A Systematic Approach to the Physiologic Adaptations of Pregnancy

Col Keiko L. Torgersen, USAF, NC, BSN, MS, RNC;
Carol A. Curran, MS, RNC, OGNP

When a woman learns that she is pregnant, her emotions are like a roller coaster. To her, she is pregnant. She begins to plan all the things that could be and is in constant motion to await the 9 months until the arrival of her bundle of joy. However, to those of us in the perinatal nursing field, it means so much more. The pregnant woman’s body goes through some profound anatomical, physiologic, and biochemical changes to adapt to and support the entire pregnancy, which ultimately support the growing fetus. Although these physiologic changes are normal, often they can be misinterpreted as disease. These changes may also unmask or worsen a preexisting condition or disease, ultimately because the pregnant woman’s body cannot adequately adapt to the changes of pregnancy. It is essential to know and understand the physiology—the inner workings—of both the mother and the fetus. This includes the basic adaptations related to pregnancy, placental physiology and action, uterine activity physiology, and fetal heart rate regulation, although this article will focus on maternal and uterine physiology only. Key words: antepartum, gestation, physiology, pregnancy

Pregnancy creates profound anatomical, physiologic, and biochemical changes to support growth and development of the fetus. Changes begin soon after fertilization and continue throughout gestation. Most of these remarkable adaptations occur in response to physiologic stimuli provided by the fetus, as well as significant hormonal alterations. Although physiologic alterations are a part of gestational evolution, they often can be misinterpreted as disease or compromise. In contrast, these changes may unmask or aggravate a preexisting condition or disease. Pregnancy adaptations may positively or negatively affect the perfusion pressure across the placental bed, affecting the fetal compartment. The skill and expertise of the clinician assists to distinguish between normal and abnormal physiologic alterations of pregnancy. It is essential for all clinicians who care for the pregnant population to understand the basic physiologic adaptations related to pregnancy as they apply to each system of the human body. Obstetrical physical assessment includes the interaction between both the maternal host and the fetus. This article will focus on alterations of the maternal hematologic, cardiovascular, respiratory, and renal systems with accompanying uterine physiology.

Pregnancy is a dynamic process that results in dramatic changes in maternal anatomy, physiology, and metabolism. These changes are required to increase maternal cardiac output and to maintain uteroplacental perfusion and fetal demands. Hemodynamic changes occur in blood volume, cardiac output, systemic blood pressure (BP), pulmonary vascular resistance, heart rate, and blood flow distribution. Overall, pregnancy is considered to be hyperdynamic,
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Table 1. General adaptations of pregnancy

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperdynamic</td>
<td>An increased workload on the heart plus an increased metabolic rate leads to an increase in maternal oxygen consumption. Underlying cardiac dysfunction may be masked, especially in early pregnancy (&lt;30 wk). Cardiac intolerance to pregnancy is typically evident during the second trimester.</td>
</tr>
<tr>
<td>Hypermetabolic</td>
<td>The increase in oxygen consumption secondary to an increase in metabolic rate, fetal demands, and labor and birth lends to an increased metabolic state and risk of decompensation. Decompensation may be evident by labored breathing (dyspnea), venous engorgement, and/or edema. As a result, there may be less oxygen available for the fetus.</td>
</tr>
<tr>
<td>Hypervolemic</td>
<td>A rise in blood volume occurs secondary to the 2–5 L increase in plasma volume. Pathologic alterations may negatively influence the perfusion needs of pregnancy and blood loss at the time of delivery. Normal adaptations of pregnancy may lead to a false sense of normalcy during a state of blood loss or hemorrhage. A pregnant woman may lose up to 35% (approximately 2500 mL) of her blood volume before showing signs of hypovolemia.</td>
</tr>
<tr>
<td>Hypercoaguable</td>
<td>Pregnancy promotes procoagulant activity (ie, a cause to clot) and decreased fibrinolytic activity (ie, a decreased ability to dissolve a clot) in order to compensate for blood loss at delivery. Pregnancy itself or a history of a clotting disorder (previous deep vein thrombosis, pulmonary embolism, Factor V Leiden disorder, or antiphospholipid antibody syndrome) may increase risk of disseminated intravascular coagulopathy.</td>
</tr>
<tr>
<td>Low resistance</td>
<td>Gestation is a state of maximum venous dilation (ie, it cannot dilate anymore). This adaptation occurs secondary to volume expansion and promotes blood flow to the uteroplacental bed. As a result, maternal vital signs should show evidence of a decrease in systemic blood pressure (particularly diastolic), mean arterial blood pressure, systemic vascular resistance, and pulmonary vascular resistance. A wide pulse pressure is a normal, and healthy, finding during pregnancy.</td>
</tr>
<tr>
<td>Compensatory</td>
<td>Pregnancy is a state of alkalemia, whereas the fetal compartment promotes acidemia, per human adult standards. An increased respiratory rate (expulsion of carbon dioxide buildup) and an increased renal excretion rate of sodium bicarbonate promotes this respiratory state. In contrast, the mother is less tolerant of hypoventilation states (ie, oversedation, magnesium sulfate therapy, respiratory disease, or depression) or periods of apnea. Supplemental oxygen should be utilized early during maternal decompensation. The mother is the oxygen tank for the fetus and the placenta is the valve.</td>
</tr>
<tr>
<td>Respiratory</td>
<td></td>
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<tr>
<td>Alkalemic</td>
<td></td>
</tr>
<tr>
<td>Diabetogenic</td>
<td>Placental hormones induce a state of increased insulin resistance. As the placenta matures it supplements the function of the pancreas as maternal insulin requirements increase. As a result, if the pregnant patient has a history of diabetes prior to pregnancy, an increase in total insulin requirements may be evident by the second trimester.</td>
</tr>
</tbody>
</table>

Hypermetabolic, hypervolemic, hypercoaguable, a “low resistance” and compensatory respiratory alkalemic state, and diabetogenic. See Table 1 for an outline of each condition.

