The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring

Update on Definitions, Interpretation, and Research Guidelines

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In April 2008, the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, the American College of Obstetricians and Gynecologists, and the Society for Maternal-Fetal Medicine partnered to sponsor a 2-day workshop to revisit nomenclature, interpretation, and research recommendations for intrapartum electronic fetal heart rate monitoring. Participants included obstetric experts and representatives from relevant stakeholder groups and organizations. This article provides a summary of the discussions at the workshop. This includes a discussion of terminology and nomenclature for the description of fetal heart tracings and uterine contractions for use in clinical practice and research. A three-tier system for fetal heart rate tracing interpretation is also described. Lastly, prioritized topics for future research are provided.

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See related editorial on page 506.

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For a list of workshop participants, see the Appendix online at www.greenjournal.org/cgi/content/full/112/3/661/DC1.

The workshop was jointly sponsored by the American College of Obstetricians and Gynecologists, the Eunice Kennedy Shriver National Institute of Child Health and Human Development, and the Society for Maternal-Fetal Medicine.

The recommendations from the National Institute of Child Health and Human Development 2008 Workshop are being published simultaneously by Obstetrics & Gynecology *and the* Journal of Obstetric, Gynecologic, & Neonatal Nursing.

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The *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD) convened a series of workshops in the mid- 1990s to develop standardized and unambiguous definitions for fetal heart rate (FHR) tracings, culminating in a publication of recommendations for defining fetal heart rate characteristics.[1] The goal of these definitions was to allow the predictive value of monitoring to be assessed more meaningfully and to allow evidence-based clinical management of intrapartum fetal compromise.

The definitions agreed upon in that workshop were endorsed for clinical use in the most recent American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin in 2005 and also endorsed by the Association of Women's Health, Obstetric and Neonatal Nurses.[2] Subsequently, the Royal College of Obstetricians and Gynaecologists (RCOG, 2001) and the Society of Obstetricians and Gynaecologists of Canada (SOGC, 2007) convened expert groups to assess the evidence-based use of electronic fetal monitoring (EFM). These groups produced consensus documents with more specific recommendations for FHR pattern classification and intrapartum management actions.[3,4] In addition, new interpretations and definitions have been proposed, including terminology such as "tachysystole" and "hyperstimulation" and new interpretative systems using three and five tiers.[3–5] The SOGC Consensus Guidelines for Fetal Health Surveillance presents a three-tier system (normal, atypical, abnormal), as does RCOG.[3,4] Parer and Ikeda[5] recently suggested a five-tier management grading system. Recently, the NICHD, ACOG, and the Society for Maternal-Fetal

Medicine jointly sponsored a workshop focused on EFM. The goals of this workshop were 1) to review and update the definitions for FHR pattern categorization from the prior workshop; 2) to assess existing classification systems for interpreting specific FHR patterns and to make recommendations about a system for use in the United States; and 3) to make recommendations for research priorities for EFM. Thus, while goals 1 and 3 are similar to the prior workshop, a new emphasis on interpretative systems (goal 2) was part of the recent workshop.

As was true in the prior publication,[1] before presenting actual definitions and interpretation, it is necessary to state a number of assumptions and factors common to FHR interpretation in the United States. These were defined in the initial publication[1] and were affirmed and/or updated by the panel:

A. The definitions are primarily developed for visual interpretation of FHR patterns. However, it is recognized that computerized interpretation is being developed and the definitions must also be adaptable to such applications.
B. The definitions apply to the interpretations of patterns produced from either a direct fetal electrode detecting the fetal electrocardiogram or an external Doppler device detecting the fetal heart rate events with use of the autocorrelation technique.
C. The record of both the FHR and uterine activity should be of adequate quality for visual interpretation.
D. The prime emphasis in this report is on intrapartum patterns. The definitions may also be applicable to antepartum observations.
E. The characteristics to be defined are those commonly used in clinical practice and research communications.
F. The features of FHR patterns are categorized as either baseline, periodic, or episodic. Periodic patterns are those associated with uterine contractions, and episodic patterns are those not associated with uterine contractions.
G. The periodic patterns are distinguished on the basis of waveform, currently accepted as either "abrupt" or "gradual" onset.
H. Accelerations and decelerations are generally determined in reference to the adjacent baseline FHR.
I. No distinction is made between short-term variability (or beat-to-beat variability or R–R wave period differences in the electrocardiogram) and long-term variability, because in actual practice they are visually determined as a unit. Hence, the definition of variability is based visually on the amplitude of the complexes, with exclusion of the sinusoidal pattern.
J. There is good evidence that a number of characteristics of FHR patterns are dependent upon fetal gestational age and physiologic status as well as maternal physiologic status. Thus, FHR tracings should be evaluated in the context of many clinical conditions including gestational age, prior results of fetal assessment, medications, maternal medical conditions, and fetal conditions (eg, growth restriction, known congenital anomalies, fetal anemia, arrhythmia, etc).
K. The individual components of defined FHR patterns do not occur independently and generally evolve over time.
L. A full description of an EFM tracing requires a qualitative and quantitative description of:
   1. Uterine contractions.
   2. Baseline fetal heart rate.
   3. Baseline FHR variability.
   4. Presence of accelerations.
   5. Periodic or episodic decelerations.
   6. Changes or trends of FHR patterns over time.

Uterine contractions are quantified as the number of contractions present in a 10-minute window, averaged over 30 minutes. Contraction frequency alone is a partial assessment of uterine activity. Other factors such as duration, intensity, and relaxation time between contractions are equally important in clinical practice.

The following represents terminology to describe uterine activity:

A. *Normal*: ≤5 contractions in 10 minutes, averaged over a 30-minute window.
B. *Tachysystole*: >5 contractions in 10 minutes, averaged over a 30-minute window.
C. *Characteristics of uterine contractions:*
   - Tachysystole should always be qualified as to the presence or absence of associated FHR decelerations.
   - The term tachysystole applies to both spontaneous or stimulated labor. The clinical response to tachysystole may differ depending on whether contractions are spontaneous or stimulated.
   - The terms hyperstimulation and hypercontractility are not defined and should be abandoned.

Fetal heart rate patterns are defined by the characteristics of baseline, variability, accelerations, and decelerations.

The *baseline* FHR is determined by approximating the mean FHR

rounded to increments of 5 beats per minute (bpm) during a 10-minute window, excluding accelerations and decelerations and periods of marked FHR variability (>25 bpm). There must be at least 2 minutes of identifiable baseline segments (not necessarily contiguous) in any 10-minute window, or the baseline for that period is indeterminate. In such cases, it may be necessary to refer to the previous 10-minute window for determination of the baseline. Abnormal baseline is termed *bradycardia* when the baseline FHR is <110 bpm; it is termed *tachycardia* when the baseline FHR is >160 bpm.

Baseline FHR variability is determined in a 10-minute window, excluding accelerations and decelerations. Baseline FHR variability is defined as fluctuations in the baseline FHR that are irregular in amplitude and frequency. The fluctuations are visually quantitated as the amplitude of the peak-to-trough in bpm.

Variability is classified as follows: *Absent* FHR variability: amplitude range undetectable. *Minimal* FHR variability: amplitude range>undetectable and ≤5 bpm. *Moderate* FHR variability: amplitude range 6 bpm to 25 bpm. *Marked* FHR variability: amplitude range >25 bpm.

An acceleration is a visually apparent *abrupt* increase in FHR. An *abrupt* increase is defined as an increase from the onset of acceleration to the peak in <30 seconds. To be called an acceleration, the peak must be ≥15 bpm, and the acceleration must last ≥15 seconds from the onset to return. A *prolonged* acceleration is ≥2 minutes but <10 minutes in duration. Finally, an acceleration lasting ≥10 minutes is defined as a *baseline change*. Before 32 weeks of gestation, accelerations are defined as having a peak ≥10 bpm and a duration of ≥10 seconds.

