OBG MANAGEMENT

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IN THIS ARTICLE

OBSTETRIC EMERGENCIES

Management of prolonged decelerations

Some are benign, some are pathologic but reversible, and others are the most feared complications in obstetrics

A prolonged deceleration may signal danger—or reflect a perfectly normal fetal response to maternal pelvic examination. Because of the wide range of possibilities, this fetal heart rate pattern justifies close attention. For example, repetitive prolonged decelerations may indicate cord compression from oligohydramnios. Even more troubling, a prolonged deceleration may occur for the first time during the evolution of a profound catastrophe, such as amniotic fluid embolism or uterine rupture during vaginal birth after cesarean delivery (VBAC). In some circumstances, a prolonged deceleration may be the terminus of a progression of nonreassuring fetal heart rate (FHR) changes, and becomes the immediate precursor to fetal death (**TABLE 1**).[1]

When FHR patterns exhibit these aberrations, we rightly worry about fetal well-being and the possible need for operative intervention. Unfortunately, the degree of fetal compromise is difficult to predict and depends on preexisting fetal condition, physiologic reserve, degree and duration of the insult, and other variables.

Ultimately, a judgment call

The 22nd edition of *Williams Obstetrics*[2] summarizes the clinical challenges involved in the management of prolonged decelerations during labor: "Management of isolated prolonged decelerations is based on bedside clinical judgment, which inevitably will sometimes be imperfect given the unpredictability of these decelerations."

"Fetal bradycardia" and "prolonged deceleration" are distinct entities

In general parlance, we often use the terms "fetal bradycardia" and "prolonged deceleration" loosely. In practice, we must differentiate these entities because underlying pathophysiologic mechanisms and clinical management may differ substantially.

The problem: Since the introduction of electronic fetal monitoring (EFM) in the 1960s, numerous descriptions of FHR patterns have been published, each slightly different from the others. The result: confusing nomenclature, miscommunication among clinicians, and stymied research efforts.

To standardize definitions of intrapartum FHR patterns so that the effectiveness of EFM could be better assessed in observational studies and clinical trials, the National Institute of Child Health and Human Development organized a workshop.[3] Its recommendations were subsequently adopted by the American College of Obstetricians and Gynecologists (ACOG).[4] Among the definitions:

- **Bradycardia:** a baseline FHR of less than 110 beats per minute.

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TABLE 1

Some causes of prolonged decelerations and bradycardias

PROLONGED DECELERATION	BRADYCARDIA
Cord compression Oligohydramnios Cord prolapse	**Congenital conduction abnormalities** Complete heart block Long QT syndrome Congenital heart defects Tachyarrhythmia (Fetal tachyarrhythmia may produce an EFM tracing that appears to be a bradycardia and can only be distinguished by ultrasound)
Uteroplacental insufficiency Anesthesia (paracervical, spinal, epidural) Maternal valsalva Maternal supine hypotension Hypertonic or prolonged contractions Abruptio placentae Uterine rupture Cocaine ingestion	**Medications** Beta blockers
Maternal hypoxia Maternal seizures, eclampsia Respiratory depression from medications Cardiopulmonary arrest Amniotic fluid embolism	**Hypothermia**
Fetal hemorrhage Vasa previa Traumatic amniocentesis	**Infection** Chorioamnionitis Endotoxemia
Fetal vagal reaction Rapid descent, impending birth Cervical examination Fetal scalp electrode placement Fetal blood sampling	
Fetal central nervous system anomalies	
Idiopathic (cord compression?)	

- **Prolonged deceleration:** a visually apparent decrease of 15 or more beats per minute below the baseline. This decrease lasts at least 2 minutes but less than 10 minutes from onset to the return to baseline (≥10 minutes is considered a baseline change).

Differentiation between the 2 entities is critical because, in many cases, bradycardias are chronic patterns that may not be associated with immediate fetal compromise and do not require immediate intervention. For example, a fetal bradycardia due to congenital heart block would not benefit from immediate delivery, especially prior to term.