HEMATOLOGIC SYSTEM

Blood volume adaptations

Adaptations to the hematologic system are critical to ensure maternal tolerance to blood
loss following placental separation at delivery. The average blood loss for a vaginal delivery is 500 to 600 mL and is 800 to 1200 mL for a cesarean or multiple gestation delivery. Significant changes in blood volume (ie, plasma volume plus red blood cells) and cardiac status are essential to support maternal and fetal circulation. Maternal blood volume begins to increase by 6 weeks' gestation and peaks at approximately 25% to 50% by 32 weeks' gestation. The increase in blood volume is essential to support the uterus, fetus, placenta, and maternal tissues with adequate perfusion and oxygenation. These alterations promote a stable temperature secondary to vasodilation of subcutaneous vessels, maintain adequate BP, and protect the mother from hypovolemic episodes.1–4 The increase in blood volume correlates closely with fetal weight, which confirms the placental influence as an arteriovenous shunt from the maternal circulation.1 Pregnancy is considered a “high flow, low resistance” state. Increases in cardiac output are accompanied by decreases in systemic vascular resistance (SVR). This inverse relationship is mandatory to maintain hematologic and cardiovascular stability. The maternal cardiovascular system accommodates an increase of 1600 mL3 in blood volume for a singleton pregnancy (1 fetus) or 2000 to 2500 mL3 for a multiple pregnancy (2 or more fetuses). However, not all pregnant women will experience this change. Hypertensive disorders of pregnancy or preexisting cardiovascular disease may not precipitate the same degree of hypervolemia and volume expansion. These states may promote hemodilution and vasoconstriction, decreasing maternal tolerance to peripartum blood loss.5

Table 2 offers an overview of the adaptations in the hematologic system.

Maternal positioning significantly influences fluctuations in fluid distribution. During supine positioning, the cumulative weight of the uterus, placenta, amniotic fluid, and fetus promotes compression of the inferior vena cava. In the extreme, this condition (vena caval syndrome) may limit central blood volume to its lowest level because of compression of the vena cava, preventing blood from flowing back to the heart, thus creating a pooling of blood in the lower extremities and the pelvis. Perfusion decreases are enhanced after 32 weeks’ gestation and precipitates after the mother lies on her back for a minimum of 1 hour.1,6 The pooling of blood shifts the hydrostatic pressure of the vessels and pushes fluid into the interstitial tissues, resulting in edema of the feet and ankles. As soon as the patient changes to a lateral or semi-Fowler’s position with her legs elevated, the fluid redistribution will correct itself. Maternal supine positioning at term may impede blood flow and oxygen to the fetus and ultimately lead to nonreassuring fetal heart rate patterns.

**Plasma component adaptations**

Plasma component (plasma proteins, electrolytes, serum iron, enzymes, and lipids) adaptations of pregnancy are outlined in Table 3. Characteristics are altered to promote the free flow of fluid to meet maternal and fetal demands of pregnancy and any unforeseen compensatory events. Hydrostatic and oncotic pressure influences the movement of fluid between the intravascular and interstitial space. **Hydrostatic pressure** is the pressure that is exerted by the volume of fluid within a given space (ie, volume of fluid contained within a vessel). Within capillary walls, this pressure is a “positive” pressure and tends to force, or push, fluid out of the vessels into the tissue (interstitial) space. **Oncotic pressure**, specifically colloid (meaning protein) osmotic pressure (COP), is the force generated by the attraction of protein macromolecules across a semipermeable capillary membrane. Colloids attract plasma, thus impacting the hydrostatic pressure. The major colloid (protein) involved is albumin, which accounts for 75% of the total COP.

Essentially, COP facilitates immediate availability of plasma within the intravascular space to ensure an adequate BP for cardiovascular stability. Normal COP values are known to decrease in pregnancy, as previously
### Table 2. Hematologic system—Blood volume adaptations

<table>
<thead>
<tr>
<th>Component</th>
<th>Adaptation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma volume</td>
<td>- Accounts for 75% of total blood volume&lt;br&gt;- Represents a 40%–50% (1250 mL) increase over nonpregnant state&lt;br&gt;- Increases by 6 wks' gestation&lt;br&gt;- Increases steadily to 28–30 wks' gestation then plateaus&lt;br&gt;- Influenced by reproductive hormones, blood flow in the uteroplacental vessels, changes in the renal system (discussed later in this article), and fluid and electrolyte homeostasis</td>
</tr>
</tbody>
</table>
| Red blood cell (RBC) volume      | - Accounts for a 25%–30% (250–400 mL) increase over nonpregnant state<br>- Increases by 10 wks' gestation<br>- Increases progressively until term gestation<br>- Increase is due to increased circulating erythropoietin and accelerated RBC production<br>- The increase is slower and later than that in plasma volume, resulting in more plasma volume than RBC volume; referred to as dilutional physiologic anemia<br>  
  - Happens because as the volume increases, the concentration decreases (also known as hemodilution)<br>  
  - Laboratory values reveal a decrease in hematocrit (volume of RBCs) and hemoglobin (iron-containing pigment of the RBC-carrying oxygen)<br>  
  - Reaches its lowest point at 30–34 wks' gestation<br>  
  - Iron supplement will not prevent this type of anemia; however, it does produce higher hemoglobin levels in the third trimester than would be possible without iron supplementation<br>- The hemodilution decreases the viscosity (stickiness or gumminess) of the blood by 20% in the first and second trimesters |
| White blood cell (WBC) volume    | - Volume increases begin in the first trimester and plateaus during the second and third trimesters<br>- Norms range from 5000 to 12,000/mm³; values as high as 15,000/mm³ have been reported<br>- Increase because of an elevation in mature leukocytes (neutrophilia)<br>- Slight increase in eosinophils (these destroy parasitic organisms; play major role in allergic reactions; make up 1%–3% of WBCs)<br>- Slight decrease in basophils (essential to nonspecific immune response to inflammation; role in releasing histamine and other chemicals to dilate blood vessels; make up <1% of all leukocytes)<br>- No changes in monocytes (the first line of defense in the inflammation process; circulate for 24 h then move into the tissues and morph to macrophages) or total lymphocytes (responsible for majority of the immune response; found in lymph nodes, spleen, and other lymphoid organs; fewer than 1% in circulating blood)<br>- Decrease slightly because of hemodilution<br>- Increase in platelet aggregation (ie, clumping) during the last 8 wk of pregnancy<br>- Norms range from 150,000 to 400,000/mm³<br>- Women with hypertensive disorders of pregnancy, including HELLP syndrome, can develop thrombocytopenia (decrease in platelets) because of the breaking down of RBCs and platelet aggregation. Platelet counts <100,000/mm³ increase the mother’s risk of developing bleeding disorders such as disseminated intravascular coagulation<br>- Platelet counts <50,000/mm³ can lead to spontaneous hemorrhage; typically requires infusion of platelets prior to operative procedures |
| Platelets                        | - Decrease slightly because of hemodilution<br>- Increase in platelet aggregation (ie, clumping) during the last 8 wk of pregnancy<br>- Norms range from 150,000 to 400,000/mm³<br>- Women with hypertensive disorders of pregnancy, including HELLP syndrome, can develop thrombocytopenia (decrease in platelets) because of the breaking down of RBCs and platelet aggregation. Platelet counts <100,000/mm³ increase the mother’s risk of developing bleeding disorders such as disseminated intravascular coagulation<br>- Platelet counts <50,000/mm³ can lead to spontaneous hemorrhage; typically requires infusion of platelets prior to operative procedures |
Table 3. Hematologic system—Plasma component adaptations 1-4, 8

