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Management of the Category II Fetal Heart Rate Tracing

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Abstract: Management of the category II fetal heart rate (FHR) tracing presents a common challenge in obstetrics. Up to 80% of women will have a category II FHR tracing at some point during labor. Here we propose a management algorithm to identify specific features of the FHR tracing that correlate with risk for fetal acidemia, target interventions to address FHR decelerations, and guide clinicians about when to proceed toward operative vaginal delivery or cesarean to achieve delivery before there is a high risk for significant fetal acidemia with potential for neurological injury or death.
Key words: FHR tracing, labor management, category II

Introduction

The categorization of intrapartum fetal heart rate (FHR) patterns into 3 categories by the National Institute of Child Health and Human Development (NICHD) in 2008 has facilitated communication among care providers and enabled some guidance around intrapartum management.[1] The American College of Obstetricians and Gynecologists (ACOG) recommends that, for category I tracings, ongoing expectant management and routine labor management is appropriate based on the high likelihood of normal fetal acid-base status.[2,3] ACOG indicates that for category III tracings, there is a higher probability of abnormal fetal acid-base status and immediate intervention is required to either resolve the abnormality or proceed with emergent delivery.[2,3] However, there is little specific guidance on the management of the broad range of category II FHR tracings. Indeed, ACOG states that these tracings "require evaluation and continued surveillance and re-evaluation, taking into account the entire associated clinical circumstances."[2] Some 65% to 85% of women will have a category II tracing at some point in labor and having a systematic approach to these tracings is paramount.[4,5]

When faced with an indeterminate, category II FHR tracing, clinicians must ask: *Can fetal metabolic acidosis be reasonably excluded? If not, then how much time remains to achieve delivery or resolution of the abnormalities before a fetal neurological injury*

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might occur? Addressing these questions requires a thorough understanding of FHR physiology to assess current fetal status and inform interventions to improve the FHR tracing. It requires thoughtful consideration of the entire clinical picture to predict the likelihood of labor progression and delivery before fetal compromise. In addition to accounting for maternal and fetal factors, management also requires taking into account the institutional and staffing variables that together determine the ability to expedite delivery when necessary.

Assessment of the current fetal acid-base status involves evaluation of the FHR tracing for variability and the presence of accelerations (spontaneous or induced by scalp stimulation). NICHD classification separates FHR variability into 4 categories: absent, minimal, moderate, and marked. Some have proposed combining these into absent/minimal and moderate/marked to reduce ambiguity and the risk of misclassification of absent as minimal variability due to signal artifact.[6] We have chosen to combine minimal and absent variability to provide a margin of safety. It is generally accepted that the presence of moderate variability and/or the presence of accelerations provides reasonable exclusion of fetal metabolic acidosis.[7] Experts agree that with previously normal fetal acid-base status, acidemia can evolve over about 60 minutes in the setting of episodic oxygen deprivation and subsequent anaerobic metabolism.[7–11] In other words, a woman has about 1 hour to achieve delivery (or for interventions to resolve the abnormal tracing) from the last time that fetal metabolic acidosis could be reasonably excluded by the presence of moderate variability and/or FHR accelerations before the risk of neurological injury from birth asphyxia becomes significantly elevated in the presence of recurrent, significant decelerations. Although the positive predictive value of fetal acidosis based on an abnormal FHR tracing is notoriously poor, this unfortunately remains our primary tool with which to determine intrapartum fetal wellbeing.[7,12] As such, we must still manage labor from within this framework. It is important to remember that acute disruption in fetal oxygen delivery can lead to fetal hypoxemia, hypoxia, and acidemia with risk for asphyxia much more quickly in certain obstetrical emergencies such as uterine rupture, placental abruption, umbilical cord prolapse, ruptured vasa previa or rare cases of maternal medical decompensation. In these cases, usually characterized by sudden and profound FHR decelerations, emergent delivery is required irrespective of the antecedent FHR tracing pattern as in-utero interventions are unlikely to resolve the abnormalities. These events are often accompanied by a category III FHR tracing wherein the need for intervention is clinically obvious, but it is important to acknowledge that when one of these causes is suspected and the tracing remains category II, emergent delivery is still indicated due to the risk for rapid maternal or fetal decompensation.

