Decelerations, tachycardia, and decreased variability: have we overlooked the significance of longitudinal fetal heart rate changes for detecting intrapartum fetal hypoxia?

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One of the most difficult challenges in obstetrics is to ensure appropriate timing of delivery of the fetus. During labor, unnecessary operative interventions may cause maternal harm whereas delayed interventions may cause fetal or neonatal death or permanent central nervous system (CNS) injury. Labor exposes the fetus to varying degrees of stress from interruptions in blood supply due to uterine contractions, umbilical cord compression, head compression, placental abruption, or rupture of fetal vessels.[1-6] Electronic fetal monitoring (EFM) was introduced in the 1960s, as a means of intrapartum fetal surveillance, in hopes of early detection and timely delivery of fetuses with inadequate reserve to tolerate the stress of labor. Since most fetal asphyxia cases occur in low-risk pregnancies,[7] it was natural for the use of EFM to become widespread—being utilized in 85% of pregnancies.[8] Unfortunately, years later there is still controversy about its fetal benefits despite the associated increased rates of cesarean and operative vaginal deliveries. One of the shortcomings of EFM has been the high interobserver and intraobserver variabilities in the interpretation and management of the various fetal heart rate (FHR) patterns.[9,10] This can foster the impression that FHR tracing interpretation is somewhat subjective, which is problematic when trying to make decisions about labor management based on signs of fetal deterioration. The use of computerized or color-coded FHR classification systems could be helpful, but they have not been studied enough and are not widely available.[11,12]

Due to the aforementioned challenges, the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, American Congress of Obstetricians and Gynecologists (ACOG), and Society for Maternal-Fetal Medicine held a workshop in April 2008 to provide an update on FHR definitions, interpretation, and research guidelines. The workshop recommended standard definitions for FHR patterns and proposed a new 3-tier classification system for interpretation and management of FHR patterns.[13] Based on the ability to predict fetal acid-base status at the time, the FHR patterns were classified as category I (normal), II (indeterminate), or III (abnormal). The workshop specifically recognized that FHR tracing categories "can and will change" since labor is a dynamic process. One of the recommended areas for research was observational studies focused on change of FHR patterns "over time." Unfortunately, very little research has been published focusing on the longitudinal (over time) FHR assessment during labor. Instead, studies have focused on the static cross-sectional evaluations of FHR tracings of limited duration just prior to delivery.[14-16]

The ACOG recommends that in low-risk patients, the FHR tracing should be evaluated by nurses or physicians, every 30 minutes in the first stage of labor and every 15 minutes during the second stage of labor.[17] However, no instructions or recommendations are provided regarding over time (longitudinal) FHR assessment including more than just the last 15 or 30 minutes. In a recent poll (unpublished data, A.M.V.) taken among labor and delivery nurses, it was found that most nurses will evaluate the last 15-30 minutes, as per ACOG guideline, and they rarely go back to evaluate the FHR tracing in its entirety. Not surprisingly, there often is a predictable change in the FHR pattern as the fetus deteriorates over time from category I to category II or III, which will be missed unless the entire FHR tracing from admission is evaluated instead of the last few minutes. Category II tracings, which are the most frequent seen in >80% of laboring women,[17-19] are quite variable in their significance and can include FHR patterns from the most benign to the most threatening. In such cases, a static evaluation of only the last 15-30 minutes may miss earlier transitional FHR changes of a deteriorating fetus. This editorial argues in favor of using the longitudinal assessment of FHR changes during labor to emphasize the point that in many cases, especially in the evolution of category II FHR patterns, evaluation of fetal status over time should include not only the last 15-30

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minutes of the FHR tracing, but the entire labor. Given the electronic storage of the FHR tracings during labor in most labor and delivery units, this task will take only few minutes.

Previous reviews have shown that undetectable or minimal FHR variability in the presence of late or variable decelerations is the most consistent predictor of newborn acidemia and that fetal acidemia with decreasing FHR variability and decelerations develops over time.[20] Although there is a positive association between the depth of decelerations or bradycardia and fetal acidemia,[20] the 2008 workshop did not consider the severity of FHR decelerations in the FHR classification; instead, it heavily relied on FHR baseline variability (moderate for category I, minimal for category II, and absent for category III). In our practical experience, we have found that one of the most frequent reasons for misinterpreting the severity of a FHR pattern is the human inability to distinguish between absent (amplitude range undetectable) vs minimal (amplitude range >undetectable and ≤5 beats per minute [bpm]) variability in a 10-minute window. As a matter of fact, it is not unusual to perceive epochs with both absent and minimal baseline FHR variability within the same 10-minute window. For this reason, we agree with Clark et al[21] who proposed to classify and manage category II FHR tracings based on: (a) moderate (6-25 bpm) vs minimal or absent (≤5 bpm) FHR variability; (b) significance (late, severe variable or prolonged) and frequency of decelerations (≥50% of the contractions); and (c) estimated time to delivery by evaluating the progress of labor. The algorithm of Clark et al[21] based on the aforementioned main criteria is definitely a step forward. However, by the time a fetus shows the most concerning features of (a) and (b) noted above, the optimum window of intervention for prevention of fetal injury may have passed. In cases of fetal hypoxia, it is very common to observe progressively more frequent episodes of tachycardia after decelerations that are initially transient and later become more consistent. This is because in response to repetitive hypoxic stress from uterine contractions, the fetus initially compensates by increasing its heart rate since its ability to increase stroke volume is not very efficient. However, the development of fetal tachycardia, as a longitudinal FHR change, in the course of fetal deterioration was not emphasized by either the 2008 workshop[10] or the Clark et al[21] report.