<table>
<thead>
<tr>
<th>Total plasma proteins decrease 10%-14%; change occurs mainly in the first trimester</th>
<th>Decrease in albumin concentration because of increased blood volume and hemodilution</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>/ Decrease in albumin causes decrease in colloid osmotic (oncotic) pressure, which ultimately leads to decrease in edema formation</td>
</tr>
<tr>
<td></td>
<td>/ Alters binding of calcium; drugs, and anesthesia</td>
</tr>
<tr>
<td></td>
<td>/ Edema formation in pregnancy because of alterations in hydrostatic and oncotic pressure changes</td>
</tr>
<tr>
<td></td>
<td>/ Globulin adaptations</td>
</tr>
<tr>
<td></td>
<td>/ (alpha) and (beta) globulins increase; facilitates transport of carbohydrates and lipids to placenta and fetus</td>
</tr>
<tr>
<td></td>
<td>/ (gamma) globulins decrease; facilitates transport of IgG to placenta and fetus</td>
</tr>
<tr>
<td></td>
<td>/ Causes decrease in erythrocyte sedimentation rate</td>
</tr>
<tr>
<td></td>
<td>/ Fibrinogen increases 50%-80%</td>
</tr>
<tr>
<td></td>
<td>/ Alters homeostasis</td>
</tr>
<tr>
<td></td>
<td>/ Causes decrease in erythrocyte sedimentation rate</td>
</tr>
<tr>
<td>Electrolytes (anions, cations, buffer base)</td>
<td>Decrease plasma osmolarity</td>
</tr>
<tr>
<td></td>
<td>Changes due to hypervolemia and hyperventilation</td>
</tr>
<tr>
<td>Iron adaptations</td>
<td>Serum ferritin decreases 30% up to 30-32 wks' gestation; evident in decreased iron stores during pregnancy</td>
</tr>
<tr>
<td></td>
<td>/ Iron-phosphorous protein</td>
</tr>
<tr>
<td></td>
<td>/ Iron stored in tissues in this form</td>
</tr>
<tr>
<td></td>
<td>/ Found in liver, spleen, and bone marrow</td>
</tr>
<tr>
<td></td>
<td>/ Good indicator of iron stores in the tissues</td>
</tr>
<tr>
<td></td>
<td>/ Initial decrease due to maternal utilization of stores to increase her red blood cell production; secondary decrease due to increased fetal utilization of maternal iron stores</td>
</tr>
<tr>
<td></td>
<td>/ Transferrin (globulin that binds and transports iron) increases 70%; facilitates iron absorption and transport</td>
</tr>
<tr>
<td></td>
<td>/ Iron-binding capacity decreases 15%</td>
</tr>
<tr>
<td>Total serum lipids increase 40%-60%</td>
<td>Cholesterol increases 40%; essential precursor for estrogen and progesterone</td>
</tr>
<tr>
<td></td>
<td>Phospholipids increase 37%; essential for maternal and fetal cell growth</td>
</tr>
</tbody>
</table>

mentioned, and reach their lowest point at 35 to 36 weeks' gestation. COP values continue to decrease in the intrapartum period, with a nadir during the first 24 hours postpartum. 9(p59) Table 4 compares normal COP values during the pregnant and nonpregnant states.

Where protein goes, water follows. Pregnancy is a state of decreased serum albumin. This predisposes the pregnant population to leaky capillaries and pooling. Pregnant patients with underlying medical complications, such as a hypertensive disorder, have an even greater risk of peripheral or central edema. As protein evacuates the vessels, plasma follows, increasing the patient’s risk of peripheral edema, pulmonary edema, and, in extreme cases, cerebral edema. Women experiencing preeclampsia, or other hypertensive disorders of pregnancy, are at the greatest risk for developing pulmonary edema because of influences of the disease on COP
Table 4. Normal colloid osmotic pressure values

<table>
<thead>
<tr>
<th></th>
<th>Normal COP values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonpregnant</td>
<td>25.4 ± 2.3 mm Hg</td>
</tr>
<tr>
<td>Antepartum</td>
<td>22.4 ± 0.5 mm Hg</td>
</tr>
<tr>
<td>Intrapartum</td>
<td>19.4 ± 2.5 mm Hg</td>
</tr>
<tr>
<td>Postpartum</td>
<td>15.4 ± 2.1 mm Hg</td>
</tr>
<tr>
<td>Antepartum with</td>
<td>17.9 ± 0.7 mm Hg</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td></td>
</tr>
<tr>
<td>Postpartum with</td>
<td>13.7 ± 0.5 mm Hg</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td></td>
</tr>
</tbody>
</table>

supplemented by the adaptations of pregnancy. Thus, it is important to understand the multifactorial influences pregnancy, disease, and labor have on COP. In the pregnant population, COP will decrease by 2 to 3 mm Hg for every 1 L of crystalloid fluids infused. As COP decreases, the risk of edema increases, making resuscitation of mother difficult, if resuscitation is needed.

Coagulation adaptations

Both coagulation and anticoagulation properties are altered during pregnancy. A pregnant woman is at increased risk for thrombosis (clot formation) and consumptive coagulopathies (disorders that use up all the platelet or coagulation components), such as disseminated intravascular coagulopathy. Table 5 reviews the adaptations in coagulation during gestation. Changes occurring in coagulation factors are seen in the activated partial thromboplastin time and the prothrombin time. The former is the total time needed for a clot to form after adding activating factor, calcium, and phospholipid mix to a blood sample. In pregnancy, a prolonged (> 40 seconds) activated partial thromboplastin time indicates a clotting abnormality. The inability to clot in a timely fashion raises concerns in all patients. Prothrombin time is the total time needed for a clot to form usually after adding calcium and thromboplastin to a blood sample. In pregnancy, the prothrombin time is used to assess unexplained bleeding or the ability of the liver to synthesize blood clotting proteins.

