Patient-Related Obstetrical Problems: The Common Calls
Abnormal Fetal Heart Rate Patterns

BACKGROUND AND DEFINITIONS

Intrapartum fetal heart rate monitoring is used to detect abnormal fetal heart rate patterns that may be associated with hypoxia, acidosis, and fetal asphyxia. Episodes of hypoxia and acidosis are commonly encountered in normal labor. These episodes are usually tolerated well by the fetus. Long-term neurological damage to the fetus is a concern only when these episodes of hypoxia and acidosis are extreme and persistent. Fetal heart rate patterns can be described as "reassuring" or "nonreassuring."

- Acidosis: Decreased pH in tissue
- Acidemia: Decreased pH in blood
- Hypoxia: Decreased oxygen level in tissue
- Hypoxemia: Decreased oxygen level in blood
- Fetal asphyxia: Hypoxia with metabolic acidosis
- Baseline fetal heart activity: Baseline characteristics of the fetal heart rate
- Periodic fetal heart rate activity: Characteristics of the fetal heart rate that are associated with uterine contractions or fetal movements.

Abnormal fetal heart rate patterns can consist of abnormalities of baseline fetal heart rate activity or abnormalities of periodic fetal heart rate activity, or both.

ABNORMALITIES IN BASELINE FETAL HEART RATE ACTIVITY

1. Abnormal rate. The normal baseline fetal heart rate in the third trimester is 120 to 160 beats/min. Occasional accelerations are associated with a normal fetal heart rate pattern (Fig. 4–1). Deceleration of the fetal heart rate is the initial response to hypoxia. If hypoxia is persistent, a baseline tachycardia often
develops. Other causes of tachycardia are maternal fever and chorioamnionitis.

- **Mild bradycardia**: Baseline rate of 100 to 119 beats/min
- **Moderate bradycardia**: Baseline rate of 80 to 99 beats/min for at least 3 minutes (Fig. 4–2)
- **Severe bradycardia**: Baseline rate of less than 80 beats/min for at least 3 minutes
- **Mild tachycardia**: Baseline rate of 161 to 180 beats/min
- **Severe tachycardia**: Baseline rate of greater than 180 beats/min.

2. **Abnormal variability.** Variability is the oscillatory appearance of the fetal heart rate tracing when recorded on graph

![Figure 4–1 Normal fetal heart rate with accelerations. (From Creasy RK, Resnik R: Maternal-fetal medicine: principles and practice, 5th ed. Philadelphia, Saunders, 2004, p 409.)](image1)

![Figure 4–2 Prolonged fetal bradycardia. (From Creasy RK, Resnik R: Maternal-fetal medicine: principles and practice, 5th ed. Philadelphia, Saunders, 2004, p 410.)](image2)
Regulated by the fetal autonomic nervous system, variability is an indicator of both activity of the fetal brain and normal cardiac responsiveness. Decreased variability can be a useful indicator of fetal hypoxia. In the situation of fetal compromise, decreased variability is often the first sign, preceding other baseline changes such as bradycardia. Normal variability is a reassuring sign of fetal well-being. However, decreased variability is not always a sign of fetal hypoxia because it has many causes (Table 4–1). Variability of the fetal heart rate consists of two components: short-term variability and long-term variability.

**Short-term variability:** The beat-to-beat change in fetal heart rate from one beat to the next. Short-term variability can be detected only by internal electronic fetal monitoring. Decreased or absent short-term variability can be an indication of fetal compromise, especially if it is persistent (Fig. 4–3).

**Long-term variability:** Oscillatory changes in fetal heart rate over 1 minute, resulting in a wavy baseline. The nor-

<table>
<thead>
<tr>
<th>TABLE 4–1 Causes of Decreased Variability</th>
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<tbody>
<tr>
<td>Fetal hypoxia</td>
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<tr>
<td>Fetal sleep cycle</td>
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<tr>
<td>Administration of drugs</td>
</tr>
<tr>
<td>Magnesium sulfate</td>
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<tr>
<td>Narcotic analgesics</td>
</tr>
<tr>
<td>Tranquilizers</td>
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<tr>
<td>Barbiturates</td>
</tr>
<tr>
<td>General anesthesia</td>
</tr>
<tr>
<td>Severe prematurity</td>
</tr>
<tr>
<td>Fetal heart block</td>
</tr>
<tr>
<td>Fetal anomalies</td>
</tr>
</tbody>
</table>