"Moderate fetal bradycardia," defined as a baseline of 100 to 119 bpm, was reported in 1.8% of 1,386 continuously monitored patients and is attributed to relative cephalopelvic disproportion, resolving after rotation of the fetal vertex and associated with normal neonatal outcome.[5,6]

Similar decelerations can reflect different events

The exact depth and duration of a prolonged deceleration leading to fetal compromise and requiring prompt delivery is difficult to define, although some observations warrant consideration. Experiments with fetal lambs show that the deceleration in response to umbilical vein occlusion is associated with a fall in fetal blood pressure, whereas deceleration in response to umbilical artery occlusion is associated with a rise in fetal blood pressure. This reflex can be abolished by vagotomy, but will eventually recur due to anoxia.[7]

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Fetal bradycardias and prolonged decelerations are 2 distinct entities; the first usually does not warrant immediate intervention

Vital clue: What happened before the prolonged deceleration?

In clinical practice, it is important to appreciate characteristics of the FHR pattern preceding the prolonged deceleration.[8] Williams and Galerneau[9] correlated baseline FHR variability and duration of prolonged decelerations with neonatal acid–base status in 186 term gestations with an identified prolonged deceleration within 30 minutes of delivery. Patients were divided into 4 groups, based on FHR variability and recovery of the FHR baseline (**TABLE 2**).

The findings:

- **Lowest cord pH** was associated with decreased variability without recovery of the FHR
- **Neonatal acidemia** was most strongly correlated with decreased variability before the prolonged deceleration

Acid–base changes likely begin within minutes of cord compression

Zilianti and colleagues[10] evaluated 29 fetuses with normal FHR patterns during labor with FHR deceleration during the expulsion phase of delivery. When the FHR deceleration was prolonged (>120 seconds), umbilical artery pH significantly decreased (7.19 vs 7.27), umbilical vein pH remained unchanged (7.32), and the umbilical venous–arterial pH difference was significantly increased (0.13 vs 0.05). Thus, acid–base changes likely begin within minutes of cord compression.

The correlation between acidemia and loss of variability

In their review of 43 vacuum extractions, Gull and colleagues[22] found that 27 infants were delivered for "end-stage bradycardia" (abrupt persistent decrease in FHR to less than 100 bpm for more than 2 minutes, or repeated deceleration more than 60 bpm below baseline with poor recovery), and 16 were delivered electively (controls). Umbilical-cord base deficit was greater in the newborns with bradycardia than in controls, and the length of time FHR variability was lost correlated with the degree of base deficit. Acidemic fetuses

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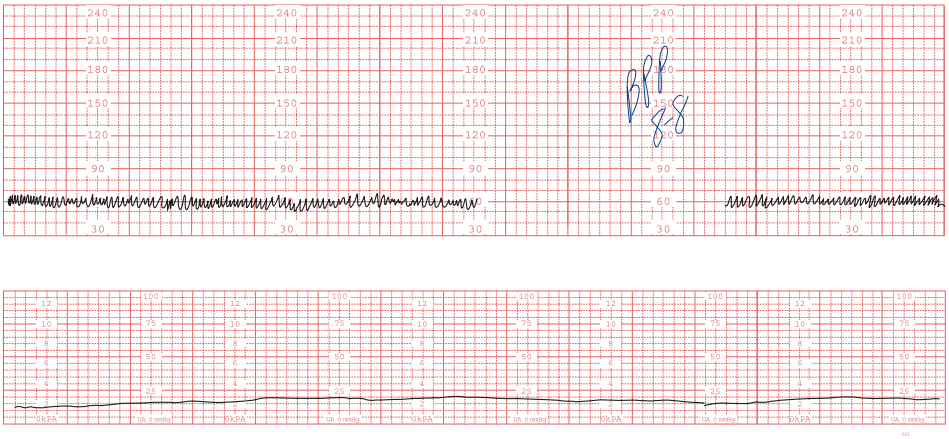
3 fetal heart rate patterns: What would you do?