When emergent delivery does not appear indicated, clinicians should ask: *What is causing impaired oxygen delivery to the fetus resulting in FHR decelerations and are there interventions that may improve oxygenation and resolve or improve the abnormal fetal heart rate tracing?*

A detailed discussion of the physiological basis of FHR decelerations as a reflection of impaired oxygen transfer is discussed in an accompanying article in this symposium (C. Heuser, *Physiology of the fetal heart rate tracing*). Variable decelerations typically reflect cord compression events while late and prolonged decelerations typically reflect poor uteroplacental perfusion and/or poor gas exchange from placental insufficiency. The NICHD nomenclature characterizes decelerations as early, late or variable. No differentiation in severity was proposed for late or variable decelerations based on the depth or duration of the event. Clearly, a variable deceleration to 15 beats below the baseline lasting 15 seconds is not equivalent to one that reaches a nadir of <60 bpm for 60 to 119 seconds with regard to the impact on oxygen transfer to the fetus. As such, many

TABLE 1. Fetal Heart Rate (FHR) Decelerations: Definitions

Type of Deceleration	NICHD Classification*	"Significant Deceleration" Definition†
Variable	Visually apparent *abrupt* decrease in the FHR An *abrupt* FHR decrease is defined as <30 s from onset to FHR nadir The decrease in FHR is calculated from the onset to the nadir of the deceleration The decrease in FHR is ≥15 bpm, lasting ≥15 s, and <2 min in duration When variable decelerations are associated with uterine contractions, their onset, depth and duration commonly vary with successive uterine contractions	Lasting >60 s and falling >60 bpm below the baseline Lasting >60 s with a nadir <60 bpm
Late	Visually apparent usually symmetrical *gradual* decrease and return of the FHR associated with a uterine contraction A *gradual* FHR decrease is defined as from the onset to the FHR nadir of ≥30 s The decrease in FHR is calculated from the onset to the nadir of the deceleration The deceleration is delayed in timing, with the nadir of the decelerations occurring after the peak of the contraction In most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively	*Any late deceleration is significant*
Prolonged	Visually apparent decrease in FHR from the baseline that is ≥15 bpm, lasting ≥2 min, but <10 min	*Any prolonged deceleration is significant*

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NICHD indicates National Institute of Child Health and Human Development.

have proposed the concept of "significant" decelerations. Clark and colleagues published a guideline recommending this designation for any late or prolonged decelerations and for certain variable decelerations[6] (Table 1). These decelerations represent significant disruptions in fetal oxygen delivery that can lead to hypoxemia, progressive tissue oxygen debt, and anaerobic fetal metabolism that in turn leads to fetal metabolic acidosis and risk for neurological injury. We present here an approach to management of category II FHR tracings that uses this nomenclature for risk stratification in certain cases.

Phased Approach to the Category II FHR Tracing

We propose a 4-phase approach to the management of category II FHR tracings as outlined in Figure 1. The goal of the algorithm is to risk-stratify patients based on specific FHR tracing characteristics that correlate with a normal, low, moderate, or high risk for progression to fetal acidemia. This stratification then informs ongoing surveillance as targeted interventions are undertaken to improve fetal oxygen delivery and thereby improve the FHR