Both of these values decrease during pregnancy. Anticoagulants are also affected by pregnancy. While protein C and antithrombin remain unchanged, protein S can decrease by as much as 40%. Another increase is seen in the thrombin-antithrombin complexes. These increases are a result of increases in the buildup of thrombin.1–3 Fibrinolytic (breakdown of clots) activity also increases, but will decrease during the third trimester. This decrease allows clots to form at the site of placental separation, with approximately 5% to 10% of total body fibrin relocating to this site at delivery.1

Adaptations to the hematologic system are critical. As a result, the pregnant woman’s ability to clot is enhanced. These changes are controlled with the increase in plasminogen and the decrease in tissue plasmin inhibitors. Therefore, a balance exists to increase blood flow during antepartum and to limit blood loss at the time of delivery. Pregnant patients are at an increased risk for thrombosis of all types (ie, pulmonary embolus, deep vein thrombosis) because of gestational adaptations. Any scenario or disease that may further precipitate significant maternal hypovolemia (ie, placental abruption, placental previa, trauma, uterine rupture, severe preeclampsia, HELLP syndrome, prolonged intrauterine fetal demise, postpartum hemorrhage) enhances the occurrence of disseminated intravascular coagulation in the pregnant population.

CARDIOVASCULAR SYSTEM ADAPTATIONS

The cardiovascular system of the gravida must meet the demands of both the mother and the fetus. Therefore, cardiovascular adaptations are profound. These changes are mediated by reproductive hormones like estrogen and progesterone and are easily reversible once the pregnancy terminates. The function of the cardiovascular system is to transport oxygen to cells and remove carbon dioxide from the lungs in accordance with the metabolic rate.
Table 5. Hematologic system—Coagulation adaptations

<table>
<thead>
<tr>
<th>Increases occur in the following clotting factors</th>
<th>Decreases occur in the following clotting factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Factor I (fibrinogen)</td>
<td>● Factor XI</td>
</tr>
<tr>
<td>○ 50% increase</td>
<td>○ Plasma thromboplastin antecedent</td>
</tr>
<tr>
<td>○ Precursor to fibrin</td>
<td>○ Responsible as contact factor for tissue factor thromboplastin</td>
</tr>
<tr>
<td>● Factor II (prothrombin)</td>
<td>● Factor XIII</td>
</tr>
<tr>
<td>○ Precursor to thrombin formation</td>
<td>○ Fibrin-stabilizing factor</td>
</tr>
<tr>
<td>○ Increase is slight, or none at all, when compared to that in other factors</td>
<td>○ Responsible for maintaining the fibrin clot</td>
</tr>
<tr>
<td>● Factor V</td>
<td></td>
</tr>
<tr>
<td>○ Is a plasma accelerator globulin</td>
<td></td>
</tr>
<tr>
<td>○ Accelerates the conversion of prothrombin to thrombin</td>
<td></td>
</tr>
<tr>
<td>● Factor VII</td>
<td></td>
</tr>
<tr>
<td>○ Proconvertin</td>
<td></td>
</tr>
<tr>
<td>○ Reacts with Factor III and calcium to activate vitamin K (Factor X)</td>
<td></td>
</tr>
<tr>
<td>● Factor VIII (cryoprecipitate)</td>
<td></td>
</tr>
<tr>
<td>○ Antihemophilic globulin</td>
<td></td>
</tr>
<tr>
<td>○ Often used for women with von Willebrand’s disease in which they lack Factor VIII</td>
<td></td>
</tr>
<tr>
<td>● Factor IX</td>
<td></td>
</tr>
<tr>
<td>○ Plasma thromboplastin component</td>
<td></td>
</tr>
<tr>
<td>○ Reacts with Factor VIII, calcium, and phospholipid to activate vitamin K (Factor X)</td>
<td></td>
</tr>
<tr>
<td>● Factor X (Vitamin K)</td>
<td></td>
</tr>
<tr>
<td>○ Known as Stuart-Prower factor</td>
<td></td>
</tr>
<tr>
<td>○ Accelerates the conversion of prothrombin to thrombin</td>
<td></td>
</tr>
<tr>
<td>● Factor XII</td>
<td></td>
</tr>
<tr>
<td>○ Known as Hageman factor</td>
<td></td>
</tr>
<tr>
<td>○ Responsible as contact factor necessary for the initiation of the clotting cascade</td>
<td></td>
</tr>
</tbody>
</table>

The cardiovascular circuit is composed of the following:

- **Heart:** Pump that provides the force that drives blood through the vascular system
- **Arteries:**
  - Delivery system that distributes cardiac output throughout the body
  - Regulates the volume of flow delivered to each organ system on a moment-to-moment basis, depending on the regional metabolic need
- **Capillaries:**
  - Exchange system (microscopic vessels where the actual exchange of respiratory gases [Oxygen, Carbon dioxide, etc], nutrients, and metabolites occurs between the plasma and the body cells)
  - Referred to as “nutrient bed”
- **Veins:**
  - Return system that brings deoxygenated blood back to the heart and lungs
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- Acts as a reservoir and accommodates approximately 70% of circulating blood volume
- Through veins constricting (vasoconstriction) or dilating (vasodilation), the venous blood volume may increase or decrease according to the needs of the cardiovascular system

**Blood:**
- Liquid medium where respiratory gases, nutrients, metabolic wastes, and hormones are dissolved
- Formed elements (red blood cells, white blood cells, platelets) are carried

Under normal conditions and with every heartbeat, equal amounts of blood pass through all areas of the cardiovascular circuit. The cardiovascular system is a closed and continuous circuit. If a disturbance of flow occurs in one area of the system, a subsequent disturbance of flow will occur in other portions of the system.