*Figure 4–3* Absence of variability. (From Creasy RK, Resnik R: Maternal-fetal medicine: principles and practice, 5th ed. Philadelphia, Saunders, 2004, p 411.)
mal frequency of the “waves” or cycle changes is 3 to 5/min. Decreased or absent long-term variability, if persistent, can indicate fetal compromise. No distinction should be made between short-term and long-term variability. Instead, they should be determined together as a unit because they are usually increased or decreased together. The characterization of variability utilizes the amplitude between the peak and trough of the fetal heart rate:

- Absent variability: amplitude not detectable
- Minimal variability: amplitude less than or equal to 5 beats/min
- Moderate variability: amplitude between 6 and 25 beats/min
- Marked variability: amplitude greater than 25 beats/min

3. Cardiac arrhythmia. Found in approximately 1% of patients monitored and can be detected only by electronic monitoring. Most arrhythmias are supraventricular and resolve spontaneously in the neonatal period. Ventricular arrhythmias are infrequent in fetuses. Most fetal cardiac arrhythmias are of little clinical significance if fetal cardiac failure, manifested by the presence of hydrops, is absent. However, the presence of cardiac arrhythmias can make interpretation of fetal heart rate patterns difficult.

### ABNORMALITIES IN PERIODIC FETAL HEART ACTIVITY

1. Early deceleration. A smooth, shallow, and symmetrical uniform deceleration in the fetal heart rate, beginning and ending with the beginning and ending of the uterine contraction, thereby resembling a mirror image of the contraction (Fig. 4–4). Rarely does the absolute heart rate fall to less than 100 beats/min or greater than 30 to 40 beats below the baseline fetal heart rate. Early decelerations are caused by compression of the fetal head during active labor.
and do not indicate fetal compromise. These decelerations are the most uncommon of all decelerations.

2. **Late deceleration.** A uniform, smooth deceleration in the fetal heart rate, usually beginning approximately 30 seconds after the beginning of the uterine contraction or later and returning to baseline after the end of the contraction (Figs. 4–5 and 4–6). The nadir of a late deceleration is reached after the peak of the uterine contraction. Late decelerations can be subtle, with a fall of only 10 to 30 beats below baseline. The heart rate rarely falls more than 40 beats below baseline. Late decelerations are not usually associated with an acceleration in the fetal heart rate immediately before or after the deceleration. Occasional late decelerations are of no clinical significance. However, repetitive late decelerations can be an indication of central nervous system hypoxia and even myocardial depression. Repetitive late decelerations can be caused by insufficiency in delivery or uptake of fetal oxygen. The depth of the late deceleration does not correlate with the degree of hypoxia.

3. **Variable deceleration.** A rapid fall in fetal heart rate with a steep downslope followed by a rapid return to baseline with a steep upslope. A typical variable deceleration resembles the shape of the letter U or the letter V but these decelerations can be variable in duration, depth, shape, size, and timing to uterine contractions (Fig. 4–7). A variable deceleration does not last more than 2 minutes in duration. A deceleration that lasts longer than 2 minutes should be called a prolonged deceleration. Each deceleration is usually preceded and followed by accelerations in the fetal heart rate.

![Figure 4-5 Late decelerations. (From Creasy RK, Resnik R: Maternal-fetal medicine: principles and practice, 5th ed. Philadelphia, Saunders, 2004, p 412.)](image-url)
Variable decelerations are the most common type of decelerations detected in labor and they are frequently seen in the second stage of labor. They are most commonly caused by umbilical cord compression but can be caused by any alteration in blood flow in the umbilical cord. These decelerations usually are coincident with uterine contractions or pushing efforts on the part of the woman. Common causes of variable decelerations are listed in Table 4–2. Variable decelerations are not usually associated with fetal hypoxia unless they are severe and repetitive. The severity of variable decelerations can be classified as mild, moderate, or severe by the following criteria:

- **Mild**: Decelerations less than 30 seconds in duration, regardless of depth
  Decelerations not lower than 80 beats/min, regardless of duration
- **Moderate**: Decelerations lower than 80 beats/min
- **Severe**: Decelerations lower than 70 beats/min for more than 60 seconds in duration

