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OBG
MANAGEMENT

Electronic fetal monitoring: The difficulty of linking patterns with outcomes

Nonreassuring tracings don't always correlate with adverse outcomes, and vice versa. A look at what we know and don't know from evidence to date.

True or false: Electronic fetal monitoring reduces the incidence of cerebral palsy and infant morbidity and mortality.

Unfortunately, the statement is false, although patients continue to believe it is true. As a result, a large proportion of obstetrics liability cases center on electronic fetal monitoring (EFM): 43% of all lawsuits alleging obstetric malpractice, 52% of cases involving a stillborn fetus or neonatal death, and 66% of cases involving a neurologically impaired infant.[1]

Although the reliability and validity of EFM in reducing perinatal morbidity and mortality leave much to be desired, they could be greatly improved by increasing our understanding of the physiologic causes of patterns, establishing formal definitions for the various fetal heart rate (FHR) patterns, and developing specific recommendations for "abnormal" patterns.

Once these goals are attained, the question of whether EFM can be used to prevent

asphyxial brain damage can be more adequately addressed.

This article discusses the following topics:
- EFM patterns and their importance,
- key research findings to date, and
- how to identify patients at greatest risk of an adverse outcome.

Unfulfilled hopes based on flawed assumption

Evaluation of the FHR in labor—first by auscultation and then by continuous EFM—has long held promise of improved outcomes. As early as the 1800s, auscultated

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● **KEY** POINTS

■ **High-risk pattern.** Fetuses at greatest risk include those with marked bradycardia, recurrent late and variable decelerations, and absent variability.

■ **Normal pattern.** In contrast, a normal baseline rate with normal, moderate variability and accelerations, and absence of periodic patterns (decelerations) predicts fetal well-being.

■ **Tips on interpretation.** Deciphering fetal heart rate patterns entails making observations over time. Many patterns acquire increased significance when there is a trend toward persistent, significant departures from baseline with decreased variability, loss of accelerations, or persistent episodic or periodic decelerations, particularly with loss of variability.

FHR decelerations and prolonged bradycardias were seen as signs of fetal distress in labor. When criteria were being developed for obstetric intervention with forceps in the late 1800s, tachycardia was included as an indication, building the foundation for contemporary definitions of nonreassuring FHR patterns.

Many decades later, abnormal FHR patterns correlated with low Apgar scores and neonatal death (although the correlation between certain patterns and poor outcomes was weak) in the findings of the American Collaborative Perinatal Study,[2] which reviewed 25,000 births monitored using intermittent auscultation from 1959 through 1965.

The largely flawed assumption that most cases of cerebral palsy are caused by intrapartum asphyxia led to a concerted effort to decrease the incidence of asphyxia through EFM. By the 1970s, the expectation was that EFM would decrease perinatal morbidity and mortality.

Since its introduction into clinical practice in the early 1970s, EFM has become increasingly common; its use grew from 61% of women giving birth in 1989 to 84% in 1998.[3]

Findings of randomized controlled trials. The first randomized controlled trial[4] of EFM in labor, in 1976, demonstrated no improvement in outcome but a significant increase in surgical births; later trials showed no decrease in perinatal mortality or cerebral palsy.

The 1985 Dublin trial,[5] which scored the highest in study design in a later meta-analysis, showed a slight protective effect of EFM overall.

A 1995 meta-analysis[6] of 12 randomized controlled trials encompassing 58,555 pregnancies identified 9 outcomes, including Apgar scores, perinatal mortality, and neonatal seizures. EFM had a consistent impact only on neonatal seizures, and had no measurable effect on morbidity and mortality. Overall, 1.1% of neonates in the auscultated group had seizures, compared with 0.8% in the EFM group. Operative deliveries also increased in the EFM group.

EFM effects on neonatal outcomes. Freeman[7] summarized the gains of EFM from 1972 to 1990 as follows:
- no effect on cerebral palsy,
- no effect on neonatal morbidity, and
- no improvement in neurologic outcomes in premature infants.

In 1995, Vintzileos and colleagues[8] studied fetal acidemia in 680 auscultated labors and compared them with 739 monitored by EFM, using umbilical artery pH of less than 7.15 as an outcome measure. EFM was superior to auscultation, with better sensitivity and higher positive and negative predictive value.

Identification of patterns still rests on visual interpretation

In 1997, after a workshop to develop standardized, unambiguous definitions for fetal heart tracings, the National Institute for Child Health and Human Development (NICHD) issued guidelines for interpreting EFM[9]; these focus on visual interpretation, though computerized analyses are being developed. The definitions emphasize intrapartum evaluation, and the major patterns are categorized as baseline, periodic, or episodic.