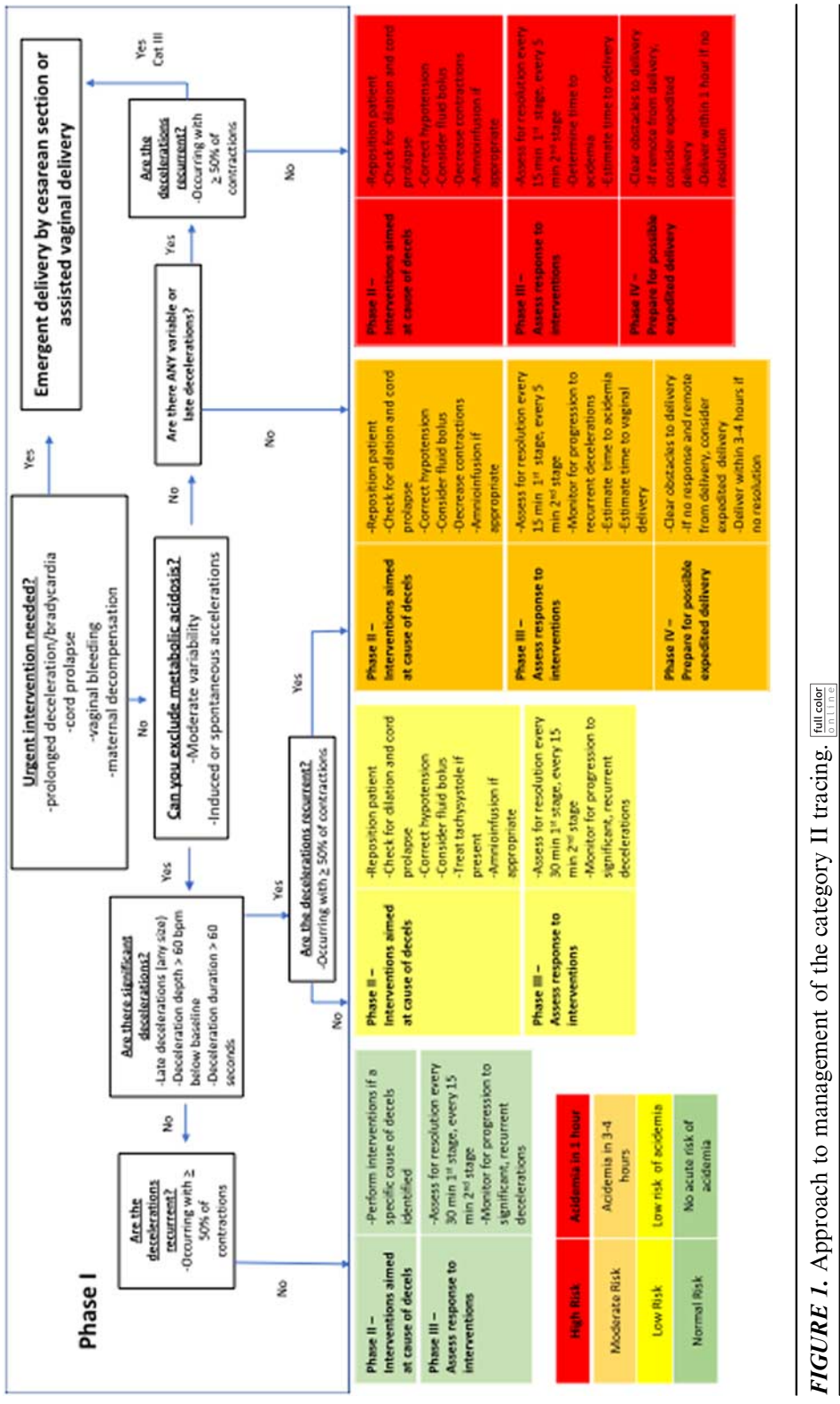

FIGURE 1. Approach to management of the category II tracing. full color online

TABLE 2. Phases of Management for Category II Fetal Heart Rate (FHR) Tracings

Phase I Identify the problem	Evaluate maternal-fetal status, characterize decelerations if present and consider etiology Consider if urgent/emergent intervention is immediately required (cord prolapse, suspected uterine rupture, major placental abruption, maternal decompensation) Consider current fetal acid-base status based on FHR tracing characteristics Establish risk for progression to fetal acidemia as *normal, low, moderate,* or *high*
Phase II Perform targeted interventions	Consider underlying causes for decelerations Variable—*cord compression (oligohydramnios, nuchal/body cords, knots)* Late/prolonged—*poor uteroplacental perfusion from hypotension or tachysystole, impaired gas exchange from abruption or intrinsic insufficiency, maternal hypoxemia* Initiate targeted corrective measures aimed at the likely cause(s) Cord compression—*reposition patient, amnioinfusion* Decreased placental perfusion—*fluid bolus, reposition patient, decrease contraction frequency* Maternal hypoxemia—*maternal O_2 administration*
Phase III Assess response to interventions, estimate time to delivery	Assess response to interventions Establish timeframe for re-evaluation Normal/low risk every 30 min in 1st stage, every 15 min in 2nd stage Moderate/high risk every 15 min in 1st stage, every 15 min in 2nd stage Estimate time before possible onset of acidemia based on tracing characteristics Re-evaluate deceleration severity and frequency Identify concerning markers—absent variability, tachycardia, absent accelerations Estimate time to achieve vaginal delivery Maternal factors—*parity, obstetrical history, labor progress* Fetal factors—*size, station, position*
Phase IV Prepare for expedited delivery	Estimate the "decision-to-delivery" time in case expedited delivery is required Maternal factors, facility/staff availability Remove barriers to rapid delivery Deliver before the likely development of significant academia

