This self study is designed as a prerequisite to the Advanced Fetal Monitoring Course and provides a core review of key principles of fetal monitoring. It is imperative that you have a working knowledge of the information contained in this self-study prior to taking the Advanced Course. In preparation for your participation in the Advanced Fetal Monitoring Course, it is necessary for you to understand fundamental information used in fetal heart monitoring (FHM). Since you are an experienced clinician, some of this information may be a review, but new aspects are also included. To streamline the Advanced Course and make it more interactive and interesting to you, this information is provided to you in a self-study format. Your participation in the course will be enhanced by your completion of this study packet prior to your attendance. This review may also enhance your clinical decision making. Your understanding of the information provided will also allow you to participate more fully in the workshop. Please be aware that the information contained in this self-study will be included in the competency test given at the conclusion of the course.
The nursing process model pictured on this slide was created for the original AWHONN FHMPP workshop (Feinstein, Torgersen & Atterbury, 2003). It provides the theoretical framework for the FHMPP and AFMC course and the textbook. The model highlights steps of the critical thinking process related to fetal heart monitoring and should be viewed as a problem-solving process represented by a continuum of assessment, interpretation, diagnosis, intervention, evaluation and collaboration among health care providers.

- **First, you perform a comprehensive initial assessment.**
- **You then combine the assessment information with your knowledge and experience to analyze and interpret the clinical situation.** It is important in this step that you examine the whole clinical picture and determine how the pieces of the data gathered interact with each other. It is also important that you base your assessment and interventions on the underlying physiology to help prioritize and plan care appropriately.
- **Individualized nursing diagnoses are developed from this interpretation and will guide your interventions.** The interventions are based on physiological goals. These diagnoses and interventions may be independent or collaborative.
- **Next, you evaluate your interventions to determine if your goals have been met.** If not, the process is repeated.
- Notice how the steps move in a circular motion and are never-ending. The intrapartum period is dynamic; therefore subsequent assessments will direct this ongoing nursing process. Decision making relating to fetal heart monitoring (FHM) data is based on critical thinking skills and their application to the nursing process.
A comprehensive approach is important when performing the initial assessment. As you can see on this slide, this approach includes gathering data from the woman’s history, including family, medical-surgical, obstetric and psychosocial issues. You should also assess medical-surgical, obstetric or psychosocial issues and concerns during the woman’s current pregnancy, including the presence and quality or absence of support persons. A confidential patient interview and complete physical assessment will provide the information needed to establish your nursing database (Mahoney, Torgersen, & Feinstein, 2003).

The patient’s stated physical and/or emotional needs typically guide the assessment process. For instance, if the woman arrives stating she feels an urge to push, your initial assessment would focus on determining if the patient is delivering imminently and, if so, then preparing for delivery. On the other hand, your assessment may uncover information that is unrelated to the woman’s expressed need and that may then become a top priority. For example, a patient may arrive complaining of uterine contractions. Upon further assessment, if she is found to have absent fetal heart tones, that data would require immediate further investigation and notification of the primary care provider; as well as provision of information and emotional support for the woman and her partner.
It is critical to include a review of the patient’s prenatal records, a patient interview and complete head-to-toe physical assessment to establish a nursing database. This database allows us to identify and confirm normal findings or risk factors. The risk assessment will play a lead role in your critical thinking and implementation of the nursing process on a continuum. This procedure may need to be abbreviated in an emergent situation with a focus on gathering data pertinent to the immediate situation. Reliance on verbal reports may be necessary initially, but it is essential to gather the complete information when possible.

