Electronic fetal monitoring (EFM) is a popular technology used to establish fetal well-being. Despite its widespread use, the terminology used to describe patterns seen on the monitor has not been consistent until recently. In 1997, the National Institute of Child Health and Human Development (NICHD) Research Planning Workshop published guidelines for interpretation of fetal tracings. This publication was the culmination of 2 years of work by a panel of experts in the field of fetal monitoring, and was endorsed in 2005 by both the American College of Obstetricians and Gynecologists (ACOG) and the Association of Women’s Health, Obstetric and Neonatal Nurses (AWHONN). In 2008, ACOG, NICHD, and the Society for Maternal-Fetal Medicine reviewed and updated the definitions for fetal heart rate (FHR) patterns, interpretation, and research recommendations. Following is a summary of the terminology definitions and assumptions found in the 2008 NICHD workshop report. Normal values for arterial umbilical cord gas values and indications of acidosis are defined in Table 1.

Assumptions from the NICHD Workshop
- Definitions are developed for visual interpretation, assuming that both the FHR and uterine activity recordings are of adequate quality
- Definitions apply to tracings generated by internal or external monitoring devices
- Periodic patterns are differentiated based on waveform, abrupt or gradual (eg, late decelerations have a gradual onset and variable decelerations have an abrupt onset)
- Long- and short-term variability are evaluated visually as a unit
- Gestational age of the fetus is considered when evaluating patterns
- Components of FHR do not occur alone and generally evolve over time

DEFINITIONS

Baseline Fetal Heart Rate
- Approximate mean FHR rounded to increments of 5 beats per minute in a 10-minute segment of tracing, excluding accelerations and decelerations, periods of marked variability, and segments of baseline that differ by >25 beats per minute
- In the 10-minute segment, the minimum baseline duration must be at least 2 minutes (not necessarily contiguous) or the baseline for that segment is indeterminate
- Bradycardia is a baseline of <110 beats per minute; tachycardia is a baseline of >160 beats per minute
Sinusoidal baseline has a smooth sine wave–like undulating pattern, with waves having regular frequency and amplitude

Baseline Variability
- Fluctuations in the baseline FHR of ≥2 cycles per minute, fluctuations are irregular in amplitude and frequency, fluctuations are visually quantitated as the amplitude of the peak to trough in beats per minute
- Classification of variability:
  - Absent: Amplitude range is undetectable
  - Minimal: Amplitude range is greater than undetectable to 5 beats per minute
  - Moderate: Amplitude range is 6–25 beats per minute
  - Marked: Amplitude range is >25 beats per minute

Accelerations
- Abrupt increase in FHR above the most recently determined baseline
- Onset to peak of acceleration is <30 seconds, acme is ≥15 beats per minute above the most recently determined baseline and lasts ≥15 seconds but <2 minutes
- Before 32 weeks’ gestation, accelerations are defined by an acme ≥10 beats per minute above the most recently determined baseline for ≥10 seconds
- Prolonged acceleration lasts ≥22 minutes but <10 minutes

Late Decelerations
- Gradual decrease in FHR (onset to nadir ≥30 seconds) below the most recently determined baseline, with nadir occurring after the peak of uterine contractions
- Considered a periodic pattern because it occurs with uterine contractions

Early Decelerations
- Gradual decrease in FHR (onset to nadir ≥30 seconds) below the most recently determined baseline, with nadir occurring coincident with uterine contraction
- Also considered a periodic pattern

Variable Decelerations
- Abrupt decrease in FHR (onset to nadir <30 seconds)
- Decrease is ≥15 beats per minute below the most recently determined baseline lasting ≥15 seconds but <2 minutes
- May be episodic (occurs without a contraction) or periodic

Prolonged Decelerations
- Decrease in the FHR ≥15 beats per minute below the most recently determined baseline lasting ≥2 minutes but <10 minutes from onset to return to baseline
  - Decelerations are tentatively called recurrent if they occur with ≥50% of uterine contractions in a 20-minute period
  - Decelerations occurring with <50% of uterine contractions in a 20-minute segment are intermittent

Sinusoidal Fetal Heart Rate Pattern
- Visually apparent, smooth sine wave–like undulating pattern in the baseline with a cycle frequency of 3 to 5 per minute that persists for ≥20 minutes