tracing placing the fetus in a more favorable risk group. By stratification in this manner, the goal is to provide a timeframe to achieve either delivery or correction of the FHR tracing abnormalities before the anticipated onset of significant fetal metabolic acidosis with risk for neurological injury or neonatal death. The algorithm is meant to provide a framework for intrapartum fetal surveillance. With each reassessment throughout labor, the intent is to return to phase I and reconsider maternal-fetal status along the spectrum from low to high risk for fetal acidemia to inform the plan of care. A description of each phase within the algorithm can be found in Table 2.

PHASE I: EVALUATE MATERNAL-FETAL STATUS AND COMMUNICATE WITH THE CARE TEAM

The first step in the management of any abnormal FHR tracing is the recognition of a potential problem and communicating it effectively to the care team. The Joint Commission published a Sentinel Event Alert about perinatal infant death and permanent disability wherein poor communication

was a root cause in 72% of cases.[13] The importance of communication in the management of category II FHR tracings cannot be overstated. Phase I represents this early recognition of an abnormal FHR tracing, accurate clinical description of the abnormalities, consideration of maternal and fetal risk factors, and effective communication between nursing and providers to create a shared mental model of the clinical situation and current risk of acidemia. The first issue to address when confronted with an abnormal FHR tracing is whether urgent delivery is required to avoid rapid progression to fetal acidemia because in-utero interventions are unlikely to be effective *or* because of concern for sudden maternal decompensation. These include the obstetrical emergencies discussed above such as uterine rupture, cord prolapse, placental abruption, or acute change in maternal status. If emergent delivery is not indicated, then attention should turn to whether or not the tracing shows sufficient reassuring features as to reasonably exclude fetal metabolic acidosis—namely moderate variability and/or the presence of induced or spontaneous accelerations.

If metabolic acidosis *cannot* be reasonably excluded, then the clinician should come to the bedside to evaluate the patient and communicate directly with the patient and care team. Immediate interventions to optimize fetal oxygen delivery should be instituted based on clinical context. If there are any *recurrent* variable or late decelerations, then this represents a category III FHR tracing requiring immediate intervention and delivery if corrective measures fail to improve the tracing within 30 minutes while preparations are made for delivery. If metabolic acidosis *can* be reasonably excluded, the next step in evaluation entails assessment for the presence of significant FHR decelerations (Table 1). When present, they should be characterized as recurrent or nonrecurrent for risk stratification. Once the initial assessment is complete, the need for immediate delivery excluded, and the team has assigned the fetal risk status (normal, low, moderate, or high), then management moves into phase II.

PHASE II: ONGOING SURVEILLANCE AND INTERVENTIONS TO IMPROVE THE FHR TRACING

With a category II FHR tracing wherein metabolic acidosis can be reasonably excluded (moderate variability and/or accelerations) and there are *no significant* decelerations, then a low risk obstetrical patient can be managed expectantly with routine surveillance per usual ACOG criteria (every 30 min in the first stage, every 15 min in the second stage). These are patients still considered normal/low risk for fetal acidosis. If the patient has other obstetrical risk factors (eg, hypertension, diabetes, fetal growth abnormalities), then heightened surveillance is warranted (every 15 min in the first stage, every 5 min in the second stage).[2] If metabolic acidosis can be reasonably excluded (moderate variability and/or accelerations) but *significant* decelerations *are* present, interventions to optimize oxygen delivery should be considered based on the clinical context. If these decelerations are sporadic in an otherwise low risk patient, routine surveillance is still appropriate. If there are *recurrent significant* decelerations, then heightened surveillance is warranted (every 15 min in the first stage, every 5 min in the second stage) with close attention to ongoing FHR variability and the presence of accelerations to assess fetal wellbeing.[2]