During pregnancy, the heart changes its position, appearance, and function. As the uterus grows and expands out of the pelvis, the diaphragm is pushed upward, along with the abdominal contents. This action displaces the heart upward, forward, and to the left side. The apex (pointed end of the heart) is rotated laterally (to the side). Therefore, the point of maximum impulse is located slightly more left lateral in the pregnant population. In addition, the left ventricular muscle size is increased, causing the heart to enlarge, especially during the second and third trimesters.

At approximately 4 to 5 weeks' gestation, the maternal heart rate begins to increase and peaks in the third trimester, with a 15% increase over the heart rate in the nonpregnant population (usually 60–80 beats per minute). This change equates to an increase of 15 to 20 beats per minute in the heart rate. During multiple gestations, the maternal heart rate can increase by as much as 40% above the rate for the nonpregnant population.

**Diastolic filling time** (time allotment for the left ventricle to fill with blood) is dependent on the heart rate. Increased heart rates lead to decreases in diastolic filling time. As a result, inadequate oxygenation and perfusion of maternal systems may result in inadequate oxygenation and perfusion of the placenta, and ultimately the fetus.

As the mother enters the third trimester, heart sounds begin to change. Approximately 90% of women have an exaggerated split of the first heart sound, with both components of the first heart sound affected (ie, mitral and tricuspid valve closures). There is also a loud, wider split of the second heart sound that usually occurs around 30 weeks' gestation and an easily heard, third heart sound. Because of the mechanical change in the heart's position, the best place to hear the heart sounds is over the left sternal border of the mother's chest, between the third and fifth intercostal space often referred to as Erb's point.

In addition to the changes in heart sounds, 90% to 95% of women develop a nonpathologic systolic murmur because of increased aortic and pulmonary artery blood flow secondary to increases in plasma volume. These murmurs are heard in early to mid-systole and are best heard along the lower left sternal border of the mother's chest in the third intercostal space. Systolic murmurs greater than a grade 2/4 or any type of late systolic, pansystolic or diastolic murmur is abnormal and requires further evaluation.

Electrocardiogram changes occur as a result of the mechanical shift in the position of the heart. Changes such as small Q wave, inversion of the P wave and the T wave, and S-T segment changes are common and considered benign. Inverted T waves may be noted in lead III during an electrocardiogram. It is also common to see arrhythmias, specifically tachyarrhythmias such as supraventricular tachycardia, secondary to the increase in size of the maternal heart. In addition, because of the cardiovascular changes, jugular vein pulsations are more readily seen with distention of the jugular vein occurring as early as 20 weeks' gestation.
Table 6. Cardiac system—Output adaptations

- Increases by 1 L/min by 8 wks’ gestation to a volume of 5–7 L/min at term
- Increases 30%–50% during pregnancy for singleton pregnancy
- Increases by an additional 15%–20% for multiple pregnancies
- Increase continues to 25–30 wks’ gestation then plateaus
- Associated with increase in venous return (blood returning to the heart) and greater right ventricular output, especially if the mother is positioned in the left lateral position
- Changes in maternal heart rate occur at 5 wks’ gestation; changes in stroke volume occur at 8 wks’ gestation
- Cardiac output is dramatically affected with maternal position change
  - Supine position can decrease cardiac output by as much as 25%–30%
  - Sitting position can decrease cardiac output by as much as 10%–15%
  - Best position for optimal cardiac output is lateral position (right or left does not matter)
- Cardiac output also affected during labor and postpartum period
  - 15% increase in latent phase of labor
  - 12%–30% increase (300–500 mL) in first stage of labor (comes from uterus and shunted into mother’s circulation)
  - Increases 45%–49% during the second stage of labor
  - Progressive increases occur with pain, fever, preterm labor, or use of sympathomimetic drugs
  - Postpartum, specifically immediately after delivery, results in 65% increase in cardiac output
- Anesthesia also affects maternal cardiac output
  - Epidural anesthesia can cause a decrease in venous return resulting in a decrease in cardiac output because of a decrease in peripheral vascular resistance
  - General anesthesia also decreases cardiac output
  - Local or paracervical anesthesia can cause an increase in cardiac output
- Normal values of cardiac output
  - Nonpregnant: 3–4 L/min
  - Pregnant, not ill: 6–7 L/min
  - Pregnant, ill: 9–11 L/min
  - Pushing: 15–18 L/min

Cardiac output is the product of heart rate and stroke volume. This is a reflection of the overall capacity of the left ventricle of the heart to maintain systemic BP and organ perfusion. Cardiac output is considered to be the most dramatic and significant hemodynamic change during pregnancy.1–3 Fluctuations in maternal cardiac output directly impact fetal cardiac output. The fetus is dependent upon a stable maternal BP. It is imperative to maintain maternal stability in order to facilitate fetal stability. Table 6 outlines cardiac output adaptations.

Stroke volume is composed of 4 factors: preload, afterload, contractility, and muscular synchrony. These affect the patient’s ability to regulate cardiac output in response to physiologic challenges. Stroke volume ultimately equals the amount of blood in milliliters the heart pumps out with each heartbeat. This is approximately 85 mL during pregnancy. Preload equals the volume of blood in the right and left ventricles. Afterload is the resistance blood meets once it is evacuated from the left or right ventricle (SVR or pulmonary vascular resistance [PVR]). PVR drops lower and longer than SVR. Contractility is basically how efficient the heart is at contracting to get the blood out of the heart. It is measured in liters per minute. It assesses the left ventricular stroke work index, ie, how hard the heart is working. Last, muscular synchrony is the synchronization of the muscles to produce the heart rate.

An easy way to remember cardiac output and its components is the mnemonic
CRAP: Contractility, Heart Rate, Afterload, and Preload. Cardiac output is the mother’s (and fetus') survival—no or poor cardiac output equals no survivability or poor outcome, for both the mother and the fetus.

CARDIOVASCULAR SYSTEM: BP AND SVR

Blood pressure

Adequate and normal BP readings reflect the heart’s ability to maintain adequate cardiac output and perfusion to the surrounding tissues. Blood pressure begins to fall during the first trimester of pregnancy, reaching its lowest point between 24 and 32 weeks' gestation, and then gradually returning to the nonpregnant levels by term. The diastolic BP decreases more dramatically than the systolic BP. Pregnancy hormones play a role in these changes. Progesterone and prostaglandins relax the walls of maternal blood vessels, thus decreasing the SVR. This change, coupled with a major portion of maternal blood flow and cardiac output directed toward support of the uteroplacental circulation, provides a basis for the decrease in maternal systemic BP. Systemic vascular resistance is lowest in the first and second trimesters, then gradually increases by term; therefore, both systolic and diastolic BPs tend to increase during the third trimester.