![Figure 4–6 Late decelerations with loss of variability. (From Newton M, Newton ER: Complications of gynecologic and obstetric management. Philadelphia, WB Saunders, 1988, p 273.)](image1)

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![Figure 4–7 Variable decelerations. (From Gabbe SG, Niebyl JR, Simpson JL: Obstetrics: normal and problem pregnancies, 4th ed. Philadelphia, Churchill Livingstone, 2002, p 416.)](image2)
Variable decelerations with slow return to baseline or blunted accelerations following the decelerations are of concern for fetal hypoxia, especially if they are associated with the absence of fetal heart rate variability.

4. Sinusoidal heart rate pattern. This is a distinct pattern consisting of regular, smooth oscillations resembling a sine wave (Fig. 4–8). This pattern is associated with fetal anemia as found in a Rh-alloimmunized fetus and also with fetal acidosis. The criteria for a sinusoidal heart rate pattern are:

- Regular sine wave pattern with a frequency of 3 to 5 cycles/min
- Amplitude of 5 to 30 beats/min
- Absence of short-term variability
- Absence of accelerations
- Duration of at least 10 minutes

Unfortunately, frequent low-amplitude accelerations of the fetal heart rate can mimic sinusoidal pattern even though these accelerations are reassuring of fetal well-being. These patterns are referred to as pseudosinusoidal patterns and do not require intervention.

5. Prolonged deceleration. A prolonged deceleration is defined as a deceleration lasting between 2 and 10 minutes in duration. A deceleration which lasts 2 minutes or less is usually a variable deceleration while one that lasts greater than 10 minutes is usually a change in the fetal heart rate baseline. Prolonged decelerations can be caused by any of the mechanisms that can cause early, late, and variable decelerations when these mechanisms

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**TABLE 4–2 Common Causes of Variable Decelerations**

<table>
<thead>
<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Nuchal cord with stretching of the cord with descent of the fetal head</td>
</tr>
<tr>
<td>Oligohydramnios</td>
</tr>
<tr>
<td>Short umbilical cord</td>
</tr>
<tr>
<td>Knot of the umbilical cord</td>
</tr>
<tr>
<td>Velamentous cord insertion</td>
</tr>
<tr>
<td>Umbilical cord prolapse</td>
</tr>
</tbody>
</table>

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are present for a longer duration and are more profound. Causes of prolonged decelerations include the following:
- Prolonged umbilical cord compression
- Prolonged uterine hyperstimulation
- Maternal hypotension
- Severe placental abruption
- Severe uteroplacental insufficiency

**CLINICAL PRESENTATION**

Abnormal fetal heart rate patterns are not necessarily associated with any specific maternal symptoms. Patients with intrauterine growth restriction and premature labor are more likely to have abnormal fetal heart rate patterns. Patients in whom fetal compromise is caused by placental abruption might have vaginal bleeding, uterine pain, or uterine tenderness. Likewise, patients with uterine hyperstimulation, excessive uterine contractions, or uterine rupture might experience increased pain. In many situations, conduction analgesia can mask this pain.

**PHONE CALL**

*Questions*

1. **What are the patient’s vital signs?**
   Maternal hypotension can result in abnormal fetal heart rate patterns.

2. **Does the patient appear to be in hypovolemic shock?**
   Hypovolemic shock caused by placental abruption or uterine rupture can cause abnormal fetal heart rate patterns.

3. **Does the patient appear to be in an excessive amount of pain?**
   Excessive pain can result from hyperstimulation of the uterus, caused by oxytocin administration, placental abruption, or uterine rupture. These are all potential causes of abnormal fetal heart rate patterns.

**Degree of Urgency**

A patient whose fetus has abnormal heart rate patterns should be seen immediately.

**ELEVATOR THOUGHTS**

What are the causes of abnormal fetal heart rate patterns?

