Fetal Monitoring

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Assessment of Fetal Maturity

One of the most important goals for the anesthesiologist caring for a pregnant women should be to maintain the uteroplacental unit and fetus in optimal condition. Hence, an adequate knowledge of uterine activity and fetal monitoring is important. Different devices are used for the intrapartum assessment of uterine activity as well as fetal well-being.

Assessment of uterine activity is important in predicting the normal progress of labor and also fetal well-being. Parameters related to uterine activity are (1) baseline uterine tone and amplitude, (2) duration of contractions, and (3) the interval between contractions. Normal baseline tone varies between 8 and 20mmHg and increases to between 25 and 75mmHg during contractions. However, the peak pressure can rise to 130mmHg with bearing-down efforts in the second stage of labor. A contraction can last from 30 to 90 seconds, and the interval between contractions normally varies from 2 to 3 minutes. A tocodynamometer can measure the parameters of uterine activity when placed on the abdominal wall.
However, one of the major limitations of external tocodynamometry is the possible reception of inaccurate data from improper positioning of the instrument on the abdominal wall. Internal monitoring is more accurate and reliable, and it measures both amniotic fluid and intrauterine pressure. The prerequisites of internal monitoring include (1) engagement of the presenting part, (2) adequate cervical dilatation, and (3) ruptured membranes. Internal measurement of uterine activity is commonly used in high-risk cases (e.g., diabetes, prematurity) as well as in parturients receiving epidural analgesia for the relief of labor pain.

**Fetal Heart Rate Monitoring**

The baseline heart rate, beat-to-beat variability (short and long term), and the fetal heart rate pattern (periodic changes) are the most important variables that should be followed in recording the fetal heart rate.

**Baseline Heart Rate**

The normal baseline fetal heart rate varies between 120 and 160 beats per minute (BPM), and it is modulated by parasympathetic and sympathetic nerve activity (Fig. 10-1).

Fetal tachycardia is diagnosed when the baseline escalates above 160 BPM. The major causes of fetal tachycardia are:
1. Fetal hypoxia due to any cause
2. Maternal fever, most often associated with infection
3. Maternal administration of sympathomimetic drugs, e.g., ephedrine, β-mimetic drugs for tocolysis (terbutaline), epinephrine
4. Maternal administration of parasympatholytic drugs, e.g., atropine, phenothiazines
5. Maternal hyperthyroidism
6. Fetal anemia
7. Fetal tachyarrhythmias

Fetal bradycardia is defined as a fetal heart rate less than 100 BPM. The following are major causes:
1. Fetal head compression or umbilical cord compression
2. Maternal administration of parasympathomimetic drugs, e.g., neostigmine
3. Maternal administration of β-blockers, e.g., propranolol
4. Prolonged fetal hypoxia for any reason
5. Fetal congenital heart block
6. Combined spinal epidural technique, especially in parturients with decreased ureteroplacental blood flow.

**Baseline Variability**

Baseline variability is generally recognized as the single most important parameter for the recognition of intrauterine fetal well-being. Baseline variability is due to a constant battle between the fetal sympathetic (increasing the heart rate) and parasympathetic systems (decreasing the heart rate). The presence of good baseline variability is an indicator of intact central nervous system as well as normal cardiac functions.
Baseline variability has been classified into (1) short-term variability, representing a beat-to-beat difference of 5 to 15 beats, and (2) long-term variability, which generally shows a frequency of 3 to 5 cycles per minute. *Fetal heart rate variability can be a very accurate indicator if it is used by direct fetal electrocardiogram monitoring*. Short-term variability is more important in predicting fetal well-being. Various factors that can affect this are as follows (Fig. 10-2):

1. *Maternal administration of narcotic drugs, e.g., meperidine (Demerol), morphine, alphaprodine (Nisentil), or butorphanol, which work by depressing the fetal central nervous system*

2. Maternally administered sedatives and hypnotics, e.g., barbiturates, diazepam, phenothiazines, or promethazine (Phenergan), which also affect the fetal central nervous system

![Figure 10-2. Decreased variability of the fetal heart rate.](image-url)
3. Maternally administered parasympatholytic drugs, e.g., atropine or phenothiazines
4. The use of inhalational anesthetics
5. Fetal sleep cycle
6. Extreme prematurity
7. Fetal tachycardia

Fetal Heart Rate Pattern (Periodic Changes)

Periodic changes are defined as transient accelerations or decelerations of short duration of the fetal heart rate followed by a return to baseline levels. There are three categories of decelerations: early, variable, and late.

**Early Decelerations**

Characteristics of early decelerations (Fig.10-3) are as follows:
1. Uniform. U-shaped deceleration
2. Slow onset and slow return to baseline
3. Exact mirror image of uterine contractions in duration
4. Acceleration of the fetal heart not preceding the onset of a contraction or following the end of a contraction
5. Fetal heart rate usually not falling below 20 to 30 BPM
6. Good beat-to-beat variability

Two mechanisms for early deceleration that have been suggested are (1) fetal head compression with increased intracranial pressure (Fig. 10-4) and (2) increased volume of blood entering the fetal circulation during contractions, thus triggering the baroceptor reflex activity. Both of these mechanisms are vagally mediated and can be prevented by atropine.  

**Variable Decelerations**

This is the most common of all fetal heart rate patterns (Fig. 10-5), and the characteristics of this pattern are as follows:
1. Variability in duration
2. Variability in shape and size
3. Variability in time
Figure 10-3. Early decelerations.
Figure 10–4. Mechanism of early decelerations (FHR = fetal heart rate). (Adapted from Freeman RK, Garite TS: Fetal Heart Rate Monitoring. Baltimore, Williams & Wilkins, 1981.)