In addition to the progesterone and prostaglandin influence, systemic BP is also influenced by the renin-angiotensin-aldosterone system. In short, renin converts to angiotensin II, a vasopressor that helps to maintain BP. Yet, during pregnancy, the levels of renin and angiotensin II increase, but for unknown reasons, most pregnant women seem to be refractory to the effects of angiotensin (ie, has no effect on them). This means that maternal BP, during pregnancy, does not increase despite increased angiotensin II levels, as would normally occur in a nonpregnant patient. Under normal circumstances, following delivery, BP levels usually return to that of the nonpregnant state, and postpartum diuresis and equilibration of normal, nonpregnant blood volume occurs.

Blood pressure equals \( \text{Force} \times \text{Resistance} \). \( \text{Force} \) is composed of cardiac output (stroke volume + heart rate). \( \text{Resistance} \) is composed of SVR and renal VR. This concept is important to remember especially when giving your patient any type of vasoactive drug (ie, any drug that acts on the blood vessels, such as those given in antihypertensive therapy). The reason for this is that your first-line antihypertensive drugs will target resistance and not force. Therefore, the resistance in the vessels may be decreased as the vessels are opened, but the work of the heart (ie, preload, afterload, and contractility) will not be affected.

Another way to assess the overall hemodynamic status, besides assessing maternal BP, is to assess the mean arterial BP. The assessment of the mean arterial BP is a better way to track BP trends than using systolic or diastolic BP alone. The mean arterial BP is determined by the following formula: Systolic BP + 2 × Diastolic BP/3.

Blood pressure measurements can also be affected by a variety of other things:

- Maternal health status
- Maternal position, especially during BP assessments
- Maternal age (systolic BP remains unchanged; however, diastolic BP increases, and continues to increase with age, especially after 35 years of age)
- Maternal parity (as parity increases, regardless of maternal age, systolic and diastolic BPs decrease; biggest difference between nulliparas [woman who had never produced a viable pregnancy] and primiparas [woman who has delivered 1 baby])
- Type of device being used to assess BP and BP cuff size

Among the most controversial issues surrounding BP assessment, the method of assessment and maternal position during evaluation appear to be of greatest concern. See Table 7 for recommended positioning for BP assessment.

Another important parameter to assess is pulse pressure (PP). Pulse pressure is not a
Table 7. Maternal positioning and blood pressure assessment

<table>
<thead>
<tr>
<th>Position</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting or standing</td>
<td>• Systolic BP shows little change</td>
</tr>
<tr>
<td></td>
<td>• Diastolic BP will typically decrease by about 5–10 mm Hg</td>
</tr>
<tr>
<td></td>
<td>• Change occurs in the first trimester and continues during the second trimester to return to prepregnancy levels by term 1,2,12</td>
</tr>
<tr>
<td>Lateral recumbent</td>
<td>• Both systolic and diastolic BP decrease</td>
</tr>
<tr>
<td></td>
<td>○ Systolic BP decreases by approximately 5–10 mm Hg</td>
</tr>
<tr>
<td></td>
<td>○ Diastolic BP decreases by about 10–15 mm Hg; reaches lowest point during second trimester; returns to prepregnancy levels by third trimester 12</td>
</tr>
<tr>
<td>Outpatient setting</td>
<td>• Semi-Fowler’s sitting position</td>
</tr>
<tr>
<td></td>
<td>• 10-minute rest period before assessment</td>
</tr>
<tr>
<td></td>
<td>• Arm position so that BP cuff is at the level of the heart</td>
</tr>
<tr>
<td>Inpatient setting</td>
<td>• Sitting, semi-Fowler’s sitting, or lateral recumbent position 13–15</td>
</tr>
<tr>
<td></td>
<td>• Arm position so that BP cuff is at the level of the heart</td>
</tr>
</tbody>
</table>

*BP indicates blood pressure. It is recommended that, whenever possible, the BP be measured in the same arm with the woman in the same position each time.1,2,12 On the basis of this recommendation, you should then include what the maternal position is and which arm was used in your documentation of the BP assessment.

concept typically assessed in the pregnant patient; however, it could be a sensitive indicator of the mother’s changing hemodynamic status. Pulse pressure reflects the acute increases or decreases in cardiac stroke volume and is almost equivalent to the stroke volume. A normal widening of the PP occurs in pregnancy secondary to hormonal changes and a drop in diastolic BP. The following is the formula for determining the PP:

\[
PP = \text{Systolic BP} - \text{Diastolic BP} = \frac{1}{2} \text{stroke volume (if multiply } PP \text{ by } 2 = \text{ stroke volume)}
\]

The normal PP is 40 to 50 mm Hg. If the pulse pressure is >50 mm Hg, it is indicative of increased stroke volume; ie, the heart may be working too hard. If the PP is <30 to 40 mm Hg, it is indicative of decreased stroke volume; ie, the heart may not be working hard enough. Either situation is not good for the mother or the fetus.

There are really 4 issues regarding methodology: (1) What sound most accurately reflects diastolic BP? (2) What is the correct size of the cuff used to measure BP? (3) What type of device should be used to measure BP? and (4) How often should you reassess the BP using the electronic devices?

**Sound**

For a number of years, there has been considerable debate on which diastolic sound to use in obtaining an accurate diastolic BP. After years of debate, the National High Blood Pressure Education Program recommended that the Korotkoff phase V sound be used as that determinate sound.15 After reviewing years of research, the National High Blood Pressure Education Program determined that the Korotkoff V sound most accurately reflected diastolic BP. The Korotkoff V sound is that point when the sound totally disappears, not just when the sound becomes muffled, which is the Korotkoff phase IV sound. If the Korotkoff IV sound is used to establish the diastolic BP, the measurement could be as much as 13 mm Hg greater than if the BP was measured with the Korotkoff V sound. Therefore, it is the disappearance of sound (Korotkoff phase V) where the diastolic BP is measured and recorded.1,14,15

**Cuff size**

Choosing the correct cuff size to measure BP will result in a more accurate reading. The cuff should comfortably encircle at least
Device

The most accurate noninvasive way to assess maternal BP is to use the “tried and true” stethoscope, manual blood pressure cuff, and the sphygmomanometer. However, with the development and overwhelming use of electronic, automated blood pressure devices, this basic technology of assessing the BP manually has been pushed out of the way. It needs to be understood that electronic devices may underestimate diastolic values by about 10 mm Hg and systolic values by about 4 to 6 mm Hg. This is due, in part, to the fact that the electronic devices were developed for the adult intensive care unit patient, one who is in a low flow (low cardiac output) and high resistive (high SVR) state, totally opposite of the pregnant woman. Electronic devices may be useful, especially if frequent BP monitoring is needed (ie, epidural insertion) or if BPs need to be trend over time (ie, hypertensive disorders of pregnancy). Despite their usefulness, you should be aware of the differences between the electronic, automated devices and the manual device. Should you ever be concerned with, or question, a BP reading obtained with the electronic device, you should confirm your findings with a manual BP reading.