Decelerations are classified as late, early, or variable based on specific characteristics (see the Box, "Characteristics of Decelerations"). Variable decelerations may be accompanied by other characteristics, the clinical significance of which requires further research investigation. Some examples include a slow return of the FHR after the end of the contraction, biphasic decelerations, tachycardia after variable deceleration(s), accelerations preceding and/or following, sometimes called "shoulders" or "overshoots," and fluctuations in the FHR in the trough of the deceleration.

A *prolonged* deceleration is present when there is a visually apparent decrease in FHR from the baseline that is ≥15 bpm, lasting ≥2 minutes, but <10 minutes. A deceleration that lasts ≥10 minutes is a *baseline change*.

A *sinusoidal fetal heart rate pattern* is a specific fetal heart rate pattern that is defined as having a visually apparent, smooth, sine wave–like undulating pattern in FHR baseline with a cycle frequency of 3–5/min that persists for ≥20 minutes.

Quantitation of Decelerations

The magnitude of a deceleration is quantitated by the depth of the nadir in beats per minute (excluding transient spikes or electronic artifact). The duration is quantitated in minutes and seconds from the beginning to the end of the deceleration. Accelerations are quantitated similarly.

Some authors have suggested grading of decelerations based on the depth of the deceleration or absolute nadir in beats per minute and duration.[4–7] These grading systems require further investigation as to their predictive value.

Decelerations are defined as *recurrent* if they occur with ≥50% of uterine contractions in any 20-minute window. Decelerations occurring with <50% of uterine contractions in any 20-minute segment are defined as *intermittent*.

General Considerations for the Interpretation of Fetal Heart Rate Patterns

A variety of systems for EFM interpretation have been developed and propagated in the United States and worldwide.[3–5] Any interpretation system must be based, to the greatest extent possible, on existing evidence (recognizing that in some areas evidence is lacking). In addition, any system should be simple and applicable to clinical practice.

Given that the fetal heart rate response is a dynamic process, and one that evolves over time, the categories of FHR patterns are dynamic and transient, requiring frequent reassessment. It is common for FHR tracings to move from one category to another over time.

The FHR tracing should be interpreted in the context of the overall clinical circumstances, and categorization of a FHR tracing is limited to the time period being assessed. The presence of FHR accelerations (either spontaneous or stimulated) reliably predicts the absence of fetal metabolic acidemia. The absence of accelerations does not, however, reliably predict fetal acidemia. Fetal heart rate accelerations can be stimulated with a variety of methods (vibroacoustic, transabdominal halogen light, and direct fetal scalp stimulation).

Moderate FHR variability reliably predicts the absence of fetal metabolic acidemia at the time it is observed. Minimal or absent FHR variability alone does not reliably predict the presence of fetal hypoxemia or metabolic acidemia. The significance of marked FHR (previously described as saltatory) variability is unclear.

Characteristics of Decelerations

Late Deceleration

- Visually apparent usually symmetrical *gradual* decrease and return of the fetal heart rate (FHR) associated with a uterine contraction.
- A *gradual* FHR decrease is defined as from the onset to the FHR nadir of ≥30 seconds.
- 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 deceleration 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.

Early Deceleration

- Visually apparent, usually symmetrical, *gradual* decrease and return of the FHR associated with a uterine contraction.
- A *gradual* FHR decrease is defined as one from the onset to the FHR nadir of ≥30 seconds.
- The decrease in FHR is calculated from the onset to the nadir of the deceleration.
- The nadir of the deceleration occurs at the same time as the peak of the contraction.
- In most cases the onset, nadir, and recovery of the deceleration are coincident with the beginning, peak, and ending of the contraction, respectively.

Variable Deceleration

- Visually apparent *abrupt* decrease in FHR.
- An *abrupt* FHR decrease is defined as from the onset of the deceleration to the beginning of the FHR nadir of <30 seconds. The decrease in FHR is calculated from the onset to the nadir of the deceleration.
- The decrease in FHR is ≥15 beats per minute, lasting ≥15 seconds, and <2 minutes in duration.
- When variable decelerations are associated with uterine contractions, their onset, depth, and duration commonly vary with successive uterine contractions.

Interpretation of Fetal Heart Rate Patterns

Based on careful review of the available options, a three-tier system for the categorization of FHR patterns is recommended (see the Box, "Three-Tier Fetal Heart Rate Interpretation System"). Although the development of management algorithms is a function of professional specialty entities, some general management principles were agreed upon for these categories. Fetal heart rate tracing patterns provide information on the current acid–base status of the fetus and cannot predict the development of cerebral palsy. Categorization of the FHR tracing evaluates the fetus at that point in time; tracing patterns can and will change. A FHR tracing may move back and forth between categories depending on the clinical situation and management strategies employed.

Category I FHR tracings are *normal*. Category I FHR tracings are strongly predictive of *normal* fetal acid–base status at the time of observation. Category I FHR tracings may be followed in a routine manner, and no specific action is required.

Category II FHR tracings are *indeterminate*. Category II FHR tracings are not predictive of *abnormal* fetal acid–base status, yet we do not have adequate evidence at present to classify these as Category I or Category III. Category II FHR tracings require evaluation and continued surveillance and reevaluation, taking into account the entire associated clinical circumstances.

Category III FHR tracings are *abnormal*. Category III tracings are predictive of *abnormal* fetal acid–base status at the time of observation. Category III FHR tracings require prompt evaluation. Depending on the clinical situation, efforts to expeditiously resolve the *abnormal* FHR pattern may include, but are not limited to, provision of maternal oxygen, change in maternal position, discontinuation of labor stimulation, and treatment of maternal hypotension.

Research Recommendations

Since the last workshop, there has not been a wealth of research on EFM. With the high penetrance of

Three-Tier Fetal Heart Rate Interpretation System

Category I

Category I fetal heart rate (FHR) tracings include all of the following:

- Baseline rate: 110–160 beats per minute (bpm)
- Baseline FHR variability: moderate
- Late or variable decelerations: absent
- Early decelerations: present or absent
- Accelerations: present or absent

Category II

Category II FHR tracings include all FHR tracings not categorized as Category I or Category III. Category II tracings may represent an appreciable fraction of those encountered in clinical care. Examples of Category II FHR tracings include any of the following:

Baseline rate

- Bradycardia not accompanied by absent baseline variability
- Tachycardia

Baseline FHR variability

- Minimal baseline variability
- Absent baseline variability not accompanied by recurrent decelerations
- Marked baseline variability

Accelerations

- Absence of induced accelerations after fetal stimulation

Periodic or episodic decelerations

- Recurrent variable decelerations accompanied by minimal or moderate baseline variability
- Prolonged deceleration ≥2 minutes but <10 minutes
- Recurrent late decelerations with moderate baseline variability
- Variable decelerations with other characteristics, such as slow return to baseline, "overshoots," or "shoulders"

Category III

Category III FHR tracings include either:

- Absent baseline FHR variability and any of the following:
  - Recurrent late decelerations
  - Recurrent variable decelerations
  - Bradycardia
- Sinusoidal pattern

this technology into obstetric practice, well-designed studies are needed to fill gaps in knowledge. Areas of highest priority for research include observational studies focused on indeterminate FHR patterns, including descriptive epidemiology, frequency of specific patterns, change over time, the relationship to clinically relevant outcomes, and the effect of duration of patterns (eg, recurrent late decelerations with minimal variability) on clinical outcomes. Other needed studies include work that evaluates contraction frequency, strength, and duration on FHR and clinical outcomes. Research also needs to be focused on the effectiveness of educational programs on EFM that include all relevant stakeholders. Although computerized interpretation systems have not developed as rapidly as anticipated, studies are needed on the effectiveness of computerized compared with provider interpretation, including the analy-

sis of existing data sets. Other areas for work include the development of new comprehensive data sets integrating outcomes with EFM in digitally addressable format and research on effectiveness of techniques supplementary to EFM, such as ST segment analysis.