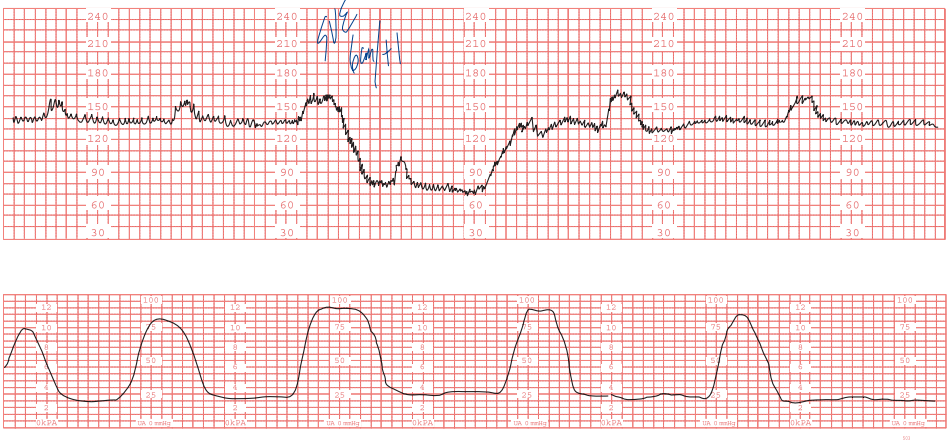
Complete heart block



Dilemma
Fetal bradycardia due to congenital complete heart block secondary to anti-SS-A/Ro and anti-SS-B/La antibodies. The fetal ventricular rate is fixed at 60 bpm

Management
At 30 weeks' gestation, with no sonographic evidence of heart failure and a biophysical profile score of 8/8, expectant management is indicated

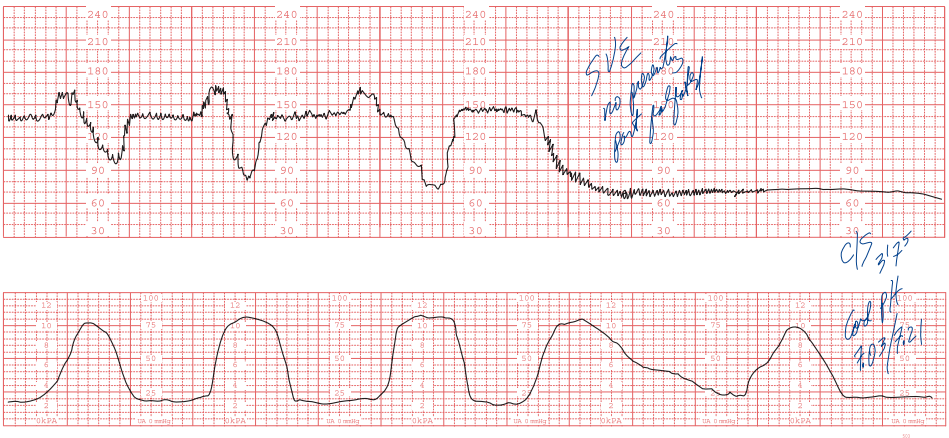
Prolonged deceleration during pelvic exam



Dilemma
Prolonged deceleration during pelvic examination in an uncomplicated term pregnancy. Note that fetal heart rate (FHR) variability was maintained during recovery of the FHR baseline

Outcome
Uneventful spontaneous vaginal delivery

Uterine rupture



Dilemma
Prolonged deceleration due to uterine rupture during trial of labor after cesarean. Repetitive variable decelerations preceded the prolonged deceleration. FHR variability was lost after several minutes

Outcome
Emergency cesarean

TABLE 2

Neonatal outcomes associated with variability and recovery of FHR patterns after prolonged deceleration

UMBILICAL ARTERY	GROUP 1 V+ R+ (N = 128)	GROUP 2 V+ R- (N = 40)	GROUP 3 V- R+ (N = 9)	GROUP 4 V- R- (N = 9)	P VALUE
pH (mean ± SD)	7.17 ± 0.09	7.13 ± 0.15	7.11 ± 0.11	6.83 ± 0.16	<.001
Base deficit (mean ± SD)	-6.5 ± 3.9	-7.2 ± 5.1	-10 ± 4	-20 ± 6	<.001
pH <7.0 (%)	2	18	44	78	<.001
pH <7.1 (%)	22	33	56	89	<.001
Base deficit <16 (%)	1	8	11	78	<.001
Base deficit <12 (%)	5	13	22	89	<.001

V = variability
R = recovery
SOURCE: Williams and Galerneau[9]

lost FHR variability during the bradycardia for more than 4 minutes, or started to lose FHR variability less than 3 minutes from the beginning of the bradycardia.

What is optimal interval between deceleration and delivery?

In a series of 106 cases of uterine rupture during VBAC, Leung et al[11] found significant neonatal morbidity when 18 minutes or more lapsed between the onset of the prolonged deceleration and delivery.