Uterine Contractions
- Quantified as the number of contractions in a 10-minute window, averaged over 30 minutes
  - Normal: ≤5 contractions in 10 minutes
  - Tachysystole: >5 contractions in 10 minutes

INTERPRETATION
A 3-tier FHR Interpretation system has been recommended as follows:
- Category I FHR tracings: Normal, strongly predictive of normal fetal acid-base status and require routine care. These tracings include all of the following:

### TABLE 1. Arterial Umbilical Cord Gas Values

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>Pco₂ (mm Hg)</th>
<th>Po₂ (mm Hg)</th>
<th>BASE EXCESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal*</td>
<td>≥7.20 (7.15 to 7.38)</td>
<td>&lt;60 (35 to 70)</td>
<td>≥20</td>
<td>≤-10 (–2.0 to –9.0)</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>&lt;7.20</td>
<td>&gt;60</td>
<td>Variable</td>
<td>≤-10</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>&lt;7.20</td>
<td>&lt;60</td>
<td>Variable</td>
<td>≥-10</td>
</tr>
<tr>
<td>Mixed acidosis</td>
<td>&lt;7.20</td>
<td>&gt;60</td>
<td>Variable</td>
<td>≥-10</td>
</tr>
</tbody>
</table>

Baseline rate: 110 to 160 beats per minute
Baseline FHR variability: Moderate
Late or variable decelerations: Absent
Early decelerations: Present or absent
Accelerations: Present or absent

- Category II FHR tracings: Indeterminate, require evaluation and continued surveillance and reevaluation. Examples of these tracings include any of the following:
  - Bradycardia not accompanied by absent variability
  - Tachycardia
  - Minimal or marked baseline variability
  - Absent variability without recurrent decelerations
  - Absence of induced accelerations after fetal stimulation
  - Recurrent variable decelerations with minimal or moderate variability
  - Prolonged decelerations
  - Recurrent late decelerations with moderate variability
  - Variable decelerations with other characteristics, such as slow return to baseline

- Category III FHR tracings: Abnormal, predictive of abnormal fetal acid-base status and require prompt intervention. These tracings include:
  - Absent variability with any of the following:
    - Recurrent late decelerations
    - Recurrent variable decelerations
    - Bradycardia
  - Sinusoidal pattern


We encourage readers to examine each strip in the case presentation and make a personal interpretation of the findings before advancing to the expert interpretation provided.

PRESENTATION

History
A 37-year-old gravida 5, para 3-0-1-3 at 39 weeks and 0 days' gestation presented to the labor and delivery department for scheduled elective induction of labor. She denied any bleeding, contractions, loss of fluid, or other complaints. The patient’s pregnancy was complicated by advanced maternal age. She underwent cell-free fetal DNA screening, which found her to be at low risk for aneuploidy. She underwent level II ultrasonography at 19 weeks and 5 days’ gestation, which demonstrated normal fetal anatomy. A posterior placenta was documented without notable placental abnormalities. The placental cord insertion was not well visualized. She had no other prenatal issues. Her obstetric history was notable for 3 prior full-term normal spontaneous vaginal deliveries without complications. She had no significant medical, surgical, social, or family history.

On admission, her blood pressure was 115/62 mm Hg, heart rate 68 beats per minute, and temperature 98.2°F. A vaginal examination revealed her cervix to be 1-cm dilated, 25% effaced, and -3 station. Bedside ultrasonography confirmed vertex presentation. Her induction of labor began using an intracervical Foley catheter with extra-amniotic saline infusion, concurrent with oxytocin administration as per institutional protocol. The initial FHR tracing at the time of admission is shown in Fig 1.

![Figure 1](http://neoreviews.aappublications.org/)

Figure 1. Electronic fetal monitoring strip 1.
Findings from EFM strip 1:

- Variability: Moderate
- Baseline rate: 130 beats per minute
- Episodic patterns: Accelerations
- Periodic patterns: None
- Uterine contractions: None

Interpretation: Category I
- Differential diagnosis: Normally oxygenated infant
- Action: Proceed with induction of labor