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Intrapartum Fetal Heart Rate Monitoring: Nomenclature, Interpretation, and General Management Principles

In the most recent year for which data are available, approximately 3.4 million fetuses (85% of approximately 4 million live births) in the United States were assessed with electronic fetal monitoring (EFM), making it the most common obstetric procedure (1). Despite its widespread use, there is controversy about the efficacy of EFM, interobserver and intraobserver variability, nomenclature, systems for interpretation, and management algorithms. Moreover, there is evidence that the use of EFM increases the rate of cesarean deliveries and operative vaginal deliveries. The purpose of this document is to review nomenclature for fetal heart rate assessment, review the data on the efficacy of EFM, delineate the strengths and shortcomings of EFM, and describe a system for EFM classification.

This Practice Bulletin was developed by the ACOG Committee on Practice Bulletins with the assistance of George A. Macones, MD. The information is designed to aid practitioners in making decisions about appropriate obstetric and gynecologic care. These guidelines should not be construed as dictating an exclusive course of treatment or procedure. Variations in practice may be warranted based on the needs of the individual patient, resources, and limitations unique to the institution or type of practice.

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Background

A complex interplay of antepartum complications, suboptimal uterine perfusion, placental dysfunction, and intrapartum events can result in adverse neonatal outcome. Known obstetric conditions, such as hypertensive disease, fetal growth restriction, and preterm birth, predispose fetuses to poor outcomes, but they account for a small proportion of asphyxial injury. In a study of term pregnancies with fetal asphyxia, 63% had no known risk factors (2).

The fetal brain modulates the fetal heart rate through an interplay of sympathetic and parasympathetic forces. Thus, fetal heart rate (FHR) monitoring can be used to determine if a fetus is well oxygenated. It was used among 45% of laboring women in 1980, 62% in 1988, 74% in 1992, and 85% in 2002 (1).

Despite the frequency of its use, limitations of EFM include poor interobserver and intraobserver reliability, uncertain efficacy, and a high false-positive rate.

Fetal heart rate monitoring may be performed externally or internally. Most external monitors use a Doppler device with computerized logic to interpret and count the Doppler signals. Internal FHR monitoring is accomplished with a fetal electrode, which is a spiral wire placed directly on the fetal scalp or other presenting part.

Guidelines for Nomenclature and Interpretation of Electronic Fetal Heart Rate Monitoring

In 2008, the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development partnered with the American College of Obstetricians and Gynecologists and the Society for Maternal–Fetal Medicine to sponsor a workshop focused on electronic FHR monitoring (3). This 2008 workshop gathered a diverse group of investigators with expertise and interest in the field to accomplish three goals: 1) to review and update the definitions for FHR pattern categorization from the prior workshop; 2) to assess existing classification systems for interpreting specific FHR patterns and make recommendations about a system for use in the United States; and 3) to make recommendations for research priorities for EFM. A complete clinical understanding of EFM necessitates discussion of uterine contractions, baseline FHR rate and variability, presence of accelerations, periodic or episodic decelerations, and the changes in these characteristics over time. A number of assumptions and factors common to FHR interpretation in the United States are central to the proposed system of nomenclature and interpretation (3). Two such assumptions are of particular importance. First, the definitions are primarily developed for visual interpretation of FHR patterns, but should be adaptable to computerized systems of interpretation. Second, the definitions should be applied to intrapartum patterns, but also are applicable to antepartum observations.

Uterine contractions are quantified as the number of contractions present in a 10-minute window, averaged over a 30-minute period. Contraction frequency alone is a partial assessment of uterine activity. Other factors such as duration, intensity, and relaxation time between contractions are equally important in clinical practice.

Listed as follows is terminology used to describe uterine activity:

Normal: five contractions or less in 10 minutes, averaged over a 30-minute window

Tachysystole: more than five contractions in 10 minutes, averaged over a 30-minute window

Characteristics of uterine contractions

- The terms hyperstimulation and hypercontractility are not defined and should be abandoned.
- Tachysystole should always be qualified as to the presence or absence of associated FHR decelerations.
- The term tachysystole applies to both spontaneous and stimulated labor. The clinical response to tachysystole may differ depending on whether contractions are spontaneous or stimulated.

Table 1 provides EFM definitions and descriptions based on the 2008 National Institute of Child Health and Human Development Working Group findings. Decelerations are defined as recurrent if they occur with at least one half of the contractions.

Classification of Fetal Heart Rate Tracings

A variety of systems for EFM interpretation have been used in the United States and worldwide (4–6). Based on careful review of the available options, a three-tiered system for the categorization of FHR patterns is recommended (see box). It is important to recognize that FHR tracing patterns provide information only on the current acid–base status of the fetus. Categorization of the FHR tracing evaluates the fetus at that point in time; tracing patterns can and will change. An FHR tracing may move back and forth between the categories depending on the clinical situation and management strategies used.

Category I FHR tracings are normal. Category I FHR tracings are strongly predictive of normal fetal acid–base status at the time of observation. Category I FHR tracings may be monitored in a routine manner, and no specific action is required.

Category II FHR tracings are indeterminate. Category II FHR tracings are not predictive of abnormal fetal acid–base status, yet presently there is not adequate evidence to classify these as Category I or Category III. Category II FHR tracings require evaluation and continued surveillance and reevaluation, taking into account the entire associated clinical circumstances. In some circumstances, either ancillary tests to ensure fetal well-being or intrauterine resuscitative measures may be used with Category II tracings.

Category III FHR tracings are abnormal. Category III tracings are associated with abnormal fetal acid–base status at the time of observation. Category III FHR tracings require prompt evaluation. Depending on the clinical situation, efforts to expeditiously resolve the

Table 1. Electronic Fetal Monitoring Definitions

Pattern	Definition
Baseline	• The mean FHR rounded to increments of 5 beats per minute during a 10-minute segment, excluding: —Periodic or episodic changes —Periods of marked FHR variability —Segments of baseline that differ by more than 25 beats per minute • The baseline must be for a minimum of 2 minutes in any 10-minute segment, or the baseline for that time period is indeterminate. In this case, one may refer to the prior 10-minute window for determination of baseline. • Normal FHR baseline: 110–160 beats per minute • Tachycardia: FHR baseline is greater than 160 beats per minute • Bradycardia: FHR baseline is less than 110 beats per minute
Baseline variability	• Fluctuations in the baseline FHR that are irregular in amplitude and frequency • Variability is visually quantitated as the amplitude of peak-to-trough in beats per minute. —Absent—amplitude range undetectable —Minimal—amplitude range detectable but 5 beats per minute or fewer —Moderate (normal)—amplitude range 6–25 beats per minute —Marked—amplitude range greater than 25 beats per minute
Acceleration	• A visually apparent abrupt increase (onset to peak in less than 30 seconds) in the FHR • At 32 weeks of gestation and beyond, an acceleration has a peak of 15 beats per minute or more above baseline, with a duration of 15 seconds or more but less than 2 minutes from onset to return. • Before 32 weeks of gestation, an acceleration has a peak of 10 beats per minute or more above baseline, with a duration of 10 seconds or more but less than 2 minutes from onset to return. • Prolonged acceleration lasts 2 minutes or more but less than 10 minutes in duration. • If an acceleration lasts 10 minutes or longer, it is a baseline change.
Early deceleration	• 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 seconds or more. • The decrease in FHR is calculated from the onset to the nadir of the deceleration. • The nadir of the deceleration occurs at the same time as the peak of the contraction. • In most cases the onset, nadir, and recovery of the deceleration are coincident with the beginning, peak, and ending of the contraction, respectively.
Late deceleration	• 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 seconds or more. • 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 deceleration 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.
Variable deceleration	• Visually apparent abrupt decrease in FHR • An abrupt FHR decrease is defined as from the onset of the deceleration to the beginning of the FHR nadir of less than 30 seconds. • The decrease in FHR is calculated from the onset to the nadir of the deceleration. • The decrease in FHR is 15 beats per minute or greater, lasting 15 seconds or greater, and less than 2 minutes in duration. • When variable decelerations are associated with uterine contractions, their onset, depth, and duration commonly vary with successive uterine contractions.
Prolonged deceleration	• Visually apparent decrease in the FHR below the baseline • Decrease in FHR from the baseline that is 15 beats per minute or more, lasting 2 minutes or more but less than 10 minutes in duration. • If a deceleration lasts 10 minutes or longer, it is a baseline change.
Sinusoidal pattern	• Visually apparent, smooth, sine wave-like undulating pattern in FHR baseline with a cycle frequency of 3–5 per minute which persists for 20 minutes or more.