Variability is defined by amplitude and includes short-term (beat to beat) and long-term variability, with no distinction between the two. Sinusoidal patterns are excluded from the definition.

According to NICHD guidelines, variabilities can be defined as follows:
- amplitude undetectable = absent variability;
- amplitude of 0 to 5 beats per minute (bpm) = minimal variability;
- 5 to 25 bpm = moderate variability; and
- amplitude exceeding 25 bpm = marked variability.

Loss of variability is the most sensitive indicator of fetal acidemia, cerebral asphyxia, and myocardial depression, when it is associated with other periodic patterns.[10]

Baseline rate. Baseline heart rate is the approximate mean rounded to 5 bpm during

a 10-minute interval, excluding periodic or episodic changes, periods of marked variability, and segments of the baseline that differ by more than 25 bpm. The baseline must continue for 2 minutes during that 10-minute period or it is considered indeterminate. A normal baseline generally ranges from 110 to 160 bpm. Below 110 bpm is bradycardia, and above 160 bpm is tachycardia.

Tachycardia and bradycardia indicate changes in the baseline rate.

Causes of tachycardia include maternal fever, sympathomimetics, and fetal arrhythmia. Tachycardia with a loss in variability and recurrent late or variable decelerations may indicate fetal acidemia.

In contrast, bradycardia ranging between 80 and 110 bpm does not indicate fetal hypoxia if the variability is retained, but a rate less than 80 bpm may indicate fetal hypoxia. The fetus may demonstrate terminal bradycardia associated with prolonged head or cord compression, but may tolerate this as long as variability is maintained. Sinusoidal patterns are excluded.

Decelerations are quantified by depth of the nadir (below the baseline) in bpm, with the duration measured in minutes or seconds from the beginning to the end of the deceleration (**FIGURES 1** and **2**). Those that occur more than 50% of the time are recurrent or periodic.

Acceleration is an abrupt change above baseline. Before 32 weeks, accelerations are defined as 10 or more bpm over baseline for 10 seconds or more. Peak acceleration is a minimum of 15 bpm above baseline, with a duration ranging from 15 seconds to 2 minutes before return to baseline. A prolonged acceleration continues for more than 2 minutes; if it lasts more than 10 minutes, it is considered a change in baseline.

Periodic and episodic patterns. Periodic patterns include early, late, and variable decelerations associated with uterine contractions. Episodic patterns are those not associated with uterine contractions.

A late deceleration is a visually apparent gradual decrease below the baseline for 30 seconds (at the nadir) that is delayed in timing relative to the uterine contraction. The recovery also is delayed relative to the end of the uterine contraction. Late decelerations that maintain variability are thought to indicate a well-oxygenated fetus and are neurogenic in origin,[11] but late decelerations without variability are thought to represent possible asphyxia. In fetuses with prolonged placental insufficiency, these smooth late decelerations are thought to reflect decreased cerebral and myocardial function.

Early decelerations are visually apparent decreases of 30 seconds (to the nadir) that start and end with a uterine contraction. They are not associated with significant fetal acidemia.

Variable decelerations are abrupt and visually apparent decelerations of 15 bpm below the baseline; they last more than 15 seconds to the nadir and less than 2 minutes overall. Variable decelerations are generally due to head compression, vagal stimulation, or cord compression. When variable decelerations are persistent and severe, tachycardia, delayed return to baseline, or decreased variability may occur, reflecting fetal acidemia.[12]

Prolonged late and severe variable decelerations may lead to fetal hypoxia. A duration between 2 and 10 minutes indicates a prolonged deceleration. A change of 10 minutes or more indicates a change in baseline.

Absent or severe variability with persistent late and prolonged decelerations is generally believed to be ominous and may correlate with hypoxia of such severity that the fetal central nervous system may have already been damaged. Surrogate markers for intrapartum asphyxia include mixed acidemia at birth, low Apgar score (less than 3 at 5 minutes), seizures within 24 hours, and multiorgan dysfunction.

High-risk pattern. Fetuses at greatest risk exhibit marked bradycardia, recurrent late and variable decelerations, and absent variability.