About 5½ hours after initiation of labor induction, early decelerations were noted on FHR tracing for a period of 15 minutes. The tracing at this time is demonstrated in Fig 2.
Findings from EFM strip 2:
- Variability: Moderate
- Baseline rate: 140 beats per minute
- Episodic patterns: None
- Periodic patterns: Early decelerations
- Uterine contractions: Every 3 minutes
- Interpretation: Category I
- Differential diagnosis: Normally oxygenated infant, fetal head compression
- Action: Continue with induction of labor

Approximately 6 hours after initiation of the extra-amniotic saline infusion, the intracervical balloon was removed. The patient’s cervix was 5-cm dilated, 50% effaced, and 0 station. Labor induction continued with oxytocin as per the protocol, and 2 hours later, her cervical examination findings were unchanged. At this point, membranes were artificially ruptured, with minimal leakage of clear amniotic fluid noted.

Approximately 1½ hours later, a cervical examination was repeated, which showed 6-cm dilation, 50% effacement, and 0 station. Given the minimal amount of fluid leakage on previous artificial rupture of the membranes, a finger was used to gently strip along the membranes to allow more fluid to exude. During the digital examination, approximately 200 mL of dark red blood was observed. The fetal heart rate tracing was then uninterpretable and a fetal scalp electrode was placed immediately. Once the fetal scalp electrode was in place, a prolonged fetal deceleration was seen, with fetal heart tones ranging between 40 and 70 beats per minute, for a duration of 4 minutes (Fig 3). At this point, the decision was made to proceed with emergent cesarean delivery.
Findings from EFM strip 3:

- Variability: Moderate at the beginning of tracing, followed by absent variability for the last 5 minutes
- Baseline rate: Indeterminate
- Episodic patterns: Prolonged deceleration with a nadir to 40 beats per minute
- Periodic patterns: None
- Uterine contractions: Unable to determine
- Interpretation: Category III
- Differential diagnosis: Acute placental abruption, uteroplacental insufficiency, umbilical cord compression, abnormal placental umbilical cord insertion, vasa previa with rupture of fetal vessel

Action: The patient was moved to the operating room for emergent cesarean delivery

The patient arrived in the operating room 7 minutes after the detection of the fetal deceleration (12 minutes from the onset of uninterpretable tracing). The incision was made 3 minutes after arrival at the operating room and the infant was delivered 2 minutes after a low transverse uterine incision was made in the skin. No intraoperative difficulties were encountered. However, inspection of the placenta after delivery revealed evidence of velamentous cord insertion (VCI) across the fetal membranes with a ruptured fetal vessel (Fig 4).

Figure 3. Electronic fetal monitoring strip 3.

Figure 4. Gross examination of placental membranes. Arrow points to site of fetal vessel rupture.
Outcome

A viable male infant weighing 3,070 g (6 lb 12 oz) was delivered via emergent cesarean delivery. The infant was limp and pale, and had apnea initially, with a heart rate of 40 to 60 beats per minute. The heart rate became undetectable 1 minute after birth. Cardiac compressions were initiated with positive pressure ventilation. The infant underwent mechanical intubation and received epinephrine without significant improvement in heart rate. At 7 minutes after birth, an umbilical artery catheter was placed and epinephrine was administered through the catheter, without improvement. Slow improvement was seen with fluid resuscitation. The heart rate was higher than 100 beats per minute by 11 minutes after birth, and spontaneous respirations occurred by 12 minutes after birth. The infant’s Apgar scores were 0 and 0 at 1 and 5 minutes, respectively. After resuscitation, the Apgar scores improved to 2 and 6 at 10 and 15 minutes, respectively. The umbilical cord blood gases revealed a severe respiratory acidosis (Table 2). The notable difference in pH between the arterial and venous blood gases demonstrates the acute nature of the insult.

Once stable, the infant was transferred to the NICU, and promptly received a transfusion of O-negative blood. The active therapeutic hypothermia protocol was initiated immediately on admission to NICU and was continued for 72 hours. Initial laboratory testing showed a hemoglobin of 3.0 g/dL (30 g/L). After transfusion of 2 units of packed red blood cells, the hemoglobin was 12 g/dL (120 g/L). The white blood cell count was 14,000/µL (34 × 10⁹/L), and platelets were 124 × 10⁹/µL (124 × 10⁹/L). Laboratory values subsequently remained stable throughout the infant’s hospital course. Treatment with ampicillin and cefotaxime was started for sepsis prophylaxis due to the severity of illness. The infant underwent extubation 1 day after birth, and subsequently was weaned to room air on day 2 after birth. A pediatric neurology consultation was obtained on day 2 after birth. Once the cooling protocol was completed, electroencephalography showed no seizure activity. Brain magnetic resonance imaging results were normal for the infant’s age. The infant was discharged from the hospital in stable condition 9 days after birth. He was subsequently seen in the outpatient pediatric neurology clinic at 1 month of age, and was observed to be doing well without clinical concerns. The mother had an unremarkable postoperative course and was discharged on postoperative day 4.