Abbreviation: FHR, fetal heart rate.

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Three-Tiered Fetal Heart Rate Interpretation System

Category I

- Category I FHR tracings include all of the following:
- Baseline rate: 110–160 beats per minute
- Baseline FHR variability: moderate
- Late or variable decelerations: absent
- Early decelerations: present or absent
- Accelerations: present or absent

Category II

Category II FHR tracings includes all FHR tracings not categorized as Category I or Category III. Category II tracings may represent an appreciable fraction of those encountered in clinical care. Examples of Category II FHR tracings include any of the following:

Baseline rate

- Bradycardia not accompanied by absent baseline variability
- Tachycardia

Baseline FHR variability

- Minimal baseline variability
- Absent baseline variability with no recurrent decelerations
- Marked baseline variability

Accelerations

- Absence of induced accelerations after fetal stimulation

Periodic or episodic decelerations

- Recurrent variable decelerations accompanied by minimal or moderate baseline variability
- Prolonged deceleration more than 2 minutes but less than10 minutes
- Recurrent late decelerations with moderate baseline variability
- Variable decelerations with other characteristics such as slow return to baseline, overshoots, or "shoulders"

Category III

Category III FHR tracings include either

- Absent baseline FHR variability and any of the following:
  - –Recurrent late decelerations
  - –Recurrent variable decelerations
  - –Bradycardia
- Sinusoidal pattern

Abbreviation: FHR, fetal heart rate

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abnormal FHR pattern may include but are not limited to provision of maternal oxygen, change in maternal position, discontinuation of labor stimulation, treatment of maternal hypotension, and treatment of tachysystole with FHR changes. If a Category III tracing does not resolve with these measures, delivery should be undertaken.

Guidelines for Review of Electronic Fetal Heart Rate Monitoring

When EFM is used during labor, the nurses or physicians should review it frequently. In a patient without complications, the FHR tracing should be reviewed approximately every 30 minutes in the first stage of labor and every 15 minutes during the second stage. The corresponding frequency for patients with complications (eg, fetal growth restriction, preeclampsia) is approximately every 15 minutes in the first stage of labor and every 5 minutes during the second stage. Health care providers should periodically document that they have reviewed the tracing. The FHR tracing, as part of the medical record, should be labeled and available for review if the need arises. Computer storage of the FHR tracing that does not permit overwriting or revisions is reasonable, as is microfilm recording.

Clinical Considerations and Recommendations

▶ *How efficacious is intrapartum electronic fetal heart rate monitoring?*

The efficacy of EFM during labor is judged by its ability to decrease complications, such as neonatal seizures, cerebral palsy, or intrapartum fetal death, while minimizing the need for unnecessary obstetric interventions, such as operative vaginal delivery or cesarean delivery. There are no randomized clinical trials to compare the benefits of EFM with any form of monitoring during labor (7). Thus, the benefits of EFM are gauged from reports comparing it with intermittent auscultation.

A meta-analysis synthesizing the results of the randomized clinical trials comparing the modalities had the following conclusions (8):

- The use of EFM compared with intermittent auscultation increased the overall cesarean delivery rate (relative risk [RR], 1.66; 95% confidence interval [CI], 1.30–2.13) and the cesarean delivery rate for abnormal FHR or acidosis or both (RR, 2.37; 95% CI, 1.88–3.00).

- The use of EFM increased the risk of both vacuum and forceps operative vaginal delivery (RR, 1.16; 95% CI, 1.01–1.32).
- The use of EFM did not reduce perinatal mortality (RR, 0.85; 95% CI, 0.59–1.23).
- The use of EFM reduced the risk of neonatal seizures (RR, 0.50; 95% CI, 0.31–0.80).
- The use of EFM did not reduce the risk of cerebral palsy (RR, 1.74; 95% CI, 0.97–3.11).

There is an unrealistic expectation that a nonreassuring FHR tracing is predictive of cerebral palsy. The positive predictive value of a nonreassuring pattern to predict cerebral palsy among singleton newborns with birth weights of 2,500 g or more is 0.14%, meaning that out of 1,000 fetuses with a nonreassuring FHR pattern, only one or two will develop cerebral palsy (9). The false-positive rate of EFM for predicting cerebral palsy is extremely high, at greater than 99%.

Available data, although limited in quantity, suggest that the use of EFM does not result in a reduction in cerebral palsy (8). This is consistent with data that suggest that the occurrence of cerebral palsy has been stable over time, despite the widespread introduction of EFM (10). The principal explanation for why the prevalence of cerebral palsy has not diminished despite the use of EFM is that 70% of cases occur before the onset of labor; only 4% of cases of encephalopathy can be attributed solely to intrapartum events (11, 12).

Given that the available data do not show a clear benefit for the use of EFM over intermittent auscultation, either option is acceptable in a patient without complications. Logistically, it may not be feasible to adhere to guidelines for how frequently the heart rate should be auscultated. One prospective study noted that the protocol for intermittent auscultation was successfully completed in only 3% of the cases (13). The most common reasons for unsuccessful intermittent auscultation included the frequency of recording and the requirements for recording.

Intermittent auscultation may not be appropriate for all pregnancies. Most of the clinical trials that compare EFM with intermittent auscultation have excluded participants at high risk of adverse outcomes, and the relative safety of intermittent auscultation in such cases is uncertain. The labor of women with high-risk conditions (eg, suspected fetal growth restriction, preeclampsia, and type 1 diabetes) should be monitored with continuous FHR monitoring.

There are no comparative data indicating the optimal frequency at which intermittent auscultation should be performed in the absence of risk factors. One method is to evaluate and record the FHR at least every 15 minutes in the active phase of the first stage of labor and at least every 5 minutes in the second stage (14).

▶ *What is the interobserver and intraobserver variability of intrapartum electronic fetal heart rate monitoring assessment?*

There is high interobserver and intraobserver variability in the interpretation of FHR tracings. For example, when four obstetricians examined 50 cardiotocograms, they agreed in only 22% of the cases (15). Two months later, during the second review of the same 50 tracings, the clinicians interpreted 21% of the tracings differently than they did during the first evaluation. In another study, five obstetricians independently interpreted 150 cardiotocograms (16). The obstetricians interpreted the tracings similarly in 29% of the cases, suggesting poor interobserver reliability.