In our view, any pattern of decelerations that cause compensatory tachycardia should be included in the list of clinically significant decelerations. We believe that the significance of fetal tachycardia as an indicator of the fetal condition is missed by many clinicians when evaluating the progression of FHR changes. The association of fetal tachycardia with fetal compromise and injury is not a new concept, with the link suggested over a century ago.[22] Ginsburg and Gerstley,[22] in 1965 assessed outcomes in 102 fetuses with tachycardia (≥180 bpm) 31 of which also had episodes of FHR decelerations, called "bradycardias." They noted that fetuses with tachycardia plus decelerations were much more likely to result in depressed infants, especially if this occurred >2 hours prior to delivery.[22] In our view, frequent episodes of fetal tachycardia, or continuous fetal tachycardia, in response to FHR decelerations are commonly the first signs of fetal struggle when a longitudinal assessment is performed of infants compromised at delivery. More recent research has strongly suggested the combination of decelerations with fetal tachycardia is the strongest predictor of metabolic fetal acidemia.[23] Fetal tachycardia is reported to be a better predictor of fetal acidemia (cord artery pH <7.10) than just late decelerations with adjusted odds ratios of 3.68 vs 2.28, respectively.[24] There may be a tendency to underestimate the importance of tachycardia when there is a known cause such as maternal fever, but the presence of tachycardia with decelerations in a febrile mother should remain concerning for substantial fetal compromise.

The progression of FHR patterns during labor could be sudden from category I to category III in the presence of acute insults such as placental abruption, uterine rupture, bleeding vasa previa, or cord prolapse. However, more frequently, the progression of category I patterns to category II or III is gradual, developing over the course of many hours. Clinicians should consider these progressive longitudinal changes as a deteriorating fetal vital sign. There are few obstetric tragedies greater than a pregnancy that enters labor with an apparently healthy fetus and delivers a compromised infant, especially when many of these cases could be preventable. Obstetric services should audit all cases with FHR accelerations and moderate variability (category I FHR pattern) on admission, which end up with a birth of a depressed neonate in the absence of a sudden unexpected sentinel event. Such audits often provide important information that can be used to improve the quality and safety of obstetrical care. Review of the longitudinal FHR changes from admission and during the labor of these depressed infants should be presented to staff in the context of a safe learning environment, to identify prevention strategies.

In our experience, as well in the experience of others,[25,26] when longitudinal assessments are performed, compromised fetuses will often exhibit a progression in FHR patterns that is quite predictable, characterized by the sequential development of FHR decelerations, loss of accelerations, significant decelerations, rise of FHR baseline with frequent episodes of tachycardia or continuous tachycardia, minimal baseline variability, worsening variability, absent variability during decelerations, and prolonged or preterminal bradycardia (Figure). This sequence of FHR pattern deterioration should be used to help appropriately time the delivery prior to the development of category III or some of the ominous types of category II FHR patterns. It should be noted that the above sequence of FHR pattern changes will not be seen in fetuses initially found to have category II or III FHR patterns on presentation, since they may already have gone through these changes prior to admission.

In the Figure, the key points in longitudinally evaluating for progressive fetal deterioration are reliance on: (a) minimal, not absent, FHR baseline variability, since it is very hard for individuals to agree or distinguish between the