- **What are some examples of risk factors commonly identified for intrapartum patients?**
  - Preeclampsia, postdates, intrauterine growth restriction (IUGR), diabetes, cigarette smoking, genetic factors, young maternal age, substance abuse, etc.
- After completing the risk assessment, as you can see on the slide, next you should think about how this will influence your nursing care for this woman.
- **What are some examples of how risk factors could affect the maternal-fetal status and our nursing care?**
  - Decreased placental functioning capacity, increased risk for fetal anomalies, oligohydramnios, decreased fetal movement, lack of coping mechanisms and/or support system, etc. Your nursing care will focus on being alert for clinical information that may be associated with these factors.
- **Let’s use an example: A patient admitted for an induction for oligohydramnios and intrauterine growth restriction (IUGR) is at risk for what potential physiologic problems?**
  - Cord compression and/or uteroplacental insufficiency.
- **What nursing diagnoses can we create for this woman?**
Two possible nursing diagnoses are:

- Risk for impaired fetal gas exchange related to umbilical cord compression secondary to oligohydramnios.
- Risk for ineffective fetal tissue perfusion related to uteroplacental perfusion insufficiency secondary to inadequate placental function.

Can you think of others?

For this type of patient, your main concerns are the increased risks for cord compression and/or decreased uteroplacental perfusion. Nursing interventions should address these concerns. Your interventions could include maintaining optimal uterine blood flow and umbilical circulation with lateral maternal positioning and hydration, as well as intrapartum fetal heart monitoring.

Your nursing diagnoses and nursing care, then, should reflect that you appreciate the patient's increased risk for what kind of fetal heart rate (FHR) characteristics? Variable and late decelerations and possibly decreased variability, depending on the patient’s individual circumstances. You would monitor for these FHR changes and intervene appropriately.
Let's now discuss extrinsic factors and their possible effects on the FHR.

**Extrinsic factors are influences outside of the fetus that affect the availability of oxygen to the fetus, thus affecting the FHR** (Goss & Torgersen, 2003).

As you can see on the slide, **maternal oxygen transport** is one part of the group of extrinsic factors that can influence the fetal heart rate. Maternal oxygen transport will be discussed in the AFMC.

Others are: **blood flow to the uterus, integrity of the placenta and the flow of blood through the cord**.

In our later discussion on interventions, you will see that oftentimes our interventions will affect one or more of these extrinsic factors, thus affecting the availability of oxygen to the fetus and potentially the fetal heart rate or pattern.

Let's take a closer look at the extrinsic factors.
Our discussion of extrinsic factors begins with uterine blood flow. First, it is important to understand how blood is supplied to the uterus.

- **Uterine blood flow is supplied through the maternal spiral arteries.** These arteries extend perpendicularly through the myometrium and endometrium, so contraction of uterine muscles compresses these arteries (King & Parer, 2000). Compression of the uterine arteries with contractions may adversely affect oxygenation of the fetus.
- Another interesting fact about maternal spiral arteries is a process that takes place in early placental development. *These tightly coiled arteries in a nonpregnant uterus should become widely dilated vessels in early pregnancy, thus accommodating the large increase in blood flow during pregnancy.* This process becomes clinically important because it causes the spiral arteries to lose their ability to autoregulate, or constrict if their internal pressure drops. *The blood supply to the placenta, then, is totally dependent on adequate blood flow to the uterus.*
- At times, the spiral arteries remain tightly coiled, therefore the blood flow to the placenta may be inadequate. This may cause a failure to meet the needs of a growing fetus (King & Parer, 2000).
As was mentioned on the previous slide, the blood flow to the placenta is totally dependent on adequate blood flow to the uterus. A variety of routine interventions can enhance uterine blood flow.

**What are some ways we can and should do this?**

- Maintaining maternal lateral position whenever possible, or ensuring a lateral tilt to avoid the supine position
- Ensuring adequate maternal hydration
- Enhancing maternal relaxation by providing labor support and comfort measures
- Providing appropriate pain management (Goss & Torgersen, 2003):

Conversely, there are factors that may decrease blood flow to the uterus.

**Can you think of some examples?**
Extrinsic Factors (cont.)

Uterine blood flow can be inhibited by:

• Supine positioning
• Maternal stress or exercise
• Uterine contractions
• Maternal hypotension
• Maternal hypertension

(Goss & Torgersen, 2003; Freeman, 2003)

Uterine blood flow can be decreased by many factors (Goss & Torgersen, 2003; Freeman, 2003):

• Supine positioning—compression of maternal great vessels may cause decreased venous return and decreased blood flow to the uterus
• Maternal stress or exercise—may divert blood away from uterus
• Uterine contractions—compression of uterine vessels
• Maternal hypotension from anesthesia or hypovolemia as examples—decreased blood flow to uterus
• Maternal hypertension—decreased blood flow to uterus and placenta

Certain medication may also affect uterine blood flow. These will be discussed throughout the course.