The interpretation of cardiotocograms is more consistent when the tracing is normal (17). With retrospective reviews, the foreknowledge of neonatal outcome may alter the reviewer's impressions of the tracing. Given the same intrapartum tracing, a reviewer is more likely to find evidence of fetal hypoxia and criticize the obstetrician's management if the outcome was poor versus good (18). Therefore, reinterpretation of the FHR tracing, especially if neonatal outcome is known, may not be reliable.

▶ *When should the very preterm fetus be monitored?*

The decision to monitor the very preterm fetus requires a discussion between the obstetrician, pediatrician, and patient concerning the likelihood of survival or severe morbidity of the preterm child (based on gestational age, estimated fetal weight, and other factors) and issues related to mode of delivery. If a patient undergoes a cesarean delivery for indications related to a preterm fetus, continuous monitoring should be used rather than intermittent auscultation. The earliest gestational age that this will occur may vary.

Nonreassuring FHR patterns may occur with up to 60% of women with preterm labor, with the most common abnormality being deceleration and bradycardia, followed by tachycardia and minimal or absent baseline variability (19). Variable decelerations are more common among preterm (55–70%) deliveries than term (20–30%) deliveries (20). If FHR abnormalities are persistent, intrauterine resuscitation, ancillary tests to ensure fetal well-being, and possibly delivery should be undertaken (21).

▶ *What medications can affect the fetal heart rate?*

Fetal heart rate patterns can be influenced by the medications administered in the intrapartum period. Most often, these changes are transient, although they sometimes lead to obstetric interventions.

Epidural analgesia with local anesthetic agents (ie, lidocaine, bupivacaine) can lead to sympathetic blockade, maternal hypotension, transient uteroplacental insufficiency, and alterations in the FHR. Parenteral narcotics also may affect the FHR. A randomized trial comparing epidural anesthesia with 0.25% of bupivacaine and intravenous meperidine reported that the variability was decreased, and FHR accelerations were significantly less common with parenteral analgesia compared with regional analgesia (22). The rates of decelerations and cesarean delivery for "nonreassuring" FHR tracings were similar for the two groups. A systematic review of five randomized trials and seven observational studies also noted that the rate of cesarean delivery for nonreassuring FHR was similar between those who did and those who did not receive epidural analgesia during labor (23).

Concern has been raised about combined spinal–epidural anesthesia during labor. An intent-to-treat analysis of 1,223 laboring women randomized to combined spinal–epidural anesthesia (10 mcg of intrathecal sufentanil, followed by epidural bupivacaine and fentanyl at the next request for analgesia) or intravenous meperidine (50 mg on demand, maximum 200 mg in 4 hours) noted a significantly higher rate of bradycardia and emergent cesarean delivery for abnormal FHR in the group randomized to combined spinal–epidural anesthesia (24). Neonatal outcome, however, was not significantly different between the two groups. There are some methodological concerns with this study. Another randomized controlled trial compared the occurrence of FHR tracing abnormalities in laboring women who received combined spinal–epidural anesthesia (n=41) to epidural anesthesia (n=46). In this study, FHR abnormalities were more common in women receiving combined spinal–epidural anesthesia (25). Additional trials are necessary to determine the potential safety and efficacy of the combined spinal–epidural technique.

Other medications that influence FHR tracing have been studied (see Table 2). Of note, multiple regression analysis indicated that decreased variability attributed to the use of magnesium sulfate was related to early gestational age but not the serum magnesium level (26). Studies report different findings with regard to the effect of magnesium on FHR patterns. Some show no independent effect; others show small changes in baseline or variability. In general, however, caution should be used in ascribing unfavorable findings on EFM to the use of magnesium alone.

Transient sinusoidal FHR patterns occurred in 75% of patients who received butorphanol during labor, but this was not associated with adverse outcomes (27). Fetuses exposed to cocaine did not exhibit any characteristic changes in the heart rate pattern, although they did have frequent contractions even when labor was unstimulated (28). As determined by computer analysis of cardiotocograms, a randomized trial reported that compared with meperidine, nalbuphine used for intrapartum analgesia decreased the likelihood of two 15-second accelerations over 20 minutes (29). In antepartum patients, administration of morphine decreased not only the fetal breathing movement but also the number of accelerations (30).

The effect of corticosteroids, which are used to enhance pulmonary maturity of fetuses during preterm labor, on FHR has been studied (Table 2). Among twins (31) and singletons (32, 33), the use of betamethasone transiently decreased the FHR variability, which returned to pretreatment status by the fourth to seventh day. There also may be a decrease in the rate of accelerations with the use of betamethasone. These changes, however, were not associated with increased obstetric interventions or with adverse outcomes (31). The biologic mechanism of this is unknown. Computerized analysis of the cardiotocograms indicates that use of dexamethasone is not associated with a decrease in the FHR variability (33).

▶ *What findings on EFM are consistent with normal fetal acid–base status?*

The presence of FHR accelerations generally ensures that the fetus is not acidemic. The data relating FHR variability to clinical outcomes, however, are sparse. Results of an observational study suggest that moderate FHR variability is strongly associated with an arterial umbilical cord pH higher than 7.15 (34). One study reported that in the presence of late or variable decelerations, the umbilical arterial pH was higher than 7.00 in 97% of the cases if the FHR tracing had normal variability (35). In another retrospective study, most cases of adverse neonatal outcome demonstrated normal FHR variability (36). This study is limited because it did not consider other characteristics of the FHR tracing, such as the presence of accelerations or decelerations. However, in most cases, normal FHR variability provides reassurance about fetal status and the absence of metabolic acidemia.

Table 2. Effects of Commonly Used Medications on Fetal Heart Rate Patterns

Medications	Comments	References
Narcotics	At equivalent doses, all narcotics (with or without added antiemetics) have similar effects: a decrease in variability and a decrease in the frequency of accelerations 75 mg meperidine = 10 mg morphine = 0.1 mg fentanyl = 10 mg nalbuphine	1–7
Butorphanol	Transient sinusoidal FHR pattern, slight increased mean heart rate compared with meperidine	8, 9
Cocaine	Decreased long-term variability	10, 11
Corticosteroids	Decrease in FHR variability with beta-methasone but not dexamethasone, abolishment of diurnal fetal rhythms, increased effect at greater than 29 weeks of gestation	12–15
Magnesium sulfate	A significant decrease in short-term variability, clinically insignificant decrease in FHR, inhibits the increase in accelerations with advancing gestational age	16, 17
Terbutaline	Increase in baseline FHR and incidence of fetal tachycardia	18, 19
Zidovudine	No difference in the FHR baseline, variability, number of accelerations, or decelerations	20

Abbreviation: FHR, fetal heart rate.

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▶ *Are there ancillary tests that can aid in the management of Category II or Category III fetal heart rate tracings?*

There are some ancillary tests available that help to ensure fetal well-being in the face of a Category II or Category III FHR tracing, thereby reducing the high false-positive rate of EFM.

In the case of an EFM tracing with minimal or absent variability and without spontaneous acceleration, an effort should be made to elicit one. A meta-analysis of 11 studies of intrapartum fetal stimulation noted that four techniques are available to stimulate the fetus: 1) fetal scalp sampling, 2) Allis clamp scalp stimulation, 3) vibroacoustic stimulation, and 4) digital scalp stimulation (37). Because vibroacoustic stimulation and digital scalp stimulation are less invasive than the other two methods, they are the preferred methods. When there is an acceleration following stimulation, acidemia is unlikely and labor can continue.

When a Category III FHR tracing is persistent, a scalp blood sample for the determination of pH or lactate may be considered. However, the use of scalp pH assessment has decreased (38), and this test may not even be available at some tertiary hospitals (39). There are likely many reasons for this decrease, including physician experience, difficulty in obtaining and processing an adequate sample in a short amount of time, and the need for routine maintenance and calibration of laboratory equipment that may be used infrequently. More importantly, scalp stimulation, which is less invasive, provides similar information about the likelihood of fetal acidemia as does scalp pH.

