscitation, likely due to impaired placental function and fetal physiological responses.[33] The presence of IUGR or oligohydramnios in the setting of abnormal FHR may indicate a need to expedite delivery more quickly that identifying the same findings in a normally grown fetus with normal fluid volumes.

Previous Cesarean

The assessment of the FHR tracing in the clinical context of a prior cesarean delivery is of particular importance because the FHR tracing is often the initial sign that there may be a concern for uterine rupture. In one study published in 2012, among cases of uterine rupture identified in 9 hospitals, > 80% of cases of uterine rupture were identified secondary to concerning FHR tracing findings, specifically the presence of severe repetitive decelerations or fetal bradycardia. Even more importantly, identification of these FHR abnormalities was crucial in accomplishing delivery in <18 minutes to avoid adverse neonatal outcomes associated with those ruptures.[34]

ACUTE CHANGES ASSOCIATED WITH POOR CLINICAL OUTCOME

Decreased Fetal Movement (DFM)

Maternal perception of fetal movement often begins in the second trimester and can vary somewhat, depending on the time of day and gestational age.[35] Although it is a common occurrence, the presence of DFM in a pregnancy with previously normal fetal movement may be a sign of evolving fetal hypoxia. Any pregnancy with a good clinical history for DFM should be evaluated. In a nonrandomized Norwegian study of > 3000 women presenting with DFM, 97.5% of the women were assessed using a FHR monitoring and 3.2% of the presentations were abnormal.[36] This demonstrates the importance of fetal monitoring as a valid screening tool in the setting of DFM, an abnormal FHR pattern may be associated with poor outcomes. This especially true when the initial FHR tracing of a patient with DFM is remarkably abnormal with findings suggesting evolving acidemia (ie, tachycardia, minimal or absent variability and repetitive decelerations). Expedited intervention should be considered if reassuring findings cannot be elicited after a short period of intrauterine resuscitation.

Maternal Infection

Chronic maternal infection can influence the FHR tracing interpretation. Kaneko and colleagues compared the incidence of abnormal FHR pattern and umbilical blood gases between 20 pregnancies affected by cytomegalovirus (CMV) infection and normal controls. They found nonreassuring FHR patterns (prolonged deceleration and recurrent late deceleration) in 8 of 20 fetuses in the CMV group and in 3 of 41 fetuses in the control group ($P < 0.05$, Fisher test). The most common abnormalities in the CMV group included prolonged decelerations, recurrent late decelerations and minimal variability and the authors concluded that CMV-infected fetuses were more likely to show abnormal intrapartum FHR patterns.[37] Parvovirus B19 is a widespread infection that may affects 1% to 5% of pregnant women, mainly with normal pregnancy outcome, but will occasionally result in fetal anemia and resultant FHR abnormalities, including decreased variability, decelerations and occasionally, a sinusodal pattern.[38]

INTRAPARTUM CLINICAL MARKERS ASSOCIATED WITH POOR OUTCOME

Meconium

Meconium, a dark green fecal material produced in the intestines of a fetus before birth, is a relatively common finding in the amniotic fluid during labor, present in 10% to 15% of pregnancies, but is more often seen in pregnancies that are post-dates, in specific maternal conditions such as diabetes and hypertension and in prolonged labors. The relationship between the presence of meconium and abnormal FHR patterns was established >2 decades ago. A study in 1990 concluded that meconium in amniotic fluid was associated with placental insufficiency.[39] In 2001, Hadar and colleagues evaluated perinatal outcomes of infants who had pathologic FHR tracings during the first stage of labor, in comparison with pregnancies with normal tracings. In this study, the presence of meconium-stained amniotic fluid was an independent factor associated with pathologic FHR monitoring during the first stage of labor in a multivariable analysis [odds ratio (OR), 1.91; 95% confidence interval (CI), 1.03%-3.3%].[40] In 2009, a retrospective cohort of 1638 patients with labors complicated by thick meconium-stained amniotic fluid evaluated the association between specific FHR patterns and adverse perinatal outcomes. In patients with thick meconium, the presence of FHR tracing abnormalities was associated with an increased risk of perinatal mortality and/or neonatal morbidity (moderately abnormal: adjusted OR, 1.67; 95% CI, 1.18-2.37; markedly abnormal: adjusted OR, 2.97; 95% CI, 1.88-4.67). The specific FHR abnormalities that were associated with this risk included prolonged decelerations (OR, 1.22; 95% CI, 1.02-1.48), severe variable decelerations (OR, 1.08; 95% CI, 1.00-1.16), bradycardia (OR, 2.49; 95% CI, 1.02-6.11), and tachycardia (OR, 2.43; 95% CI, 1.49-3.94).[41] These findings confirm that abnormalities in the FHR in the presence of meconium are associated with an increased risk for adverse outcome. Thus, the clinician evaluating the abnormal FHR tracing, should assess the situation for the presence of meconium and consider this finding as an important clinical indicator for decision making.