• The fetus may respond to decreased uterine blood flow by exhibiting particular fetal heart rate patterns. It is important to consider uterine blood flow and its effects on the oxygenation of the fetus when caring for your patients. Interventions should be directed to maximize uterine blood flow.
• Remember that uterine blood flow is important because it affects blood supply to the placenta, about 70–90% of the blood flow to the uterus passes through the intervillous spaces of the placenta (Goss & Torgersen, 2003).
The placenta functions as the fetus’s lungs, kidneys, and gastrointestinal and integumentary systems. It also functions as a protective barrier to the fetus and an endocrine organ by releasing hormones. (Freeman, 2003) The placental function focus for this class will be fetal oxygenation, since the purpose of continuous electronic FHR monitoring is to assess the fetal oxygenation status.

- The maternal side of the placenta at term is made up of 15 to 20 cotyledons or lobules. Within each cotyledon is a unit of maternal-fetal circulation. As we look closely at this picture of the placental structure, we can see from the center portion of our picture that the maternal arterial blood is pumped at high pressures into the intervillous space. The forceful presence of the arterial blood, with a mean arterial pressure (MAP) of approximately 90 mmHg, encourages the passive return of fetal oxygen-poor blood into the lower pressures of the maternal venous system (Goss & Torgersen, 2003).
- On the fetal side, each lobule contains the branches of a single large main stem villus.
- The maternal and fetal circulations are separated by two layers—the fetal connective tissue within the branches of the villus and the fetal capillary wall (Freeman, 2003).
- The intervillous space serves as the means for maternal-fetal transfer of oxygen, carbon dioxide (CO₂), nutrients and waste products. Oxygen and CO₂ exchange occurs by the method of passive diffusion (substances pass from gradients of high to low concentration) (Goss & Torgersen, 2003).
The integrity of the placenta and the blood supply to the placenta are extremely important! Anything that decreases the effective surface area of the placenta and/or the blood supply to the placenta will increase the potential for fetal malnutrition and potentially decrease oxygen delivery to the fetus.

- This slide illustrates the effects of placental transfer capacity on the limits of fetal transfer of nutrients, O₂ and CO₂.
- As you can see here, at a transfer capacity near 100%, the fetus is supplied with approximately twice the resources to meet its needs.
- When the transfer capacity decreases to around 75%, the transfer of the larger nutrient molecules are affected, and fetal malnutrition can result, potentially leading to intrauterine growth restriction (IUGR).
- At a transfer capacity of approximately 50%, O₂ and CO₂ exchange is decreased, which can result in significant fetal compromise.
When you perform your assessments and assign risk factors to patients, consider conditions that may affect the integrity of the placenta.

What are some factors that can affect the integrity of the placenta?

- **Maternal hypertension**—constriction of maternal vessels may decrease blood flow to the placenta. This can cause infarcts of the placenta, thus decreasing the functioning placental surface area.
- **Placental aging** can cause calcifications which, again, may decrease the functioning placental surface area.
- **Abruption** will also decrease the functional placental surface area. The amount of the abruption will determine the amount of placental surface area affected.
- **Abnormal placental development**, such as extrachorial placentation, circummarginate and circumvillate placentas, and inadequate lengthening of maternal spiral arteries (which we previously discussed) can also have an effect on placental function (Harvey & Chez, 1997).
- You should consider each patient’s placental function and observe fetal heart rate data for information indicating the possibility of poor placental function.

What FHR might indicate decreased placental function?
We would most likely see abnormal baseline ranges, late decelerations and/or absent FHR variability.
Blood flow through the umbilical cord is the last extrinsic factor we will examine.