In one study, the sensitivity and positive predictive value of a low scalp pH (defined in the study as less than 7.21 because it is the 75th percentile) to predict umbilical arterial pH less than 7.00 was 36% and 9%, respectively (40). More importantly, the sensitivity and positive predictive value of a low scalp pH to identify a newborn with hypoxic–ischemic encephalopathy was 50% and 3%, respectively. However, the greater utility of scalp pH is in its high negative predictive value (97–99%). There are some data to suggest that fetal scalp lactate levels have higher sensitivity and specificity than scalp pH (40). However, a recent large randomized clinical trial that compared the use of scalp pH assessment to scalp lactate level assessment in cases of intrapartum fetal distress did not demonstrate a difference in the rate of acidemia at birth, Apgar scores, or neonatal intensive care unit admissions (41). Although scalp stimulation has largely replaced scalp pH and scalp lactate assessment in the United States, if available, these tests may provide additional information in the setting of a Category III tracing.

Pulse oximetry has not been demonstrated to be a clinically useful test in evaluating fetal status (42–44).

▶ *Are there methods of intrauterine resuscitation that can be used for Category II or Category III tracings?*

A Category II or Category III FHR tracing requires evaluation of the possible causes. Initial evaluation and treatment may include the following:

- Discontinuation of any labor stimulating agent
- Cervical examination to determine umbilical cord prolapse, rapid cervical dilation, or descent of the fetal head
- Changing maternal position to left or right lateral recumbent position, reducing compression of the vena cava and improving uteroplacental blood flow
- Monitoring maternal blood pressure level for evidence of hypotension, especially in those with regional anesthesia (if present, treatment with volume expansion or with ephedrine or both, or phenylephrine may be warranted)
- Assessment of patient for uterine tachysystole by evaluating uterine contraction frequency and duration

Supplemental maternal oxygen commonly is used in cases of an indeterminate or abnormal pattern. There are no data on the efficacy or safety of this therapy. Often, the FHR patterns persist and do not respond to change in position or oxygenation. In such cases, the use of tocolytic agents has been suggested to stop uterine contractions and perhaps avoid umbilical cord compression. A meta-analysis reported the pooled results of three randomized clinical trials that compared tocolytic therapy (terbutaline, hexoprenaline, or magnesium sulfate) with untreated controls in the management of a suspected nonreassuring FHR tracing (45). Compared with no treatment, tocolytic therapy more commonly improved the FHR tracing. However, there were no differences in rates of perinatal mortality, low 5-minute Apgar score, or admission to the neonatal intensive care unit between the groups (possibly because of the small sample size). Thus, although tocolytic therapy appears to reduce the number of FHR abnormalities, there is insufficient evidence to recommend it.

Tachysystole with associated FHR changes can be successfully treated with β_2-adrenergic drugs (hexoprenaline or terbutaline). A retrospective study suggested that 98% of such cases respond to treatment with a β-agonist (46).

When the FHR tracing includes recurrent variable decelerations, amnioinfusion to relieve umbilical cord compression may be considered (47). A meta-analysis of 12 randomized trials that allocated patients to no treatment or transcervical amnioinfusion noted that placement of fluid in the uterine cavity significantly reduced the rate of decelerations (RR, 0.54; 95% CI, 0.43–0.68) and cesarean delivery for suspected fetal distress (RR, 0.35; 95% CI, 0.24–0.52) (48). Because of the lower rate of cesarean delivery, amnioinfusion also decreased the likelihood that either the patient or the newborn will stay in the hospital more than 3 days (48). Amnioinfusion can be done by bolus or continuous infusion technique. A randomized trial compared the two techniques of amnioinfusion and concluded that both have a similar ability to relieve recurrent variable decelerations (49).

Another common cause of a Category II or Category III FHR pattern is maternal hypotension secondary to regional anesthesia. If maternal hypotension is identified and suspected to be secondary to regional anesthesia, treatment with volume expansion or intravenous ephedrine or both is warranted.

Summary of Recommendations and Conclusions

The following recommendations and conclusions are based on good and consistent scientific evidence (Level A):

- The false-positive rate of EFM for predicting cerebral palsy is high, at greater than 99%.
- The use of EFM is associated with an increased rate of both vacuum and forceps operative vaginal delivery, and cesarean delivery for abnormal FHR patterns or acidosis or both.
- When the FHR tracing includes recurrent variable decelerations, amnioinfusion to relieve umbilical cord compression should be considered.
- Pulse oximetry has not been demonstrated to be a clinically useful test in evaluating fetal status.

The following conclusions are based on limited or inconsistent scientific evidence (Level B):

- There is high interobserver and intraobserver variability in interpretation of FHR tracing.
- Reinterpretation of the FHR tracing, especially if the neonatal outcome is known, may not be reliable.
- The use of EFM does not result in a reduction of cerebral palsy.

The following recommendations are based on expert opinion (Level C):

- A three-tiered system for the categorization of FHR patterns is recommended.
- The labor of women with high-risk conditions should be monitored with continuous FHR monitoring.
- The terms hyperstimulation and hypercontractility should be abandoned.

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Studies were reviewed and evaluated for quality according to the method outlined by the U.S. Preventive Services Task Force:

- I Evidence obtained from at least one properly designed randomized controlled trial.
- II-1 Evidence obtained from well-designed controlled trials without randomization.
- II-2 Evidence obtained from well-designed cohort or case–control analytic studies, preferably from more than one center or research group.
- II-3 Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments also could be regarded as this type of evidence.
- III Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

Based on the highest level of evidence found in the data, recommendations are provided and graded according to the following categories:

Level A—Recommendations are based on good and consistent scientific evidence.

Level B—Recommendations are based on limited or inconsistent scientific evidence.

Level C—Recommendations are based primarily on consensus and expert opinion.

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ACOG Committee Opinion

Committee on Obstetric Practice

Number 348, November 2006

Umbilical Cord Blood Gas and Acid-Base Analysis

ABSTRACT: Umbilical cord blood gas and acid-base assessment are the most objective determinations of the fetal metabolic condition at the moment of birth. Moderate and severe newborn encephalopathy, respiratory complications, and composite complication scores increase with an umbilical arterial base deficit of 12–16 mmol/L. Moderate or severe newborn complications occur in 10% of neonates who have this level of acidemia and the rate increases to 40% in neonates who have an umbilical arterial base deficit greater than 16 mmol/L at birth. Immediately after the delivery of the neonate, a segment of umbilical cord should be double-clamped, divided, and placed on the delivery table. Physicians should attempt to obtain venous and arterial cord blood samples in circumstances of cesarean delivery for fetal compromise, low 5-minute Apgar score, severe growth restriction, abnormal fetal heart rate tracing, maternal thyroid disease, intrapartum fever, or multifetal gestation.