1. **Maternal hypoperfusion**
   Hypotension from the use of conduction analgesia
   Decreased blood return due to uterine compression of the vena cava in the supine position
2. **Excessive uterine activity, usually from oxytocin administration**
   - Hyperstimulation with elevated resting uterine tone
   - Excessively frequent uterine contractions with inadequate rest periods (lasting <1 minute) between contractions
   - Prolonged uterine contractions (lasting >90 seconds)

3. **Uterine rupture**

4. **Decreased umbilical cord blood flow**
   - Umbilical cord compression
   - Umbilical cord knot
   - Nuchal cord
   - Short umbilical cord
   - Umbilical cord prolapse
   - Velamentous cord insertion
   - Oligohydramnios

5. **Placental dysfunction**
   - Maternal hypertension
   - Maternal diabetes mellitus
   - Maternal autoimmune disorders
   - Placental abruption.

**MAJOR THREAT TO FETAL LIFE**

Fetal asphyxia
   - Abnormal fetal heart rate patterns can indicate fetal asphyxia with possible neonatal morbidity and mortality.

**BEDSIDE**

**Quick Look Test**

**What types of abnormal fetal heart rate patterns are present?**
   - Certain fetal heart rate patterns, such as early decelerations and mild, variable decelerations can be detected in normal pregnancies and are not worrisome. Other patterns, such as persistent late decelerations and sinusoidal patterns may be suggestive of fetal compromise.

**How long have the abnormal fetal heart rate patterns been present?**
   - Occasional isolated abnormal heart rate patterns are usually of no significance, whereas persistent abnormal patterns can be more ominous.

**Is the patient in labor? If so, what is the quality and frequency of her contractions?**
   - Hyperstimulation of the uterus by oxytocin administration can cause abnormal heart rate patterns. Hyperstimulation is defined by more than 5 uterine contractions in 10 minutes, contractions
occurring with less than a 1-minute resting period between them, and contractions with durations of greater than 2 minutes.

*How much is the patient’s cervix dilated and how close is the patient to delivery?*

If the fetus has a nonreassuring heart rate pattern and is also close to delivery, facilitation of delivery by forceps or by vacuum should be considered.

**Vital Signs**

Hypotension caused by poor blood return in a supine position, placental abruption, and uterine rupture can result in abnormal heart rate patterns.

**Selective History and Chart Review**

1. Does the patient have any medical conditions such as hypertension, diabetes mellitus, or collagen-vascular disorders that cause chronic placental dysfunction?
2. Has there been evidence of intrauterine growth restriction? Intrauterine growth restriction can result from chronic placental dysfunction, which may in turn be associated with a higher incidence of abnormal fetal heart rate patterns.

**Selective Physical Examination**

<table>
<thead>
<tr>
<th>Abdominal</th>
<th>Tender in placental abruption</th>
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<tbody>
<tr>
<td>Pelvic</td>
<td>Normal unless there is excessive vaginal bleeding or meconium passage</td>
</tr>
<tr>
<td>External genitalia and vagina</td>
<td>Usually normal</td>
</tr>
<tr>
<td>Cervix</td>
<td>Normal unless there is uterine tenderness, irritability, or hypertonus caused by placental abruption, hyperstimulation, or uterine rupture</td>
</tr>
<tr>
<td>Uterus and adnexa</td>
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</tbody>
</table>

**Orders**

1. Start an intravenous (IV) fluid infusion if the patient does not already have one.
2. Turn the patient on her side to increase blood return through the inferior vena cava.
3. Administer supplemental oxygen to the mother, with a face mask and at an oxygen flow rate of 8 to 10 L/min.
4. Have available a fetal scalp electrode for internal fetal heart rate monitoring.

**MANAGEMENT OF NON-REASSURING FETAL HEART RATE PATTERN**

Abnormal fetal heart rate patterns can be caused by factors other than fetal compromise. While a normal fetal heart rate pattern is highly...
predictive of fetal well-being, an abnormal fetal heart rate pattern is 
not reliably predictive of fetal hypoxia and acidemia. If non-reassur-
ing fetal heart rate patterns are present, conservative steps can be used 
to correct the abnormal patterns. If these steps are not successful, 
diagnostic tests can then be used to determine whether or not fetal 
hypoxia and acidemia are present. Close monitoring, corrective steps, 
and/or diagnostic tests are warranted for the following non-reassur-
ing fetal heart rate patterns, especially if delivery is not imminent:

1. Recurrent prolonged decelerations
2. Recurrent late decelerations occurring with more than half 
of the contractions
3. Severe variable decelerations
4. Variable decelerations with a slow return or late component
5. Variable decelerations with decreased variability and tachy-
cardia

1. Place the patient in the lateral position  
   Placement of the patient in the lateral position displaces the 
   uterus from the midline and relieves compression of the 
   vena cava, resulting in increased blood return to the heart.