The umbilical cord at term is approximately 50 to 60 cm long. It contains three vessels: one umbilical vein carrying oxygenated blood to the fetus, and two umbilical arteries carrying deoxygenated blood away from the fetus. These vessels are surrounded by a protecting substance called Wharton’s jelly (Harvey & Chez, 1997).

- Blood must flow freely through the cord to maximize oxygen delivery to the fetus. If the blood is not able to flow freely through the umbilical cord, you may see changes in the FHR.
- **What changes would you most likely see?** Variable decelerations.
- The assessment of the fetal tolerance to decreased umbilical blood flow should include an assessment of the entire clinical situation with the FHR response. Clinical interventions can then be made based on those assessments in collaboration with other team members.
- **Umbilical blood flow can be affected by** (Goss & Torgersen, 2003; Harvey & Chez, 1997):
  - **Structural abnormalities** such as a velamentous insertion, decreased number of vessels in the cord or hematomas
  - **Cord compression** caused by mechanical conditions such as maternal/fetal position in relation to cord location, loops of cord wrapped around parts of the fetal body or cord prolapse
  - **Cord cushioning** affected by decreased amounts of amniotic fluid and/or Wharton’s jelly
Intrinsic Factors

- Fetal oxygen transport
- Fetal circulation
- Fetal nervous system
- Baroreceptors
- Chemoreceptors
- Hormones
- Fetal reserves
- Fetal homeostatic mechanisms

(Feinstein & Atterbury, 2003)

Let's now shift our attention to intrinsic factors affecting fetal oxygenation and the fetal heart rate. Intrinsic factors are those factors found within the fetal body that provide for oxygenation, growth and responses when fetal physiology is stressed. They include (Feinstein & Atterbury, 2003):

- Fetal oxygen transport
- Fetal circulation
- Fetal nervous system
- Baroreceptors
- Chemoreceptors
- Hormones
- Fetal reserves
- Fetal homeostatic mechanisms
The first intrinsic factor we will discuss affecting the fetal heart rate is the fetal nervous system. This should be familiar information, but it is always important to review as primary fetal heart regulation physiology.

The two branches of the autonomic nervous system exert opposing influences on the FHR. Parasympathetic, or vagus nerve stimulation, decreases the heart rate. Sympathetic stimulation, through nerve fibers widely distributed on the fetal myocardium, will increase the fetal heart rate.

Chemoreceptors and baroreceptors are other intrinsic factors. Chemoreceptors detect biochemical changes in the fetal blood stream, such as changes in the pH, O₂ or CO₂. Baroreceptors detect changes in the fetal blood pressure (Feinstein & Atterbury, 2003).

The parasympathetic and sympathetic branches respond to the messages sent from chemoreceptors and baroreceptors to increase or decrease the heart rate according to the message received (Harvey & Chez, 1997).

For example, fetal hypoxemia (decreased O₂ in the blood) will be detected by the chemoreceptors. A message will then be sent through the Central Nervous System (CNS) by the release of specific hormones to the sympathetic nervous system to increase the heart rate.
Let’s take a closer look at the hormones and their specific functions.

The hormones—another intrinsic influence—function as the messengers. The hormones tell each player what to do.

Examples of hormones that influence the FHR are listed on this slide. Let’s discuss the roles of these hormones.

**Catecholamines:** The release of epinephrine and norepinephrine will cause an increase in the FHR and peripheral vasoconstriction. A stimulus that will cause their release is oxygen deprivation. Release of catecholamines will cause blood to be shunted toward the vital organs; the brain, heart and adrenals in the fetus, and decrease blood flow to the periphery. For example, gastrointestinal and renal blood flow.

**Vasopressin:** This antidiuretic hormone will result in an increase in blood pressure and stabilization of the hemodynamic system.

**Renin-angiotensin:** It is released by the kidneys to produce vasoconstriction to minimize blood pressure changes in the fetus (Feinstein & Atterbury, 2003).