Phase II also involves targeted interventions to improve fetal oxygenation and reduce the frequency and severity of FHR decelerations, particularly in the moderate and high-risk groups. Variable and late FHR decelerations represent interruptions in fetal oxygen delivery. Common causes of reduced oxygen delivery that may be amenable to intervention include maternal hypotension, umbilical cord compression, and uterine tachysys-

tole. A review of the common causes of interrupted oxygen delivery from mother to fetus and common intrapartum interventions to optimize fetal oxygenation is discussed in an accompanying article in this symposium (A. Eller, *Interventions for intrapartum fetal heart rate abnormalities.*). Maternal left lateral positioning is recommended to relieve aortocaval compression and improve uteroplacental perfusion. Administration of a maternal fluid bolus can address hypotension and hypovolemia. Pharmacologic intervention should also be considered to correct iatrogenic hypotension following regional anesthesia. When significant variables are encountered, a vaginal examination to exclude occult cord prolapse and the need for emergent delivery is required. When cord prolapse has been excluded, consideration should be given to amnioinfusion if cord compression is likely (eg, recent rupture of membranes or known oligohydramnios). Exogenous oxytocin should be discontinued or reduced during stimulated labor when decelerations are present in the context of uterine tachysystole. Tocolysis with the short-acting beta-agonist terbutaline may also be considered for tachysystole when significant FHR decelerations are present. Supplemental maternal oxygen should be reserved for cases of suspected or confirmed maternal hypoxemia. Interventions to improve the FHR tracing should be directed at the most likely cause of interrupted oxygen delivery between mother and fetus.

PHASE III: ASSESS RESPONSE TO INTERVENTIONS, ANTICIPATE TIME TO POSSIBLE FETAL ACIDOSIS

Phase III involves assessment of the fetal response to interventions as evidenced by improvement in the FHR tracing. Consideration must be given to the likelihood of achieving a vaginal delivery before the possible onset of fetal acidemia and potential neurological injury in a moderate or high-risk patient with a category II FHR tracing.

The timeframe to achieve delivery or improvement in the FHR tracing before possible fetal acidemia is ~1 hour from the last time when moderate variability and/or FHR accelerations were noted. If there is minimal/absent variability and no induced or spontaneous accelerations, then fetal acidosis can no longer be excluded. If there are any *recurrent* late or variable decelerations, this represents transition to an essentially category III FHR tracing (acknowledging that the NICHD definition uses *absent* variability to define a category III FHR tracing) and immediate delivery is recommended if initial interventions do not improved the FHR tracing within 30 minutes. If there is minimal/absent variability with no FHR accelerations and there are *any* significant decelerations, the patient should be considered high risk for fetal acidosis and delivery should occur within 1 hour if the tracing does not improve with interventions. When no FHR decelerations are noted but there is minimal/absent variability and the absence of induced or spontaneous accelerations the patient should be considered at moderate risk for fetal acidemia. The team should remain on high alert and aim for delivery within 3 to 4 hours if no improvement in the FHR tracing occurs. In such cases, the onset of *any* variable or late decelerations should prompt more rapid intervention as the progression to acidemia may occur more rapidly in that clinical setting. When metabolic acidosis can still be reasonably excluded but there are *recurrent significant* decelerations, the patient should also be considered at moderate risk for progression to fetal acidemia. Over time, this recurrent disruption in fetal oxygen delivery (evidenced by the significant decelerations) leads to accumulating fetal oxygen debt, reduced buffering capacity in the fetal blood as evidenced by increasing base deficit, and a gradual decline in fetal blood pH making the fetus more vulnerable to acute decompensation and progression to