2. Perform pelvic examination  
   Pelvic examination can be performed to rule out prolapse of 
   the umbilical cord or rapid descent of the presenting 
   part of the fetus, both of which can be associated with 
   non-reasing fetal heart rate patterns. Pelvic examination will 
   also determine if the patient is completely dilated and if 
   assisted delivery with forceps or vacuum can be considered.

3. Administer supplemental oxygen  
   Administration of supplemental oxygen to the mother via a 
   tight-fitting face mask has been recommended when non-
reasing fetal heart rate patterns are noted. Unfortunately, 
   this long-standing practice results in only a small increase in 
   fetal Po2.

4. Decrease uterine contractions  
   a. Discontinue oxytocin  
      Every uterine contraction is associated with a transient 
      decrease in blood flow to the placenta, and subsequently to 
      the fetus. Therefore, if the patient is receiving oxytocin, dis-
continuation of the infusion will decrease both the intensity 
      and the frequency of uterine contractions and increase uter-
ine blood flow. Discontinuation of oxytocin is also the best 
      treatment for uterine hyperstimulation. After resolution of 
      the hyperstimulation and/or improvement in the fetal heart 
      rate pattern, oxytocin can be restarted at a lower infusion rate.
   b. Administer a tocolytic agent  
      Tocolytic drugs such as terbutaline sulfate or magnesium 
      sulfate can be administered to decrease both the intensity 
      and the frequency of uterine contractions, regardless of 
      whether the patient is receiving oxytocin.
(1) Terbutaline sulfate 0.25 mg subcutaneously or 0.125 to 0.25 mg IV
   If a decrease in uterine activity is not achieved in 15 to 30 minutes, a second dose can be administered.
(2) Magnesium sulfate 2.0 g IV over 10 minutes

5. Correct maternal hypotension
   Maternal hypotension can decrease uterine blood flow, which in turn can cause abnormal fetal heart rate patterns. Hypotension is often the result of conduction analgesia used during labor.
   a. Increase IV infusion rate or give IV bolus of 500 to 1000 mL lactated Ringer’s solution.
   b. Administer ephedrine sulfate 10 to 25 mg intramuscularly (IM) or IV
   c. Displace the uterus to the left to increase blood flow back to the heart

6. Amnioinfusion
   Amnioinfusion can decrease both the frequency and the severity of variable decelerations, especially in a patient with oligohydramnios. Furthermore, amnioinfusion may decrease the incidence of fetal meconium aspiration by dilution of meconium and also by decreasing severe variable decelerations which can cause fetal hypoxia and fetal gasping. Amnioinfusion is performed through an intrauterine catheter and can be performed as a bolus or a continuous infusion. Bolus infusion of 500 to 800 mL of room-temperature normal saline is administered at a rate of 10 to 15 mL/min. The bolus infusion of a similar or smaller amount can be repeated depending on the response of the fetal heart rate pattern, sonographic assessment of intra-amniotic fluid, and ongoing loss of fluid as labor progresses. Continuous infusion is initiated by infusing 10 mL/min of room-temperature normal saline for 1 hour followed by a maintenance infusion of 3 mL/min. Improvement in the fetal heart rate pattern usually occurs no sooner than 20 to 30 minutes after amnioinfusion is begun. Overdistention of the uterine cavity should be avoided because it can result in increased uterine tone and also deterioration of the fetal heart rate pattern.

If the above steps do not resolve the abnormal fetal heart rate patterns, then testing can be performed to attempt determine whether or not fetal hypoxia is present.

TESTING FOR FETAL HYPOXIA

1. Fetal pulse oximetry
   Fetal pulse oximetry received approval from the Food and Drug Administration for use in fetuses with non-reassuring
fetal heart rate patterns. This test utilizes a sensor plate which is placed transcervically against the fetal cheek to monitor oxygen saturation and heart rate. A fetal oxygen saturation of 30% or greater is reassuring. An oxygen saturation usually must remain below 30% for at least 10 minutes in order for fetal acidosis to develop. Fetal oximetry requires that the membranes are ruptured and that the cervix is dilated to at least 2 cm.