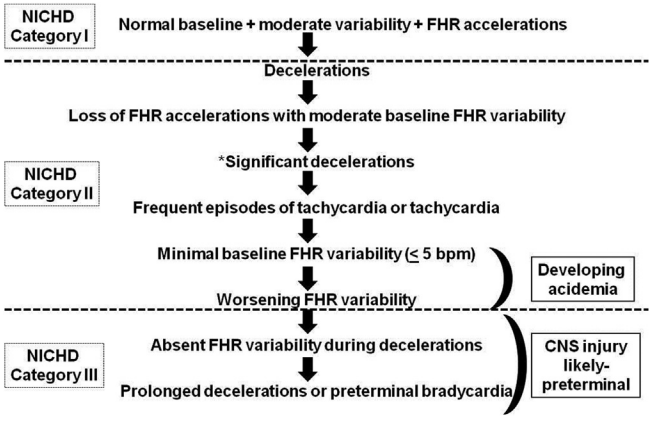
two; and (b) frequent episodes of tachycardia or continuous tachycardia with worsening variability over time. If decelerations are not accompanied by a rise in FHR baseline (or tachycardia) with worsening variability they do not represent fetal hypoxia.[25,26] We have found that most fetuses are developing acidemia when their FHR tracing is still category II, and exhibit tachycardia with decelerations and worsening variability. This is when fetuses progress from adaptation to deterioration. In the absence of a correctable etiology, this may be the most appropriate time for a delivery intervention. By the time that decelerations (prolonged or not) continue with no variability in them and bradycardia sets in, severe fetal acidemia and fetal CNS injury are likely. At this point, it should be emphasized that fetuses can be acidemic while having category II FHR pattern so that transition to category III is not a must for developing acidemia. As a matter of fact, the majority of acidemic fetuses exhibit category II tracings and only 15% will exhibit category III FHR pattern.[27] It should also be noted that even in the absence of prior tachycardia, when a category I FHR pattern suddenly develops bradycardia and converts to category II or III during the second stage of labor, delivery should be effected since this can be an ominous finding. In such cases, regardless of variability, there is correlation between the severity and duration of the bradycardia and fetal acidemia at birth (cord artery pH $\leq$7.10) with the time-to-delivery thresholds being 25, 13, 8, 6, and 5 minutes for bradycardias at 80, 70, 60, 50, and 40 bpm, respectively.[28]

Fetal reserve as well as the nature, severity, chronicity, and duration of the insults during labor will determine the acid-base status and specific FHR pattern. Reduced fetal reserve should be suspected in the presence of certain conditions such as fetal growth restriction, preeclampsia, prematurity, meconium, chorioamnionitis, vaginal bleeding, or pregestational diabetes mellitus.[29] Fetuses with reduced reserve may deteriorate and become acidemic very rapidly during labor so that reduced fetal reserve should be taken into consideration, especially in the interpretation and (more aggressive) management of category II FHR patterns.

Unfortunately, studies examining the relationship of longitudinal FHR pattern changes over time to fetal acid-base status at birth or relevant clinical outcomes are scarce. Studies of FHR patterns over time can be difficult to perform for a number of reasons including difficulty in selecting the appropriate outcomes. We believe that determination of cord blood gases at birth will help us understand and define clinically important FHR pattern abnormalities and is an important outcome to measure. However, the appropriate gold standard should be used for defining the type of acidemia at birth after taking into consideration the presence or absence of active labor.[30] Importantly, the identification of an abnormal FHR tracing often leads to a delivery intervention. In this situation, clinical outcomes can be altered and the natural history outcomes of concerning FHR tracings may not be able to be evaluated. This is confounding by indication and can be hard to address.[31] Nevertheless, efforts to perform longitudinal evaluation studies should be undertaken to understand progression of fetal deterioration and help decide the appropriate timing for delivery. We hope that future studies will focus in the value of longitudinal evaluation of FHR during labor in preventing fetal death and/or CNS injury during labor. ■

FIGURE

Progression of fetal heart rate (FHR) pattern of deteriorating fetus



Progressive fetal heart rate changes.

*Significant decelerations include: (a) variables lasting >60 seconds and reaching nadir <60 beats per minute (bpm) below baseline; (b) variables lasting >60 seconds and reaching nadir <60 bpm regardless of baseline; (c) lates of any depth; (d) prolonged deceleration (>2 to <10 min); and (e) decelerations accompanied by compensatory tachycardia.

CNS, central nervous system; *NICHD*, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development.

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REFERENCES

1. Borell U, Fernstroem I, Ohlson L, Wiqvist N. Effect of uterine contractions on the human uteroplacental blood circulation: an arteriographic study. Am J Obstet Gynecol 1964;89:881-90.
2. Borell U, Fernstroem I, Ohlson L, Wiqvist N. Influence of uterine contractions on the uteroplacental blood flow at term. Am J Obstet Gynecol 1965;93:44-57.
3. Amiel-Tison C, Sureau C, Schnider SM. Cerebral handicap in full-term neonates related to the mechanical forces of labor. Baillieres Clin Obstet Gynaecol 1988;2:145-65.
4. Matsuda Y, Ogawa M, Konno J, Mitani M, Matsui H. Prediction of fetal acidemia in placental abruption. BMC Pregnancy Childbirth 2013;13:156.
5. Usui R, Matsubara S, Ohkuchi A, et al. Fetal heart rate pattern reflecting the severity of placental abruption. Arch Gynecol Obstet 2008;277:249-53.
6. Baumfeld Y, Gutvirtz G, Shoham I, Sheiner E. Fetal heart rate patterns of pregnancies with vasa previa and velamentous cord insertion. Arch Gynecol Obstet 2016;293:361-7.
7. Low JA, Pickersgill H, Killen H, Derrick EJ. The prediction and prevention of intrapartum fetal asphyxia in term pregnancies. Am J Obstet Gynecol 2001;184:724-30.
8. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson ML. Births: final data for 2002. Natl Vital Stat Rep 2003;52:1-113.