Timing

As a practitioner, it is imperative to understand the maternal physiology and how it relates to monitoring the BP. Because of the amount of time it takes to open up and reperfuse the peripheral vessels, the BP should be taken no more often than every 2 to 5 minutes.

Systemic vascular resistance

Systemic vascular resistance is the measure of tension required for ejection of blood into the circulation. Simply put, it is the amount of resistance the blood meets as it leaves the heart. The amount of resistance the blood meets will depend on how open (vasodilated) or closed (vasoconstricted) the vessels are. Systemic vascular resistance is decreased during pregnancy and parallels the decrease in BP (Table 8).

RESPIRATORY SYSTEM ADAPTATIONS

Changes that occur within the respiratory system are mediated by hormonal changes, biochemical changes, and mechanical changes caused by the growing uterus. See Table 9 for mechanical changes of the respiratory system.

Biochemical changes, along with hormonal influences, are responsible for most of the changes that occur in the respiratory system (Table 10).

Lung volume is also affected with pregnancy, beginning in the second trimester and continuing until term. One of the most significant changes is the 30% to 40% (from 500 to 700 mL) increase in tidal volume (amount of air inspired/exhaled in normal breath). The other significant changes are the 20% decrease in expiratory reserve volume (amount of air expired from resting expiratory level), 20% decrease in residual volume (amount of air in lungs after maximum expiration), and 20% decrease in functional reserve capacity (amount of air remaining in the lungs after normal expiration). Oxygen consumption is also increased. With a singleton pregnancy, the increase is usually 15% to 20%, but the increase can be higher for multiple pregnancies. Increased oxygen consumption is essential to meet the almost 50% increased demand that is placed on the mother by the growing fetus. Other changes include the following:

- Total lung volume decreases by 5%
- Minute ventilation increases by 40%
- pH increases from 7.35 to 7.45 to 7.40 to 7.45
- $\text{PaO}_2$ increases from 90 to 100 mm Hg to 104 to 108 mm Hg
- $\text{PaCO}_2$ decreases from 35 to 45 mm Hg to 27 to 32 mm Hg
- $\text{HCO}_3$ decreases from 22 to 26 mm Hg to 18 to 21 mm Hg
Table 8. Systematic vascular resistance\textsuperscript{1-4,8}

- Decreases by 5 wks' gestation
- Reaches the nadir (lowest point) between 16 and 34 wk, then increases until term
- Progesterone and prostaglandins causes relaxation of the smooth muscle and vasodilation (vessels dilate)
- Decreases in systemic and renal vascular tone occur early in pregnancy and precede the changes that occur in the blood volume
- Uteroplacental circulation is a low-resistance circuit that decrease cardiac afterload (ie, reduces the resistance)
- Increased fetal heat production also contributes to decreased vascular resistance due to vasodilation of the vessels, particularly in the areas that are known for heat loss (ie, hands, feet)\textsuperscript{1}
- Other areas with increased blood flow due to decreased systematic vascular resistance:
  - Mammary blood flow
  - Coronary blood flow (due to increase in workload of the left ventricle secondary to increased cardiac output, blood volume, fetal and uterine growth, and maternal weight gain)
  - Uterine blood flow
    - Rise noticeable at 10 wks' gestation and continues to increase to term
    - Blood flow is not autoregulated meaning that the amount of oxygen available to the fetus is regulated by an increase in oxygen concentration in the blood and not by increased uterine blood flow
    - Blood vessels at maximum dilation; therefore, they do not respond to circulating pressor agents or changes/influences in the autonomic nervous system
  - Renal blood flow (discussed later in this article)
  - Epidermal (skin) blood flow
    - Slow rise up to 18–20 wks' gestation, then marked increase between 20 and 30 wks' gestation
    - Leads to increase in skin temperature, sweaty or clammy hands, vascular spider and palmar erythema, and hypertrophy of mucous membranes (ie, nasal congestion)
  - Peripheral (extremity) blood flow
    - Changes occur early in gestation and up to 6–8 wks' gestation
    - Multiple pregnancies can see changes continuing up to 30 wks' gestation
    - Affects arms and legs
  - Pulmonary vascular blood flow
    - Increase is secondary to increases in blood volume and cardiac output
    - Pulmonary vascular resistance decreased
  - Areas unaffected by decreased vascular resistance, thus no significant change in blood flow
    - Hepatic blood flow, includes liver
    - Cerebral blood flow

The oxygen-hemoglobin dissociation curve is a graphic representation of the equilibrium between oxygen and hemoglobin. The curve shows the relationship between $P_{O_2}$ and the percentage of saturated hemoglobin. The curve can shift to the left or right of normal. The shift is indicative of the increased affinity for oxygen and favors the uptake of oxygen by the hemoglobin molecule.