Laboratory research demonstrates a complex relationship between fetal (antepartum and intrapartum) asphyxia, newborn asphyxia, and possible resulting brain damage. The degree, duration, and nature of the asphyxic insult are modulated by the quality of the cardiovascular compensatory response. A task force set up by the World Federation of Neurology Group defined asphyxia as a condition of impaired blood gas exchange, leading, if it persists, to progressive hypoxemia and hypercapnia (1). This is a precise definition of asphyxia as it may affect the fetus and neonate. In the American College of Obstetricians and Gynecologists' Task Force on Neonatal Encephalopathy and Cerebral Palsy report, asphyxia is defined as:

> . . . [a] clinical situation of damaging acidemia, hypoxia, and metabolic acidosis. This definition, although traditional, is not specific to cause. A more complete definition of birth asphyxia includes a requirement for a recognizable sentinel event capable of interrupting oxygen supply to the fetus or infant. This definition fails to include conditions that are not readily recognized clinically, such as occult abruption, but is probably correct in a majority of cases. (2)

Asphyxia may occur in a transient fashion that, although of physiologic interest, has no pathologic sequelae. Significant fetal exposure to asphyxia

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leads to tissue oxygen debt, accumulation of fixed acids, and a metabolic acidosis. Thus, for intrapartum fetal asphyxia the following addition is proposed for this definition:

> Fetal asphyxia is a condition of impaired blood gas exchange leading to progressive hypoxemia and hypercapnia with a significant metabolic acidosis. The diagnosis of intrapartum fetal asphyxia requires a blood gas and acid-base assessment. The important question for the clinician is what is the threshold of metabolic acidosis beyond which fetal morbidity or mortality may occur?

Low and associates have proposed a scoring system for predicting the likelihood of neonatal encephalopathy (3). They defined umbilical arterial base deficits at birth as mild at 4–8 mmol/L, moderate at 8–12 mmol/L, and severe at greater than 12 mmol/L. Newborn complications in the central nervous system, respiratory system, cardiovascular system, and kidney during the 5 days after delivery were documented. Assessment of the central nervous system included clinical evidence of newborn encephalopathy defined as minor with irritability or jitteriness, moderate with profound lethargy or abnormal tone, and severe with coma or abnormal tone and seizures. Cardiovascular complications were classified as minor with bradycardia (100 beats per minute or less) or tachycardia (170 beats per minute or more), moderate with hypotension or hypertension (defined by the 95% confidence limits for blood pressure in term neonates), and severe with abnormal electrocardiographic or echocardiographic findings. Respiratory complications were classified as minor if requiring supplementary oxygen, moderate if requiring continuous positive airway pressure or ventilation less than 24 hours, and severe if requiring mechanical ventilation more than 24 hours. Abnormalities of renal function were classified as minor if hematuria was observed, moderate with an elevation of serum creatinine level (greater than 100 mmol/L)*, and severe with anuria or oliguria (less than 1 mL/kg/h). A scoring system expressed the magnitude of the complications in each neonate. The score for each complication was "1" for minor, "2" for moderate, and "4" for severe. The maximum complication score was "16". Moderate and severe newborn encephalopathy, respiratory complications, and composite complication scores were increased with an umbilical arterial base deficit of 12–16 mmol/L. Moderate or severe newborn complications occurred in 10% of neonates with this level of acidemia, increasing to 40% in neonates with an umbilical arterial base deficit greater than 16 mmol/L at birth. Low and associates concluded that the threshold of fetal metabolic acidosis at delivery associated with moderate or severe newborn complications was an umbilical arterial base deficit of 12 mmol/L and that increasing levels of metabolic acidosis were associated with a progression of the severity of newborn complications (3). At the mild base deficit range, there is no association with abnormal newborn outcome. A similar threshold for neonatal neurologic complications has been reported by other investigators (4, 5). Importantly, and in contrast to moderate or severe levels of acidemia, term neonates exposed to mild antepartum fetal asphyxia were not at an increased risk of minor motor or cognitive defects at the age of 4–8 years compared with controls with no evidence of asphyxia (6).

*In the United States, creatinine level is expressed in mg/dL. To convert creatinine in mmol/L to mg/dL, the value should be divided by 88.4. In this case, 100 mmol/L is 1.14. mg/dL.

Term Infants

The prevalence of fetal asphyxia, ranging from mild to severe at delivery, in the term infant is reported at 25 per 1,000 live births; of these, 15% are either moderate or severe (3.75 per 1,000) (7). Even at these levels of acidemia, it must be appreciated that most fetuses will not be injured, yielding a final overall incidence of neonatal encephalopathy attributable to intrapartum hypoxia, in the absence of any other preconception or antepartum abnormalities, of approximately 1.6 per 10,000 (8, 9). Similar observations have been reported from Japan, where among a series of 10,030 infants there were nine cases of cerebral palsy at age 1 year or older diagnosed by pediatric neurologists. Analysis of these cases reveals that preexisting asphyxia existed before the initiation of fetal monitoring in six cases; two of the cases involved cytomegalovirus infections and one case involved a maternal amniotic fluid embolism (10). These investigators concluded that in low-risk pregnancies, cerebral palsy caused by intrapartum asphyxia was restricted to unavoidable intrapartum accidents.

Preterm Infants

Low and colleagues reported that the prevalence of asphyxia in preterm infants was 73 per 1,000 live

births (7). Of these, 50% were at the moderate to severe level of asphyxia. The authors caution that it remains to be determined how often the asphyxia recognized at delivery may have been present before the onset of labor. This point is particularly germane in the preterm infant, inasmuch as medical or obstetric complications or both often are the preceding event necessitating the preterm delivery. Examples include significant degrees of intrauterine growth restriction, placental abruption, chorioamnionitis with funisitis, and severe preeclampsia, each of which has been shown to be a significant independent risk factor for moderate or severe neonatal encephalopathy (8, 9).

Acidemia and Cerebral Palsy

Both the International Cerebral Palsy Task Force and the American College of Obstetricians and Gynecologists' Task Force on Neonatal Encephalopathy and Cerebral Palsy have published criteria to define an acute intrapartum event as sufficient to cause cerebral palsy (2, 11). Among the essential criteria cited by both task forces is evidence of metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH less than 7 and base deficit greater than or equal to 12 mmol/L) (see box). Additionally, the National Collaborating Center for Women's and Children's Health, commissioned by the National Institute for Clinical Excellence, has recommended that umbilical artery pH be performed after all cesarean deliveries for suspected fetal compromise, to allow review of fetal well-being and to guide ongoing care of the infant (12).

Technique for Obtaining Cord Blood Samples

Immediately after the delivery of the neonate, a segment of umbilical cord should be double-clamped, divided, and placed on the delivery table pending assignment of the 5-minute Apgar score. Values from the umbilical cord artery provide the most accurate information regarding fetal and newborn acid-base status. A clamped segment of cord is stable for pH and blood gas assessment for at least 60 minutes, and a cord blood sample in a syringe flushed with heparin is stable for up to 60 minutes (13, 14). If the 5-minute Apgar score is satisfactory and the infant appears stable and vigorous, the segment of umbilical cord can be discarded. If a serious abnormality that arose in the delivery process or a problem with the neonate's condition or both persist at or beyond the first 5 minutes, blood can be drawn from the cord segment and sent to the laboratory for blood gas analysis. Analysis of paired arterial and venous specimens should prevent debate over whether a true arterial specimen was obtained. Therefore, the Committee on Obstetric Practice recommends obtaining an arterial umbilical cord blood sample, but, where possible, obtaining both venous and arterial samples (paired specimen). It is important to label the sample as either venous or arterial. Similarly, in known high-risk circumstances, such as severe growth restriction, an abnormal fetal heart rate tracing, maternal thyroid disease, intrapartum fever, or multifetal gestations, it is prudent to obtain blood gas and acid-base assessments (2). It should be noted that it occasionally may be difficult to obtain an adequate cord arterial blood sample. If the practitioner encounters difficulty in obtaining arterial blood from the umbilical cord (ie, in a very preterm infant), a sample obtained from an artery on

Criteria to Define an Acute Intrapartum Hypoxic Event as Sufficient to Cause Cerebral Palsy

Essential criteria (must meet all four):

1. Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit ≥12 mmol/L)
2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type*
4. Exclusion of other identifiable etiologies, such as trauma, coagulation disorders, infectious conditions, or genetic disorders

*Spastic quadriplegia and, less commonly, dyskinetic cerebral palsy are the only types of cerebral palsy associated with acute hypoxic intrapartum events. Spastic quadriplegia is not specific to intrapartum hypoxia. Hemiparetic cerebral palsy, hemiplegic cerebral palsy, spastic diplegia, and ataxia are unlikely to result from acute intrapartum hypoxia (Nelson KB, Grether JK. Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. Am J Obstet Gynecol 1998;179:507–13.).