We know that the maturity of these systems is gestational age dependent. An immature fetus will not have the ability to respond to stressors like a mature fetus. It is thus very important to take into account the fetus’s gestational age when assessing fetal well-being from an electronic FHR tracing.
• The previous slide explained the fetal response of shunting blood toward the vital organs when there is decreased oxygenation. Because of these responses, a healthy fetus can maintain aerobic metabolism under conditions that produce transient decreases in oxygen availability. Examples of these conditions may include supine positioning, uterine contractions and/or intermittent cord compression.

• **The ability of the fetus to maintain this aerobic metabolism is accomplished by the redistribution of blood to the vital organs—the brain, heart, and adrenals, as depicted in this slide.** With the release of catecholamines, and the resulting peripheral vasoconstriction, blood is directed away from the periphery. The peripheral vasoconstriction also causes blood to be shunted toward the brain, heart and adrenals. Thus, the fetus may survive periods of decreased oxygenation without damage to its vital organs (King & Parer, 2000).
Episodes of decreased oxygenation may occur abruptly, such as in the case of placental abruption or umbilical cord prolapse. They can also occur intermittently as with uterine contractions or maternal exercise.

For example, in a fetus with a nuchal cord, initially you may see typical variable decelerations occurring with uterine contractions. However, if the uterine activity is excessive and/or if the labor is prolonged, the FHR pattern may evolve into tachycardia, with atypical variable deceleration characteristics and/or minimal/absent variability.

If episodes of decreased oxygenation occur chronically, such as with preeclampsia or smoking, or if there is chronic malnourishment of the fetus, the fetal response of shunting blood toward the vital organs will be ongoing. The result may be asymmetrical intrauterine growth restriction (IUGR) with more growth and development of the areas containing the vital organs (head and chest) and less growth and development of the abdomen and periphery. This is sometimes called brain-sparing and the newborn may look like the one pictured on the right side of the slide.

The type of insult may be reflected in the characteristics of the fetal heart rate patterns (Feinstein & Atterbury, 2003; King & Parer, 2000).
Fetal responses to hypoxemia or hypoxia can also include those that are evident outside of FHR changes. These may include:

- **Oligohydramnios**—In the absence of abnormal fetal kidney function, can be the result of chronic hypoxia and its result of distribution of blood away from nonvital organs, including the kidneys (Vintzileos & Hanley, 2000).
- **Gradual hypoxia effect**—Biophysical activities that appear first in fetal life are the last to disappear during fetal hypoxia. This means that the parameters of FHR reactivity (early third trimester) and fetal breathing movements (~21 weeks), which are the last to develop, will become compromised first with the presence of acidemia (<7.20). With advancing acidemia (>7.10<7.20), fetal body movements (~9 weeks) and fetal tone (~7.5-8.5 weeks) will decrease. And when the acidemia progresses to <7.10, fetal body movements and fetal tone will become absent (Vintzileos & Hanley, 2000). This will be further discussed in the AFHMPP Workshop.
- **IUGR** may also be apparent as a result of chronic hypoxia and the resultant redistribution of blood to the vital organs (Feinstein, et al. 2003)
- The fetal response to hypoxemia and/or hypoxia depends on the degree and duration of the insult. The tolerance of a specific fetus to varying degrees and durations of insults will depend on its fetal reserve.
Fetal Reserve

The degree of hypoxemia that the fetus can tolerate before true tissue hypoxia and acidosis occur.

(Feinstein & Atterbury, 2003)

- Fetal reserve is defined as the **degree of hypoxemia that the fetus can tolerate before true tissue hypoxia and acidosis occur** (Feinstein & Atterbury, 2003).
- You will recall from our discussion about the integrity of the placenta that a healthy well-nourished and well-oxygenated fetus is supplied with approximately two times the resources it needs to thrive and grow.
- The tissue requirement of the fetus is typically less than the resources delivered. This reserve allows the fetus to withstand temporary changes in oxygen supply that are common during labor. When O₂ supply is decreased, preferential blood flow to the vital organs can allow the fetus to compensate for periods of transient hypoxemia, such as commonly occur during labor. This process of preferential blood distribution can also occur in a chronic state of decreased oxygen and/or nutrition to the fetus. This allows growth and development of the vital organs, such as the brain, heart, and adrenals. These fetuses, though, may show an intolerance to stress, such as labor, because of inadequate fetal reserves.
**Fetal Reserve (cont.)**