This is probably caused by compression of the umbilical cord against the fetal body parts, e.g., head, neck, or shoulder. Because the maximum pressure on the cord is generated at the time of uterine contractions, the pattern coincides with uterine contraction. These decelerations are usually very abrupt in relation to both the onset and the return to baseline levels. Depending upon the magnitude of the decrease in the fetal heart rate, the variable decelerations have been further subdivided into (1) mild (duration less than 30 seconds and deceleration not below 80 BPM), (2) moderate (regardless of the duration, the fetal heart rate is less than 80 BPM), and (3) severe (duration greater than 60 seconds and a fetal heart rate less than 70 BPM). Occasionally, variable decelerations can change to late decelerations or severe fetal bradycardia.
Figure 10-5. Variable decelerations.
Late Decelerations

Definitely a sign of uteroplacental insufficiency, late decelerations (Fig. 10-6) are characterized by the following:
1. The onset of deceleration usually starts 30 seconds or more after the onset of uterine contraction.
2. The peak of the deceleration arrives long after uterine contraction.
3. The onset and return are gradual and smooth.
4. The drop in fetal heart rate usually varies between 10 and 20 BPM and rarely falls lower than 30 to 40 BPM.
5. Although not always, there is a correlation between the magnitude of decelerations and the degree of fetal hypoxia. The major cause of late decelerations is reduced placental perfusion, as can be seen during supine hypotensive syndrome because of aortocaval compression, severe hypotension following regional anesthesia, abruptio placentae, postmaturity, diabetes mellitus; pre-eclampsia/eclampsia, etc.

Besides these three main patterns, the other patterns that have been described are prolonged decelerations and a sinusoidal pattern.

Prolonged decelerations may be associated with a loss of variability and a baseline fetal heart rate below 70 BPM; once the duration exceeds 2 to 3 minutes, urgent intervention is usually necessary.

A sinusoidal pattern is associated with a sine wave pattern above and below the baseline with a cyclicity of about 4 to 8 minutes. Actually, there is an increased long-term variability. One of the major causes of this pattern is the severe fetal anemia usually associated with Rh incompatibly. A benign sinusoidal pattern has been associated with narcotics like alphaprodine (Nisentil) or butorphanol (Stadol).

Besides fetal heart rate monitoring, fetal scalp pH sampling is also very important in making an ultimate judgement of fetal well-being. Normal fetal scalp pH varies between 7.25 and 7.32; mild acidosis is documented when the pH varies between 7.20 and 7.24; and severe acidosis is noted when the pH becomes lower than 7.20. A good correlation has been observed between severity of the fetal heart rate pattern and fetal
Figure 10–6. Late decelerations. (Adapted from Martin R: Prepartum and intrapartum fetal monitoring, in Datta S (ed): Anesthetic and Obstetric Management of High Risk Pregnancy. Chicago, Mosby–Year Book, 1991.)
acidosis as observed by fetal scalp pH. At the present, most obstetricians are in favor of confirming fetal compromise as shown in fetal heart rate monitoring by routine sampling of fetal scalp pH.

**Biophysical Profile**

Peripartum evaluation of the high-risk fetus is done by evaluation of immediate biophysical activities: (1) fetal movement, (2) fetal tone, (3) fetal breathing movements, (4) heart rate activity, and (5) volume of amniotic fluid. The first four parameters reflect the presence of normal fetal central nervous system activity, whereas amniotic fluid volume is an indicator of long-term or chronic fetal compromise. These parameters are all measured by ultrasound except for the fetal heart rate. The variables are scored 2 if normal and 0 if abnormal. Fetal heart rate activity is measured by nonstress testing and the oxytocin challenge test.

**Nonstress Test**

This involves the detection of changes in the fetal heart rate and fetal movement in association with uterine contraction.

Usually this test is described as reactive if there are two fetal movements in 20 minutes with accelerations of the fetal heart rate of at least 15 BPM. The test is described as nonreactive in the absence of fetal movement and accelerations of the fetal heart rate.

**Contraction Stress Test or Oxytocin Challenge Test**

In the presence of a nonreactive nonstress test, the oxytocin challenge test becomes an important issue. Intravenous oxytocin is used, beginning at a rate of 0.5–1.0 units/min, to induce three adequate uterine contractions within a 10-minute period. The oxytocin challenge test is considered to be positive if persistent late decelerations exist, whereas the test is interpreted as negative in the presence of normal fetal heart
The oxytocin challenge test is contraindicated if there is history of classical cesarean section or placenta previa and if the parturient is at risk of premature labor.

A poor biophysical score (5 or less out of 10) indicates that close supervision of the fetus is necessary.

Assessment of Fetal Maturity

Phospholipids, the major components of lung surfactant, are produced by fetal alveolar cells in a sufficient amount by 36 weeks’ gestation. The lecithin/sphingomyelin ratio (L/S) is commonly used to predict fetal lung maturity and is said to be normal when the ratio is 2 in normal pregnancies. For diabetic parturients, the ratio should be at least 3.5 or higher. Measurements of saturated phosphotidylcholine are occasionally used in normal parturients; the normal value is 500 mg/dL, whereas in diabetics it is 1,000 mg/dL.

Recently, the TDx fetal lung maturity (FLM) test has become popular. It is expressed in milligrams of surfactant per gram of albumin (the cutoff value is 50 mg/g).

A FLM value of <50 mg/g indicates immature lung, between 50 and 70 mg/g is borderline, and >70 mg/g predicts adequate lung maturity. In the author’s institution, FLM is not used in diabetic parturients.

In summary, an adequate knowledge of the detection of normal uterine activity and fetal well-being is necessary so that anesthetic techniques do not interfere with uterine activity, the fetoplacental unit, or above all, the fetus.

References