Intrapartum Bleeding

Intrapartum vaginal bleeding should prompt immediate evaluation from the clinical team given the possibility that the hemorrhage may be originating from the fetal or placental unit. Placental abruption, uterine rupture and placenta previa often present with vaginal bleeding and have demonstrable changes in the FHR secondary to interrupted flow to the placenta and sometimes, maternal instability.[23] These FHR changes include tachycardia, bradycardia, repetitive variable decelerations, late decelerations and prolonged decelerations. Vasa previa represents direct hemorrhage from the fetal umbilical cord blood vessels and although rare, when this occurs, it almost always results in an abrupt fetal bradycardia.[42]

Intrapartum Infection

In 2008, Buhimschi and colleagues hypothesized that abnormal FHR monitoring may occur more often in pregnancies complicated by intra-amniotic inflammation which disrupts placental transfer to the fetus. The researchers evaluated 87 singleton pregnancies delivered within 48 hours of amniocentesis and found that the fetuses of women with severe intra-amniotic inflammation had a higher FHR baseline and increased frequency of non-reactive FHR tracing.[43] However, it is not clear if abnormal FHR tracings associated with infection and inflammation can be accurately translated into fetal acidosis in labor. In 1983, Duff et al[44] evaluated 65 patients with chorioamnionitis in labor and found that the most common FHR

abnormalities were diminished or absent variability (77%) and tachycardia (67%); however, despite the high prevalence of abnormal FHR tracings, only 1 infant had a 5-minute Apgar score <7. A more recent study evaluated 86 infants diagnosed with cerebral palsy postdelivery. These infants all had abnormal FHR tracings and were further subdivided into 1 of 2 groups depending on the presence of absence of clinical chorioamnionitis. The frequency of severe fetal acidemia in the group without chorioamnionitis was 26.3%, compared with 74.6% in the group with an infection. The authors concluded that the observation of clinical intrapartum infection should be included as a risk factor for acidemia in the standardized clinical interpretation of FHR patterns.[45]

PUTTING IT ALL TOGETHER

Several studies have attempted to use the contribution of clinical risk factors with the interpretation of FHR patterns, particularly those in category II. Parer and Ikeda.[46] identified 134 FHR patterns, classified by baseline rate, baseline variability, and type of deceleration, and used to assign a risk of newborn infant acidemia or low 5-minute Apgar score They also evaluated each pattern for the risk that it would evolve into a pattern with a higher risk of acidemia. Each FHR pattern was color-coded, from no threat of fetal acidemia (green, no intervention required) to severe threat of acidemia (red, rapid delivery recommended). The authors established 3 intermediate categories (blue, yellow, and orange) that would require escalation for intervention and resuscitation, and possibly, preparation for urgent delivery. Although this was very helpful in establishing the concerning patterns and standardizing a uniform response, it did not consider the antepartum or intrapartum clinical setting for the patient presenting in labor.

In 2012, Holmgren et al[23] published the development and evaluation of a labor risk model consisting of a combination of antepartum risk factors and intrapartum FHR characteristics. Using these factors, the goal was to reliably identify those infants at risk for adverse neonatal outcome in labor. Using a nested case-control study of term singleton deliveries at the 9 hospitals, an initial risk score was determined using data available at 1 hour after admission. Data from >50,000 patients was used and of 31 antepartum variables evaluated, 10 were associated with an adverse outcome including maternal age above 35 years, increasing body mass index, increasing gestational age, nulliparity, maternal diabetes, maternal hypertension, pre-eclampsia, placental abruption and induction of labor. Quite importantly, a major risk factor in this calculation was the presence of a category II FHR in the first hour of monitoring. Additional evaluation of intrapartum characteristics in these patients, including chorioamnionitis, minimal FHR variability, recurrent FHR variable decelerations, FHR tachycardia and prolonged FHR decelerations, further predicted adverse outcome. The women with a high initial risk score and high intrapartum risk score had an 11.3% risk of adverse neonatal outcome and a cesarean delivery rate of 40%.

The Advanced Life Support in Obstetrics (ALSO) curriculum has developed the mnemonic "Dr C. Bravado" to teach a systematic, structured approach to continuous EFM interpretation that incorporates the NICHD definitions. Using this system, a clinical risk status (low, medium, or high) of each fetus is assessed in conjunction with the interpretation of the continuous EFM tracing. A term, low-risk baby may have higher reserves than a fetus that is preterm, growth restricted, or exposed to uteroplacental insufficiency because of pre-eclampsia. An increase in risk status during labor, such as the diagnosis of chorioamnionitis, may necessitate a change in monitoring from structured intermittent auscultation to continuous EFM.[47]

Conclusions

Unfortunately, the original goal of FHR monitoring, reduction of intrapartum hypoxic-ischemic injury, has not yet been achieved and instead, there have been increased rates of surgical delivery and increased cost associated with FHR monitoring. One of the limitations in the use of FHR monitoring to detect evolving fetal acidemia may be the failure to interpret the FHR within the clinical context. Chronic and acute clinical factors may indicate an increased *a* priori risk for the development of acidemia and may thus improve the predictive value of this commonly used screening test.

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