Excerpted from American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Neonatal encephalopathy and cerebral palsy: defining the pathogenesis and pathophysiology. Elk Grove Village (IL): AAP; Washington, DC: ACOG; 2003. Modified from MacLennan A. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. BMJ 1999;319:1054–9.

the chorionic surface of the placenta will provide accurate results (15). These arteries are relatively easy to identify because they cross over the veins.

Conclusion

Umbilical cord arterial blood acid-base and gas assessment remains the most objective determination of the fetal metabolic condition at the moment of birth. Thresholds have been established below which it is unlikely that an intrapartum asphyxial insult will have resulted in neurologic injury to the infant. Additionally, most infants born with umbilical arterial metabolic acidemia at a level consistent with causing a neurologic injury will, in fact, develop normally.

Physicians should attempt to obtain venous and arterial cord blood samples in the following situations:

- Cesarean delivery for fetal compromise
- Low 5-minute Apgar score
- Severe growth restriction
- Abnormal fetal heart rate tracing
- Maternal thyroid disease
- Intrapartum fever
- Multifetal gestations

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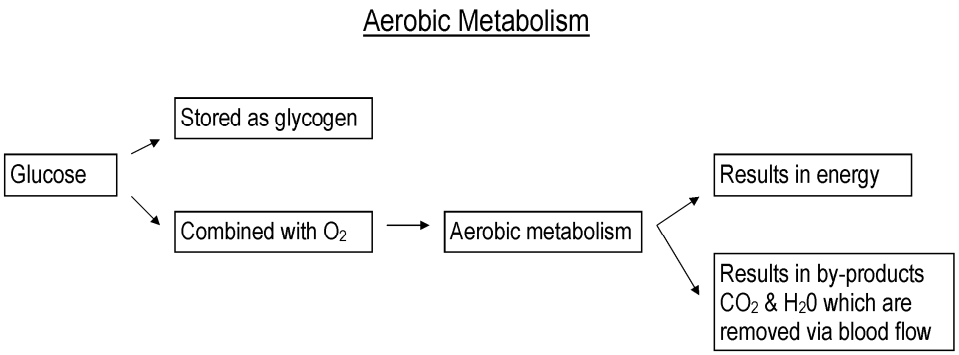
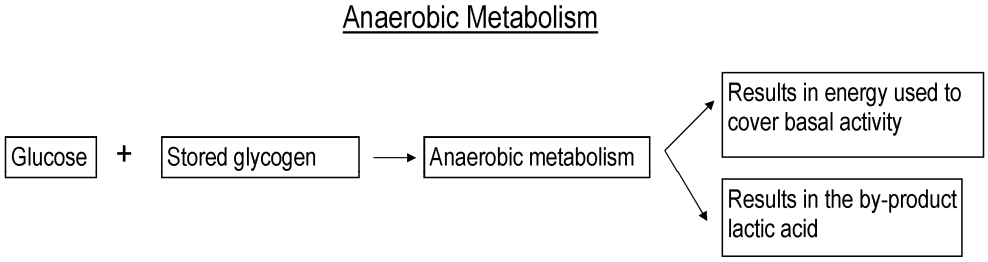
Understanding Fetal Acid-Base Evaluation

Aerobic vs. Anaerobic Metabolism

To understand acid-base evaluation in the fetus, it is helpful to have a brief review of fetal metabolism. In a healthy, well-oxygenated fetus, the primary mode of producing energy is via aerobic metabolism which is oxygen dependent.



When oxygen isn't available for this process, the fetus uses anaerobic metabolism, which depends upon glucose and conversion of glycogen stores. The energy produced by anaerobic metabolism is much less than that produced by aerobic metabolism and is used to cover basal metabolic needs.



Respiratory vs. Metabolic Acidemia

Remember, in normal *aerobic* metabolism, the end products are energy, CO_2 & H_2O. When uterine, placental & cord blood flow are all functioning properly, the by-products CO_2 & H_2O are efficiently cleared. If blood flow is decreased, CO_2 may not be effectively removed and will accumulate, quickly turning into hydrogen & bicarbonate ions. The bicarbonate ions shift into the tissue. The accumulation of free hydrogen ions in the blood causes a decrease in pH. This results in a *respiratory* acidemia and is related to the accumulation of CO_2.

Should blood flow decrease resulting in significant hypoxia, the peripheral tissues will shift into *anaerobic* metabolism, utilizing glucose as well as any stored glycogen. Lactic acid is the by-product here and when the amount of lactic acid exceeds fetal buffering capacity, *metabolic* acidosis is the result. Should the hypoxia become severe enough (or prolonged enough), metabolic acidosis may occur not only in the peripheral tissues, but it may extend to the vital organs (brain, heart, adrenals) where blood flow was initially redistributed as a protective mechanism. Once metabolic acidosis reaches these vital organs, the fetus is at risk for organ damage. Because clinicians cannot directly measure metabolic *acidosis* (tissues), cord gases are evaluated for *acidemia* (blood) as the blood levels represent what is happening in the tissues. Most clinicians use the terms acidosis & acidemia interchangeably in clinical practice, but it is important to note that when reviewing the fetal response to ongoing hypoxemia, the progression is always hypoxemia >>> hypoxia >>> metabolic acidosis >>> metabolic acidemia.

The severity of metabolic acidemia is evidenced by the base deficit (reported as a positive number, known as base excess when reported as a negative number). The greater the base deficit, the more the fetus has “used up”, or exceeded, its buffering capacity and therefore the more severe the metabolic acidemia.

The following tables provide information on normal umbilical venous and arterial blood gases as well as respiratory vs. metabolic acidosis.

Normal Cord Blood Gas Values:

	VEIN	ARTERY
pH	7.28 $\pm$ 0.05	7.25 $\pm$ 0.05
p02	29 $\pm$ 5.9 mmHg.	18 $\pm$ 6.2 mmHg.
pC02	38 $\pm$ 5.6 mmHg.	49 $\pm$ 8.4 mmHg.
HCO3	20 $\pm$ 2.1	22 $\pm$ 2.5
Base Excess	-4 $\pm$ 2 mEq/Liter	-4 $\pm$ 2 mEq/Liter

Adapted from: Yeomans ER, Hauth JC, Gilstrap LC III, Stickland DM. Umbilical cord pH, P_{CO_2}, and bicarbonate following uncomplicated term vaginal deliveries. Am J Obstet Gynecol 1985;151:798–800. Normal values are considered to be those within $\pm$ 2SDs.

Respiratory vs. Metabolic Acidemia in Cord Gases

	Respiratory Acidemia	Metabolic Acidemia
pH	< 7.20	< 7.20 (significant is < 7.0)
P02	>20 mmHg	< 20 mmHg
pC02	> 60 mmHg	< 60 mmHg.
HCO3	> 22 mEq/liter	< 22 mEq/liter
Base Excess	< -12 mEq/liter	> -12mEq/liter

Respiratory Acidemia	Metabolic Acidemia	Mixed Acidemia
Low pH	Low pH	Low ph
High pC02	Normal pC02	High pCO2
Normal pO2 & HCO3	Low pO2 & HCO3	Low pO2 & HCO3
Normal base excess	Elevated base excess	Elevated base excess

Resources & Additional Reading

Books

Pocket Guide to Fetal Monitoring: A Multidisciplinary Approach (6th ed.)
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Mistakes Were Made, But Not By Me
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