<table>
<thead>
<tr>
<th>Present Fetal Reserve</th>
<th>Decreased Fetal Reserve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal baseline range</td>
<td>Abnormal baseline range</td>
</tr>
<tr>
<td>Accelerations</td>
<td>No accelerations</td>
</tr>
<tr>
<td>Moderate variability</td>
<td>Minimal/absent variability</td>
</tr>
<tr>
<td>No decelerations</td>
<td>Decelerations</td>
</tr>
</tbody>
</table>

- Think of fetal reserves as a gas tank. A healthy fetus will come to labor with a full tank of gas, on which it can rely to meet its needs during labor. A fetus that has been chronically stressed may not have enough gas in its tank to rely on during labor. Then again, some fetuses will not have any gas because they have had to use all of their supplies of oxygen and/or nutrients. Or, they may not have been supplied with the extra resources that they usually need.
- You may see fetal heart rate characteristics that indicate the presence or absence of fetal reserve. **What patterns would indicate the presence of fetal reserve (or a full tank of gas)?**
  - A normal baseline range, moderate variability, no decelerations and, certainly, accelerations
- **What patterns could indicate a possibly diminished fetal reserve (or an empty tank of gas)?**
  - Minimal or absent variability, late decelerations, absence of accelerations
  - One of our continual assessments during labor is that of the presence/absence of fetal reserve. How much gas does this fetus have?

Let’s briefly review the tools of FHR monitoring that we can use to help us assess the presence or absence of fetal reserve.
It is necessary in the process of fetal heart monitoring interpretation that the practitioner know the different techniques available to assess the FHR and uterine activity (UA).

These are listed on the slide:

**The FHR can be assessed by a fetoscope, an ultrasound transducer or a fetal spiral electrode.**

**Uterine activity can be assessed by palpation, a tocotransducer or an intrauterine pressure catheter.**

- It is also important to know how each method works and its benefits and limitations. A thorough review is in the FHMPP textbook.
- Clinical decisions can be made regarding the method of monitoring according to the information that is needed in a particular clinical situation.
- When deciding on whether direct, or more invasive techniques are necessary, you should consider whether the added information received is worth the added risk of introducing an internal device.
- It is also necessary to know troubleshooting techniques for each device in case problems occur during monitoring.
Assessment and Interpretation

<table>
<thead>
<tr>
<th>Fetal Heart Rate</th>
<th>Uterine Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Baseline</td>
<td>• Frequency</td>
</tr>
<tr>
<td>• Variability</td>
<td>• Duration</td>
</tr>
<tr>
<td>• Periodic/Episodic changes</td>
<td>• Intensity</td>
</tr>
<tr>
<td></td>
<td>• Resting tone</td>
</tr>
</tbody>
</table>

• Again, since you are experienced in FHR tracing interpretation, our discussion of FHR patterns and uterine activity assessment will be brief. More information can be found in the FHMPP textbook.

• FHR assessment includes an evaluation of:
  • Baseline
  • Variability
  • Periodic/Nonperiodic changes

• Uterine activity assessment includes an evaluation of:
  • Frequency
  • Duration
  • Intensity
  • Resting tone

• When assessing uterine activity, you should consider what effect the UA has on the FHR. It is also essential, in each clinical situation, to consider whether the UA is appropriate or excessive.

• With each assessment, a decision is made whether the FHR and uterine activity interpretation, along with its application to the entire clinical picture, is reassuring or nonreassuring. Then interventions can be made according to the applicable physiologic goals. Let’s first talk some more about your decisions based on assessments.
Fetal Homeostatic Mechanisms

Reassuring (Favorable physiologic response)  Nonreassuring (Unfavorable physiologic response)

Nonhypoxic Reflex Response  Compensatory Response to Hypoxemia  Impending Decompensation

Note: This diagram is not intended to be all inclusive. All patterns must be treated with interventions that are based upon suspected underlying physiologic causes and evaluation of individual patient presentation.