significant acidemia. This likely occurs over a period of 3 to 4 hours in a fetus with a previously normal acid-base status based on episodic cord occlusion studies in ovine animal models.[14–16] During this phase, ongoing attention to the presence of FHR variability and accelerations (induced or spontaneous) is particularly important to reasonably exclude the presence of fetal acidemia. Reduction in FHR variability, the loss of induced accelerations, and the development of fetal tachycardia in the context of a category II FHR tracing should raise concern about the potential for progression to acidemia. Thus, it is imperative to retain situational awareness about the estimated time to delivery taking into account the entire clinical picture. This includes such factors as parity, obstetrical history, labor progress, maternal expulsive efforts, estimated fetal weight, position, station, and potential contraindications to operative vaginal delivery. Clinicians must take into consideration a multitude of maternal and fetal factors in estimating how long it may take the patient to achieve vaginal delivery and how much longer to attempt corrective interventions before proceeding to operative vaginal delivery or cesarean.

PHASE IV: PREPARE FOR EXPEDITED DELIVERY

When interventions fail to resolve significant decelerations and spontaneous vaginal delivery is not imminent, management moves into phase IV, preparation for expedited delivery. The team must consider the potential barriers to achieving rapid delivery if the tracing deteriorates further or delivery cannot be achieved within the acceptable timeframe. Obstetrical providers often refer to the "decision to delivery time," which refers to the anticipated time it will take to actually achieve delivery once the decision has been made to actively proceed with an operative birth (vaginal or cesarean). This varies widely depending on a multitude of maternal, fetal, and institutional factors. Maternal variables such as obstetrical and surgical history, body mass index, medical comorbidities and the presence of regional anesthesia all play a significant role in how quickly delivery might be accomplished. In some cases, maternal willingness to undergo recommended interventions can present a significant barrier and the care team should identify this early in the labor whenever possible to enable sufficient anticipatory guidance about the potential need for operative delivery or cesarean and obtain informed consent if tracing abnormalities arise. When the need for expedited delivery is anticipated, regional anesthesia should be strongly considered if not already in place. Fetal variables include estimated weight, position, station and potential contraindications to operative vaginal delivery. Facility and staffing variables include the immediate availability of an anesthesiologist, a provider capable of performing a cesarean or operative vaginal delivery (and their skill level and comfort with the procedures), an available operating room and personnel, and pediatric staff for potential neonatal resuscitation. Estimating the decision-to-delivery time and using the 60 minute estimate for the evolution of acidemia, providers must extrapolate the residual time available to either achieve a spontaneous vaginal delivery or for interventions to improve the FHR tracing before proceeding with expedited delivery to reduce the risk of fetal injury.

Use of the Algorithm

In the practice of obstetrics, algorithms may help guide the care team but the unique characteristics of each maternal-fetal pair must always be taken into consideration to provide optimal care. This phased approach to evaluation and management of indeterminate category II FHR tracings is meant to be dynamic. Ongoing surveillance should provide reassessment of fetal risk over time

as interventions are undertaken to improve or resolve FHR decelerations. For example, an initially moderate risk fetus with recurrent variable decelerations in the setting of moderate variability and accelerations may become low risk if interventions lead to resolution of the decelerations. Likewise, a normal or low risk fetus can become moderate or high risk if recurrent, significant decelerations develop or when there is evolution of concerning features such as loss of variability and/or accelerations or the development of fetal tachycardia. Heightened vigilance is also required when there are concurrent clinical events, such as the development of maternal fever or severe hypertension. It is important to remember that an acute change in maternal or fetal status should always prompt return to phase I and evaluation for clinical events that require urgent/emergent intervention regardless of the antecedent tracing. Finally, the utility of any algorithm for intrapartum care relies heavily on team communication between nursing and physicians or midwives. The importance of effective ongoing communication between members of the care team cannot be overstated; maintaining a shared mental model of the clinical situation is the cornerstone of safe and effective maternal care. Category II FHR tracings are common in obstetrics and a systematic approach to evaluation, risk stratification, and therapeutic intervention is paramount to providing optimal care. We hope that this phased approach provides guidance to nurses and clinicians in the management of these sometimes challenging cases.

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