Normal pattern. In contrast, a normal baseline with moderate variability and accelera-

FIGURE 1

Late decelerations

A

B
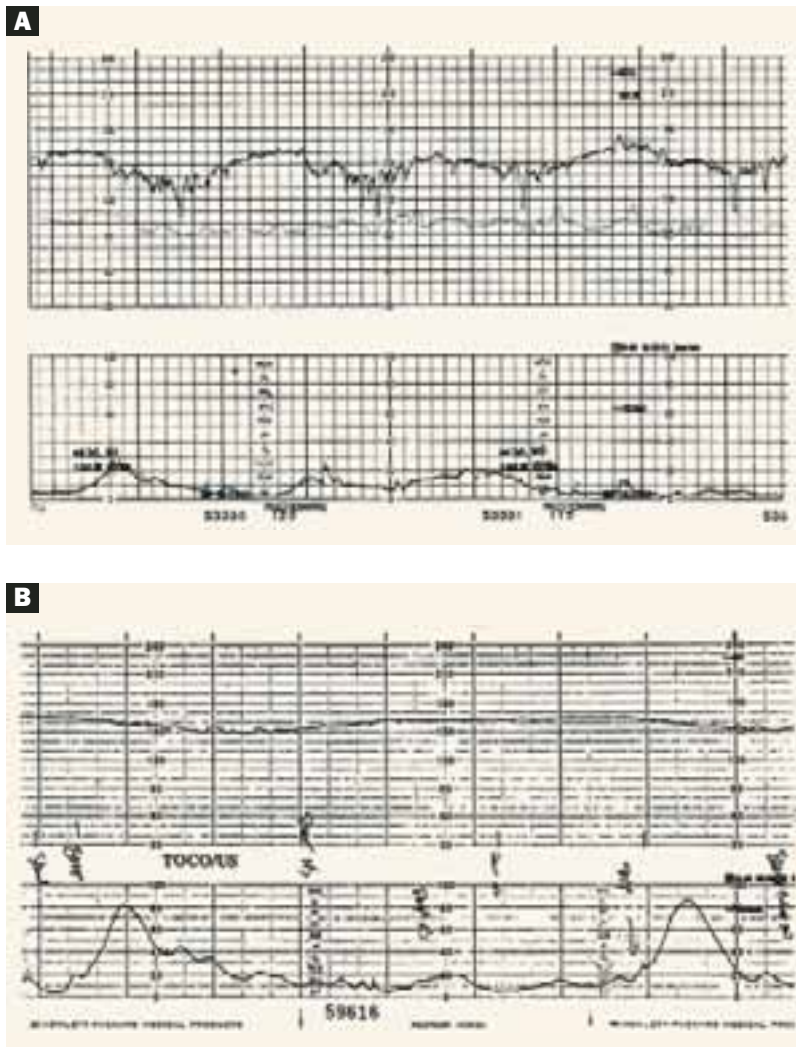
Late decelerations with **A)** variability and **B)** no variability. Late decelerations without variability are thought to represent possible asphyxia.

tions and absence of periodic patterns (decelerations) is predictive of fetal well-being.

Tips on interpretation. Deciphering EFM patterns entails making observations over time. Many patterns acquire increased significance when there is a trend toward persistent, significant departures from baseline with decreased variability, loss of accelerations, or persistent episodic or periodic decelerations, particularly with loss of variability.

Human factors

Agreement between observers. The reliability and reproducibility of EFM interpretation have been reviewed, but prospective studies to evaluate them are lacking. Interobserver agreement is reasonably high for the baseline rate, accelerations, and decelerations, but lower for variable decelerations and lower still for variability. Computers have been investigated for standardizing EFM interpretation.

Competency of physicians and nurses. A study[14] assessing competency in the evaluation of EFM tracings surveyed 43 Ob/Gyn training programs.

Researchers found that the majority (79%) use clinical experience along with structured lectures (87%) to train residents and fellows. Perinatal morbidity and mortality conferences were used by 85% of programs, and maintenance of EFM skills involved both clinical experience and case studies.

Although registered nurses typically undergo annual competency evaluation in EFM, physician competency is not formally evaluated. Thus, some other mechanism for ensuring expertise would be helpful.

Unreliable link to outcomes

The correlation between FHR patterns and neonatal outcomes is difficult to establish. The causative event in 70% of neonatal encephalopathy cases is believed to have occurred before the onset of labor[13]; therefore, few cases of cerebral palsy and neonatal encephalopathy are amenable to interventions during labor. Abnormal patterns that do not allow sufficient time for intervention also may have poor outcomes.

Fetuses that have intrapartum asphyxia usually have abnormal heart rate patterns. However, most women with abnormal FHR patterns give birth to neonates with normal Apgar scores and outcomes.

The central tenet of the 2003 monograph[13] by the American College of Obstetricians and Gynecologists is that in order for an intrapartum event to progress to cerebral palsy, the pathway must proceed through neonatal encephalopathy. However, only 30% of neonatal encephalopathy cases are due to events during labor. Further, epilepsy, mental retardation, and attention deficit disorder are not caused by birth asphyxia.