Fetal status frequently changes according to its intrinsic response to extrinsic factors. Ultimately, you will decide whether you are reassured or not by information you have gathered related to maternal-fetal status. The illustration seen on this slide represents the changes that may occur with their possible resulting FHR pattern.

• You may see a nonhypoxic reflex response, such as an acceleration. This is a favorable, or reassuring, response. The nurse could interpret this type of tracing, taking into account the entire clinical picture, as “green light.” The interpretation of this tracing is reassuring; physiologic goals are being met.

• You may also see a compensatory response to hypoxemia, such as those seen in the middle, yellow circle. A typical variable deceleration would be an example of this. Tracings that fall into this category could be called “yellow light.” Interventions should be directed to meet physiologic goals and hopefully shift the FHR pattern to “green light.”

• The tracing could also have characteristics that are included in the red circle. This fetus may be exhibiting signs of impending decompensation, such as repetitive late decelerations, and be categorized as “red light.” Interventions, again, would be made to maximize physiologic goals, but realize these fetuses may no longer be able to compensate especially if the situation cannot be corrected or if the fetus has been in the “red light” zone for an extended period of time, leading to a loss of fetal compensatory mechanisms (Feinstein, Torgersen & Atterbury, 2003).

• Notice on the slide how the arrows move between the circles and how they overlap. That is because the labor process is a very dynamic time, and FHR patterns will change according to the clinical situation.

• Some tracings may not exhibit characteristics that are easily interpreted or defined-they may not fit into a category listed in this illustration. If the tracing causes confusion, it should be investigated further in collaboration with other members of the team of care providers.

• As the tracing exhibits features that are exhibited further to the right in this illustration, the concern about adequacy of oxygenation of the fetus should increase.
Determining appropriate interventions for observed FHR patterns and clinical situations requires us to integrate the information we have gathered, including our nursing database, initial and subsequent assessment data, interpretations of FHR and UA patterns and their relating physiology. We must examine the relationship between the pieces of information and decide the appropriate interventions.

These interventions are based on four physiologic goals:

- Maximize utero-placental blood flow
- Maximize umbilical circulation
- Maximize oxygenation
- Reduce uterine activity

It is important to consider the underlying physiology when determining what goals should be targeted in a clinical situation. The goals help guide you from assessment to intervention.

Let’s take a closer look at specific interventions targeted toward these goals.
By maximizing uterine blood flow, we can increase the oxygen delivery to the placental/fetal unit. Interventions to maximize utero-placental blood flow may include those shown on this slide.

Maternal position change
Hydration
Medication
Anxiety reduction

How might changing the maternal position maximize uterine blood flow?
• By avoiding the maternal supine position, you will prevent compression of the abdominal aorta and vena cava so cardiac return and cardiac output will be maximized.
• Hydration will potentially maximize intravascular volume.
• Medications, such as a tocolytic, will reduce uterine activity, thus enhancing utero-placental blood flow. A reduction or discontinuance of uterotonic medications may have a similar effect.
• Anxiety and/or pain reduction measures may reduce the release of catecholamines which cause blood to be shunted away from the uterus.
• What FHR characteristics might indicate the need to address this goal?
  Absent or minimal variability; late decelerations, prolonged decelerations
Maximize Umbilical Circulation

- Maternal position change
- Elevation of presenting part
- Amnioinfusion

What type of FHR pattern may indicate a need to address this goal?
Variable decelerations

What are some interventions that may maximize umbilical blood flow?
- A change in the mother’s position may correct cord compression.
- Elevation of the presenting part in the situation of a cord prolapse may minimize cord compression.
- Amnioinfusion can reestablish fluid volume in the uterus, thus, potentially creating a fluid cushion for the cord. This may also dilute meconium in an effort to prevent meconium aspiration.
Maximize Oxygenation

- Maternal position change
- Maternal supplemental oxygen
- Maternal breathing techniques
- Correct or treat underlying disease

What are some ways we can maximize oxygenation of the fetus?