9. Nielsen PV, Stigsby B, Nickelsen C, Nim J. Intra- and inter-observer variability in the assessment of intrapartum cardiotocograms. Acta Obstet Gynecol Scand 1987;66:421-4.
10. Chauhan SP, Klauser CK, Woodring TC, Sanderson M, Magann EF, Morrison JC. Intrapartum nonreassuring fetal heart rate tracing and prediction of adverse outcomes: interobserver variability. Am J Obstet Gynecol 2008;199:623.e1-5.
11. Parer JT, Ikeda T. A framework for standardized management of intrapartum fetal heart rate patterns. Am J Obstet Gynecol 2007;197:26.e1-6.
12. 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.e1-8.
13. Macones GA, Hankins GD, Spong CY, 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;112:661-6.
14. Cahill AG, Caughey AB, Roehl KA, Odibo AO, Macones GA. Terminal fetal heart decelerations and neonatal outcomes. Obstet Gynecol 2013;122:1070-6.
15. Duffy CR, Odibo AO, Roehl KA, Macones GA, Cahill AG. Effect of magnesium sulfate on fetal heart rate patterns in the second stage of labor. Obstet Gynecol 2012;119:1129-36.
16. Cahill AG, Roehl KA, Odibo AO, Macones GA. Association of atypical decelerations with acidemia. Obstet Gynecol 2012;120:1387-93.
17. American College of Obstetricians and Gynecologists. Intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. ACOG Practice bulletin no. 106. Obstet Gynecol 2009;114:192-202.
18. Jackson M, Holmgren CM, Esplin ES, et al. Frequency of fetal heart rate categories and short-term neonatal outcome. Obstet Gynecol 2011;118:803-8.
19. Parer TJ, Ikeda T, King TL. The 2008 National Institute of Child Health and Human Development report on fetal heart rate monitoring. Obstet Gynecol 2009;114:136-8.
20. Parer JT, King T, Flanders S, Fox M, Kilpatrick SJ. Fetal acidemia and electronic fetal heart rate patterns: is there evidence of an association? J Matern Fetal Neonatal Med 2006;19:289-94.
21. Clark SL, Nageotte MP, Garite TJ, et al. Intrapartum management of category II fetal heart rate tracings: towards standardization of care. Am J Obstet Gynecol 2013;209:89-97.
22. Ginsburg SJ, Gerstley L. Fetal tachycardia in labor. Am J Obstet Gynecol 1965;92:1132-9.
23. Holzmann M, Wretler S, Cnattingius S, Nordström L. Cardiotocography patterns and risk of intrapartum fetal acidemia. J Perinat Med 2015;43:473-9.
24. Cahill A, Tuuli MG, Stout MJ, Deych E, Shannon W, Macones GA. Electronic fetal monitoring patterns are associated with academia [abstract no. 345]. Am J Obstet Gynecol (Suppl) 2016;214:S194-5.
25. Schifrin BS. The CTG and the timing and mechanism of fetal neurological injuries. Best Pract Res Clin Obstet Gynaecol 2004;18:437-56.
26. Schifrin BS, Ater S. Fetal hypoxic and ischemic injuries. Curr Opin Obstet Gynecol 2006;18:112-22.
27. Di Tommaso M, Seravalli V, Cordisco A, Consorti G, Mecacci F, Rizzello F. Comparison of five classification systems for interpreting electronic fetal monitoring in predicting neonatal status at birth. J Matern Fetal Neonatal Med 2013;26:487-90.
28. Tranquilli AL, Biagini A, Greco P, Di Tommaso M, Giannubilo SR. The correlation between fetal bradycardia area in the second stage of labor and acidemia at birth. J Matern Fetal Neonatal Med 2013;26:1425-9.
29. Oyelese Y. Intrapartum fetal monitoring. In: Apuzzio JJ, Vintzileos AM, Iffy L, eds. Operative obstetrics, 3rd ed. London (United Kingdom); Taylor and Francis: 2006:223-239.
30. Vintzileos AM, Egan JFX, Campbell WA, et al. Asphyxia at birth as determined by cord blood pH measurements in preterm and term gestations: correlation with neonatal outcome. J Matern Fetal Med 1992;1:7-13.
31. Ananth CV, Smulian JC, Vintzileos AM. Epidemiology of antepartum fetal testing. Curr Opin Obstet Gynecol 1997;9:101-6.