A 99% false-positive rate may result when nonreassuring EFM tracings are used to predict cerebral palsy. For this reason, there is still considerable disagreement about whether EFM can reduce the incidence of cerebral palsy. Using the criteria for establishing acute hypoxic-ischemic insult (TABLE 1) and for determining that intrapartum events were sufficient to cause cerebral palsy (TABLE 2), only 9% of cerebral palsy cases are attributable to possible birth asphyxia.[11]

FIGURE 2

Severe deceleration
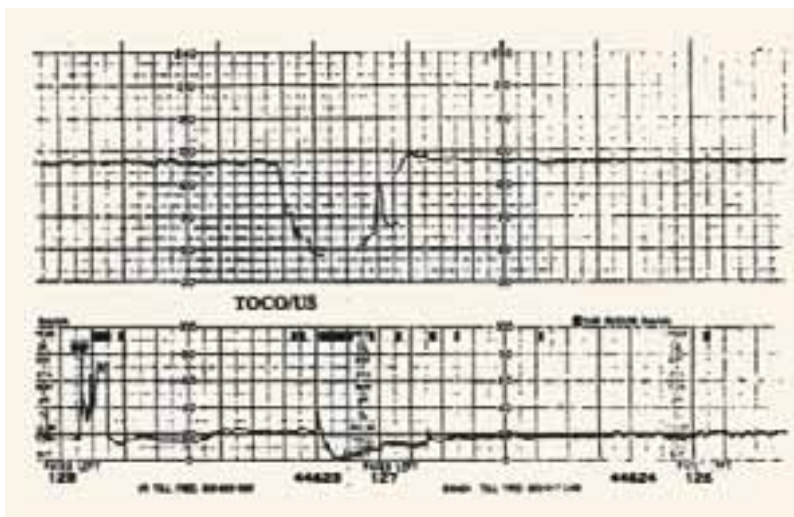
A severe deceleration with variability. When variable decelerations are persistent and severe, fetal acidemia may be present.

Most cases of catastrophic hypoxia ultimately lead to fetal or neonatal death; a minority of infants survive and develop cerebral palsy. The pattern of neurologic injury following acute catastrophic hypoxic-ischemic insult can involve the basal ganglia and thalami; it may be different from the pattern that commonly follows chronic insult, which involves primarily the cerebral cortex and subcortical white matter.

Little time to react during labor

Severe intrapartum hypoxic-ischemic insult may be detected by EFM but, in some cases, the reaction time to prevent fetal brain injury is very short. These intrapartum events include umbilical cord prolapse, shoulder dystocia, uterine rupture, and maternal cardiopulmonary arrest. With uterine rupture, asphyxia may occur as quickly as 10 minutes after onset of bradycardia when the prolonged deceleration is preceded by severe late decelerations of 30 to 90 minutes. The average time from onset of prolonged bradycardia has been reported as 13 ± 6.5 minutes.[13]

TABLE 1

Criteria for establishing acute hypoxic-ischemic insult

Metabolic acidosis evident in fetal umbilical cord arterial blood at delivery
(pH <7.0 or base excess ≥12 mmol/L)

Early onset of severe or moderate neonatal encephalopathy in infants delivered at ≥34 weeks' gestation

Cerebral palsy of the spastic quadriplegic or dyskinetic type

Exclusion of other identifiable causes

Data from American College of Obstetricians and Gynecologists[13]

TABLE 2

Criteria to establish intrapartum event as cause of cerebral palsy

Sentinel hypoxic event occurring immediately before or during labor

Sudden sustained fetal bradycardia or absence of fetal heart rate variability in presence of persistent late or variable decelerations

Apgar score of less than 3 at 5 minutes

Onset of multisystem involvement within 72 hours of birth

Early imaging studies showing evidence of acute nonfocal abnormality.

Data from American College of Obstetricians and Gynecologists[13]

The 30-minute rule. Because mortality and morbidity may depend on how much time elapses from onset of the catastrophic event to delivery, ACOG developed guidelines for timing emergent cesarean. The 30-minute rule[13] advises that a cesarean be performed within 30 minutes of the decision to proceed. Unfortunately, in many cases, acute hypoxic-ischemic insult occurs in less than 30 minutes.

Many hospitals and obstetric services no longer offer vaginal birth after cesarean (VBAC) if they cannot guarantee that anesthesia, nursing, pediatric, and operating room services will be available within this time frame. However, other causes of sudden hypoxia—besides the small chance of uterine rupture with VBAC—may be neither predictable nor preventable.

General recommendations

In cases of nonreassuring FHR tracings, the options include maternal positioning to achieve uterine displacement; hydration; correction of hypotension, which may be useful when regional anesthetics are used; tocolytic use for treating a uterine contraction; maternal oxygen; amnio-infusion for thick meconium in the presence of a nonreassuring FHR tracing; and assessment and correction of the oxytocin dose when augmentation or induction of labor is occurring. ■

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