- A lateral maternal position that enhances utero-placental perfusion will maximize oxygen delivery to the fetus.
- With the application of maternal supplemental oxygen, we can maximize the oxygen delivery to the placenta by increasing maternal oxygen content.
- Maternal breathing techniques can be implemented to prevent hyperventilation and/or decrease maternal anxiety.
- Correct or treat underlying disease. What are some examples of diseases that might affect maternal or fetal oxygenation?

  Anemia, cardiac structural defects, infection, pulmonary edema, hypertension.

  Depending on the disease, the treatment may affect differing components of maternal or fetal oxygen transport. For example, treating infection will potentially decrease maternal and fetal oxygen consumption. Treatment for maternal anemia may improve ability of the mother’s hemoglobin’s to carry oxygen. Maternal or fetal transfusion of blood products may also increase maternal or fetal SaO₂. Treatment of a maternal respiratory disease may affect all components of maternal oxygen transport.

  What component is targeted in the treatment of maternal hypertension?

  Oxygen delivery to the tissues may improve with decreased vasoconstriction, allowing increased utero-placental blood flow.
Our last goal addresses a reduction in uterine activity. Certainly, this goal would need to be addressed if the uterine activity is excessive. And, conversely, it would not be significant if the mother is not contracting. No matter what the uterine activity, it is necessary to assess the fetal tolerance to contractions. Even very minimal activity may not be tolerated by a fetus who may be compromised—this goal may even need to be addressed if the uterine activity is less than “adequate,” as with commonly described parameters for “active labor.”

Interventions to reduce uterine activity may include:

- Maternal position change
- Reduce/discontinue uterotonic drugs
- Hydration
- Modified pushing
- Tocolytic medication

We have addressed all of these except the fourth one: modified pushing.

How can maternal pushing efforts be modified to address uterine activity?

Techniques such as delayed and nondirected pushing, open glottis pushing, and upright positioning can enhance maternal pushing efforts and/or fetal descent and decrease the time necessitated for maternal active pushing (AWHONN, 2000).

Be familiar with AWHONN’s Evidence-Based Clinical Practice Guideline: Nursing Management of the Second Stage of Labor.
FHM is just one tool used to assess the maternal-fetal status. Traditionally, methods to assess fetal oxygenation have been achieved indirectly. These include those listed in this slide (Cypher & Adelsperger, 2003):

- **FHM**–including intermittent auscultation and palpation and electronic fetal monitoring
- **Scalp Stimulation**
- **Vibroacoustic stimulation**
- **Fetal movement assessment**
- **Fetal pulse oximetry**

These methods are based on agreement among experts that FHR accelerations usually indicate that a fetus is nonacidotic. **Fetal movement assessment** is based on the premise that the presence of fetal movement is a reassuring sign.

**Fetal pulse oximetry** produces a real-time assessment of fetal oxygen saturation.
Direct methods of fetal oxygenation involve actual blood sampling. The two most commonly used methods are:

- **Fetal scalp sampling** and
- **Umbilical cord blood sampling.**

These two methods are discussed in length in the AWHONN FHMPP workshop, and, since you are experienced practitioners, our discussion here will be brief.

- Fetal scalp sampling is not presently utilized often in practice. It involves obtaining a sample of blood from the fetal scalp, testing its pH and watching the trend from multiple samples. Some reasons for its decline in use include its high cost, the difficulty in performing the procedure and the difficulty in obtaining a pure specimen.
- Umbilical cord blood sampling involves obtaining blood from the umbilical vessels and performing a blood gas analysis. Cord blood gas measurements provide an objective assessment of fetal status at delivery (Cypher & Adelsperger, 2003).
- A detailed description of these procedures and their interpretation is found in the AWHONN FHMPP textbook if you need more information.

**Review Chapter 8 in the FHMPP textbook for further information.**
This concludes the review of information in the study guide. Additional information can be found in the AWHONN FHMPP textbook (specifically Chapters 2 and 3). To enhance your review and preparation for the advanced course additional information can also be obtained from the sources included in the reference list.