Gross examination of the placenta revealed a VCI with vessels that traversed the fetal membranes. A ruptured fetal vessel was noted (Fig 4, arrow). Given the clinical presentation, these findings were consistent with a ruptured vasa previa and fetal hemorrhage.

**DISCUSSION**

Vasa previa is a placental abnormality in which fetal blood vessels that are unprotected by the umbilical cord or placenta course through the fetal membranes over or near the cervix. (1)(2)(3) It is a rare condition, with an incidence of approximately 1 in 2,500 deliveries. (4) Vasa previa can lead to perinatal demise due to fetal exsanguination from the rupture of fetal vessels during labor or rupture of membranes, as well as fetal asphyxia from pressure on the unprotected fetal vessels. (5)(6) Two major risk factors for vasa previa are VCI and bilobed or succenturiate placenta. (2)(3) VCI is defined as umbilical vessels that insert into the choriamnionic membranes beyond the margin of the placenta. (6) Similar to vasa previa, the umbilical vessels in a VCI are vulnerable to compression because they are unprotected by Wharton jelly. (6)(7) In addition, a patient with a suspected second-trimester low-lying placenta or placenta previa remains at risk for vasa previa, regardless of whether or not the placenta remains low lying at the time of delivery. (5)(8)(9)

Prenatal diagnosis has been demonstrated to be an independent predictor of perinatal survival in a case of vasa previa. In a multicenter, retrospective study involving 155 pregnancies complicated by vasa previa, Oyelese and colleagues found a perinatal survival rate of 44% if the vasa previa was not diagnosed antenatally, compared with a perinatal survival of 97% with a prenatal diagnosis. (5) Multivariate regression analysis showed prenatal diagnosis of vasa previa and gestational age at the time of delivery to be the only factors independently associated with neonatal survival. Prenatal diagnosis of vasa previa and VCI relies on ultrasonography and is best detected by second-

**TABLE 2. Arterial and Venous Cord Blood Gas Analysis**

<table>
<thead>
<tr>
<th>ARTERIAL</th>
<th>VENOUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>6.89</td>
</tr>
<tr>
<td>PCO₂</td>
<td>122</td>
</tr>
<tr>
<td>PO₂</td>
<td>15</td>
</tr>
<tr>
<td>Base excess</td>
<td>–14.5</td>
</tr>
<tr>
<td>pH</td>
<td>7.27</td>
</tr>
<tr>
<td>PCO₂</td>
<td>47</td>
</tr>
<tr>
<td>PO₂</td>
<td>42</td>
</tr>
<tr>
<td>Base excess</td>
<td>–5.5</td>
</tr>
</tbody>
</table>
trimester transvaginal ultrasonography, in combination with pulsed-wave and color Doppler. (3)(4) Ultrasonography findings suggestive of a vasa previa include a linear, tubular, echolucent structure overlying the cervix or near the cervix in the lower uterine segment. (2)(4)(5) Similarly, a diagnosis of VCI can be made with color Doppler ultrasonography, by visualizing at least 1 cm of umbilical vessels coursing through the fetal membranes beyond the placental edge. (6)(7) Once a diagnosis of VCI is made, it is important to look for signs of vasa previa, because approximately 3% to 4% of women with a VCI may also have a vasa previa. (9) According to a systematic review, ultrasonography is associated with a 93% sensitivity and 99% specificity for vasa previa. (10)

In the present case, gross examination of the placenta showed a VCI, and a vasa previa was suggested by the acute onset of bleeding and fetal distress during digital examination. If a prenatal diagnosis of vasa previa had been established, a scheduled cesarean delivery at 34 to 35 weeks’ gestation would have been recommended to avoid a catastrophic fetal hemorrhage from the rupture of a fetal vessel. (5)(11)

References

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