2. Fetal scalp stimulation tests

Fetal scalp stimulation can be performed by three methods: (1) fetal scalp puncture, (2) Allis clamp pinching of the fetal scalp, and (3) digital scalp stimulation. Of the three methods, digital scalp stimulation is most often utilized because it does not require any instruments. The cervix must be dilated to at least 2 cm and the test can be performed with intact membranes. Digital scalp stimulation is performed by gentle digital stroking of the fetal scalp for 15 seconds. For any of the three methods of scalp stimulation, a reassuring response is defined as an acceleration in the fetal heart rate of at least 15 beats per minute, lasting at least 15 seconds. However, the absence of an acceleration is not always associated with fetal acidosis.

3. Vibroacoustic stimulation (VAS)

Vibroacoustic stimulation is performed by applying the stimulator continuously for 3 to 5 seconds to the maternal abdomen in the location of the fetal head. A reassuring response is an acceleration in the fetal heart rate of at least 15 bpm, lasting at least 15 seconds. Advantages of this test include its use with an undilated cervix and intact membranes.

4. Fetal scalp blood sampling

Fetal scalp blood sampling is performed by placing an endoscope with a light source into the vagina and against the fetal scalp. The scalp is then coated with a silicone gel which causes the fetal blood to form into globules and a punch incision is made into the fetal scalp to a depth of approximately 2 mm. Fetal scalp blood is then collected with a heparinized glass capillary tube and the pH is measured to identify the fetus with acidosis. A fetal capillary blood pH that is greater than 7.25 is considered reassuring and labor can be allowed to continue with electronic fetal monitoring (Table 4–3). If the pH is between 7.20 and 7.25, fetal scalp blood sampling is usually repeated within approximately 30 minutes, depending on the subsequent fetal heart rate tracing. If the pH is less than 7.20, delivery or immediate repeat fetal scalp blood sampling is recommended. The cervix must be dilated at least 2 to 3 cm, and the membranes must be ruptured, to perform fetal scalp blood sampling. Fetal scalp blood sampling is utilized extremely infrequently in current obstetrical practice. It is
technically difficult, uncomfortable for patients, and requires repeat testing in many cases. Furthermore, when compared with other tests for fetal hypoxia discussed above, fetal scalp blood sampling takes longer both to perform and to obtain results.

Depending on the results of these tests and the severity of the abnormal fetal heart rate pattern, several decisions will need to be made concerning delivery or continued labor. A clinician will need to utilize judgment and consider a multitude of factors to determine whether the patient should be delivered and if so, how soon and by what route, vaginal delivery, or operative vaginal delivery, or Cesarean delivery. Factors that might need to be considered include the availability of an anesthesiologist, the skill set of the clinician, the stage of labor that the patient is in, how long before spontaneous delivery can be anticipated, the safety of an operative vaginal delivery, and the risk of Cesarean delivery. Repeat testing and continued labor might be appropriate in a patient with a non-reassuring fetal heart rate pattern who is remote from delivery. In contrast, operative vaginal delivery might be appropriate in a patient with the same or even less concerning fetal heart rate pattern who is close to delivery.

### TABLE 4–3 Fetal Scalp Blood Values in Labor*

<table>
<thead>
<tr>
<th></th>
<th>Early First Stage</th>
<th>Late First Stage</th>
<th>Second Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.33 ± 0.03</td>
<td>7.32 ± 0.02</td>
<td>7.29 ± 0.04</td>
</tr>
<tr>
<td>PCO₂ (mm Hg)</td>
<td>44 ± 4.05</td>
<td>42 ± 5.1</td>
<td>46.3 ± 4.2</td>
</tr>
<tr>
<td>PO₂ (mm Hg)</td>
<td>21.8 ± 2.6</td>
<td>21.3 ± 2.1</td>
<td>16.5 ± 1.4</td>
</tr>
<tr>
<td>Bicarbonate (mmol/L)</td>
<td>20.1 ± 1.2</td>
<td>19.1 ± 2.1</td>
<td>17 ± 2</td>
</tr>
<tr>
<td>Base excess (mmol/L)</td>
<td>3.9 ± 1.9</td>
<td>4.1 ± 2.5</td>
<td>6.4 ± 1.8</td>
</tr>
</tbody>
</table>

*Mean ± standard deviation.