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Amnioinfusion for cord compression reduces variable FHR decelerations and the need for cesarean section

First, remain calm when decelerations occur

Freeman and colleagues[12] advocate staying calm and avoiding overreaction, because many cases will resolve spontaneously. Nonetheless, prolonged decelerations should prompt the physician to:

- consider the underlying pathophysiology and implement corrective interventions (**TABLE 3**)
- further assess fetal condition
- prepare for the possibility of immediate delivery (**TABLE 4**)

Consider amnioinfusion when cord compression is suspected

Many cases of prolonged decelerations are secondary to cord compression resulting from oligohydramnios. Miyazaki[13] showed that saline amnioinfusion helped correct the FHR problem in most cases of repetitive variable decelerations (19 of 28) and prolonged decelerations (12 of 14 cases).

Several randomized clinical trials analyzed in a recent Cochrane Review[14] suggest that amnioinfusion for cord compression reduces the occurrence of variable FHR decelerations and the need for cesarean section; this applies to settings in which nonreassuring FHR patterns were not further assessed by fetal blood sampling, which is reflective of practice in most US labor units.

The recent ACOG practice bulletin on intrapartum monitoring[4] advocates amnioinfusion for recurrent variable FHR decelerations, but does not address prolonged decelerations specifically.

Although most data on amnioinfusion address treatment of recurrent variable FHR decelerations, it also seems reasonable to consider this option for prolonged decelerations when oligohydramnios is suspected.[12]

Other possible causes of prolonged decelerations

Vasa previa. A sudden prolonged deceleration following rupture of membranes with concomitant vaginal bleeding should prompt the physician to consider the pos-

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TABLE 3

6 pearls for managing prolonged decelerations

	GOAL	PEARL
1	Reduce aorto-caval and/or cord compression	Change patient positioning
2	Restore intravascular volume	Administer intravenous fluid bolus
3	Reduce uterine activity	Discontinue oxytocin drip and give tocolytic therapy (terbutaline)
4	Enhance oxygen delivery to fetus	Give supplemental oxygen
5	Resolve hypotension	Administer vasopressor therapy (ephedrine)
6	Resolve oligohydramnios and cord compression	Perform transcervical amnioinfusion

sibility of a disrupted velamentous cord insertion (vasa previa), which can lead to rapid fetal exsanguination.[15]

Acute profound maternal hypoxemia may lead to a first prolonged FHR deceleration, often preceded by increased uterine tone, as described in both eclampsia[16] and amniotic fluid embolism.[17] With eclampsia, the prolonged deceleration is reversible; treatment and expectant management will allow for fetal recovery after the seizure abates.

When acute amniotic fluid embolism leads to profound cardiovascular collapse, prompt perimortem cesarean delivery may be required within minutes if CPR does not restore normal maternal cardiopulmonary function and recovery of FHR.

When is scalp stimulation helpful?

Stimulation of the fetal scalp is an effective technique for assessing fetal status during periods of nonreassuring FHR patterns.[18] However, the technique is intended to be performed during periods of FHR baseline and is sometimes misapplied during prolonged decelerations. Scalp stimulation during a prolonged deceleration would not likely provide valid information or change clinical management and could in theory exacerbate fetal compromise if additional parasympathetic tone were elicited.

Avoid fetal pulse oximetry

Although fetal pulse oximetry is FDA-approved and commercially available in the United States, and may be well suited for monitoring fetal arrhythmias,[19,20] a prolonged deceleration is an absolute contraindication to its use.[21]

Summary

Overall, in managing a delivery marked by prolonged decelerations, we should strive to minimize maternal–fetal complications by carefully assessing the clinical situation, correcting reversible problems, and preparing for expeditious delivery if the fetal condition is of sufficient concern that further expectant management is unlikely to allow for safe spontaneous delivery. Still, "...bedside judgment inevitably will sometimes be imperfect given the unpredictability of these decelerations."[2] ■

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Fetal scalp stimulation to assess fetal status should be done during periods of FHR baseline

TABLE 4

Stepwise management of prolonged decelerations

Examine the cervix
- Check for umbilical cord prolapse
- Check progress of dilation and descent
- Place internal monitors, if indicated

Determine probable cause

Start therapies

Prepare for intervention by operative delivery
- Intravenous access
- Blood type and screen
- Indwelling urinary catheter
- Obtain consents for operative vaginal delivery and cesarean delivery
- Notify appropriate personnel (eg, anesthesiology, pediatrics)

Deliver
- If fetal condition is nonreassuring despite therapies
- If prolonged decelerations recur and spontaneous delivery is remote (cases must be individualized)

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