- Top of curve flattens out
  - Shows an increase in $P_{O_2}$
  - Little increase in hemoglobin saturation
- Bottom of curve steep
  - Shows that at low $P_{O_2}$ levels, small changes in the $P_{O_2}$ level will result in large changes in saturation of the hemoglobin
  - Fetal hemoglobin has a lower affinity for hemoglobin (readily takes up more oxygen) than do maternal hemoglobin
  - If curve is shifted to the right of normal $\Rightarrow$ oxygen molecule loosely bound
Table 9. Respiratory system mechanical changes\(^1-^4\)

- Diaphragm elevated 4 cm past its original position
  - Increases respiratory rate
  - Pregnancy changes from costal breathing to diaphragmatic breathing
- Transverse diameter expands 2–5 cm
  - Lower ribs also flare outward
  - Allows for the shifting of abdominal contents to accommodate the growing uterus
- Subcostal angle increases approximately 35 degrees
  - Secondary to relaxin effects as the rib ligaments are loosened
  - Allows for the shifting of abdominal contents to accommodate the growing uterus

Table 10. Biochemical changes\(^1-^4\)

- Progesterone levels increase and are responsible for changes in ventilation
  - Increases minute ventilation (volume of air inhaled/exhaled in 1 min)
  - Respiratory rate increased from 16 to 20 breaths/min
  - Enhances maternal response to increased carbon dioxide in the blood (hypercapnia)
- Lowers respiratory center threshold for carbon dioxide
  - Results in dyspnea (air hunger) or hyperventilation (during the second stage of labor)
  - Decreases airway resistance
  - Decreases the work of breathing
  - Increases airflow
- Prostaglandins
  - Relaxes the bronchial smooth muscles
  - Modifies respirations to accommodate fetal needs
  - Increases tidal volume (amount of air inspired/expired with each normal breath)
  - Decreases PCO\(_2\) = maternal respiratory alkalosis (promotes oxygen exchange with the fetus)
  - Acids taken out of maternal and fetal systems
  - Maintains pH on the high side of normal
- Increase oxygen availability
  - Increased oxygen consumption
  - Increased amount of circulating hemoglobin
  - Increased amount of circulation oxygen

RENAL SYSTEM ADAPTATIONS

Changes occurring in the renal system during pregnancy are a result of functional and structural adaptations. The kidneys are critical to the body’s ability to maintain homeostasis through the regulation of water and electrolyte balance. It is imperative that these
renal changes occur to support the essential changes that occur in the cardiovascular system. Refer to Table 11 for renal changes.

Proteinuria is more common in pregnant women than in nonpregnant women. A urine dipstick value of +1 is common and is not evident of resultant pathology or preeclampsia. An abnormal protein excretion rate is greater than 300 mg in 24 hours, and the patient should be evaluated for preeclampsia or other hypertensive disorders of pregnancy as well as previously undiagnosed renal disease.

Significant decreases in a patient’s SVR will affect her kidneys. This is because the blood needed to perfuse the kidneys for optimal operation is being shunted to vital organs instead of to the kidneys. The pregnant woman’s body does not recognize the kidneys as a vital organ. As a result, if blood is shunted away from the kidneys, the mother will not produce urine and will not be able to void. Therefore, it is important to assess her urine output to ensure it is not less than 30 mL/h.

UTERINE PHYSIOLOGY

To ensure adequate oxygen supply to the fetus, it is essential that the mother have a sufficient amount of hemoglobin to carry oxygen, appropriate oxygen content in her blood, and effective uterine blood flow to transport oxygen to the placenta. Conditions that adversely affect any of these steps, whether acutely over minutes, subacutely over days, or chronically over weeks, will come to bear on the fetal adaptive responses and the potential for abnormal development. Because uterine blood flow is one of the prime determinants of oxygen exchange between maternal and fetal systems, it is important to understand uterine activity physiology. The uterus supplies the basis for the support of the entire maternal-fetal unit because it is the prime method substances are passed through the placenta to the fetus. Physiologic exchange to support the growing fetus occurs through the placenta, which is essentially a union of maternal and fetal tissue. An increase in uterine blood flow is noticeable at approximately 10 weeks’ gestation. As the pregnancy approaches term, 85% of the total uterine blood flow supplies the placental circulation, whereas the additional 10% to 15% perfuses the uterus each minute (700–800 mL/min). The rate of blood flow through the uterus and placenta is relatively high and regulated by maternal BP and SVR.16

Because the uterine vascular bed is maximally dilated, blood flow is not autoregulated meaning that the amount of oxygen available to the fetus is regulated by an increase in oxygen concentration in the blood and not by an increase in uterine blood flow. In addition, the vessels do not respond to circulating pressor agents or changes/influences in the autonomic nervous system.

The uterine arteries supply uterine blood flow. These arteries pass through all 3 muscle layers of the uterus before reaching the placenta. Because they must pass through the uterine muscle, when the uterus contracts the vessels can be squeezed, causing the myometrial pressure to exceed the arterial pressure and ultimately decreasing the uterine blood flow. Myometrial pressure (ie, pressure in the uterine muscle) is measured at 10 mm Hg, whereas the mean arterial BP is 85 mm Hg. In addition to the uterine muscle, anything that decreases cardiac output will also decrease uterine blood flow.

Some factors that decrease uterine blood flow as well as placental blood flow or diffusion capability include the following:

- Maternal position
  - Supine
    - Compresses the inferior vena cava, the aortoiliac vessels, or both
    - Resultant decrease in maternal cardiac output and maternal hypotension
    - Causes uteroplacental insufficiency
    - Increased incidence of late decelerations during labor1–3,17

- Exercise
  - Excessive exercise can divert blood from the uterus to the maternal muscles
### Table 11. Renal changes

<table>
<thead>
<tr>
<th>Change</th>
<th>Description</th>
</tr>
</thead>
</table>
| **Kidneys** | lengthened by 1–1.5 cm secondary to:  
  - Increased renal blood flow  
  - Increased vascular volume (30%)  
  - Hypertrophy |
| **Renal calyces, pelvis, and ureters** | dilate:  
  - Dilated; right ureter > left ureter  
  - Ureters elongated, decreased motility  
  - Decreased peristaltic movements  
  - Results in:  
    - Increased risk of urinary tract infection  
    - Altered accuracy of 24-h urine collection |
| **Bladder** |  
  - Decreased tone  
  - Increased capacity  
  - Displaced in pregnancy  
  - Mucosa edematous and hyperemic  
  - Can hold as much as 300 mL in ureter  
    - Secondary to incompetent vesicoureteral valve  
    - Can alter 24-h urine collections  
  - Results in:  
    - Increased risk of infection  
    - Urinary frequency and incontinence  
    - Increased risk of trauma  
    - Increased risk of reflux and resultant infection |
| **Renal blood flow** | increases 35%–60% = increased filtration and excretion of water and solutes |
| **Glomerular filtration rate** |  
  - Increases 40%–50%  
  - Results in:  
    - Increased filtration and excretion of water and solutes  
    - Increased urine flow and volume  
    - Decreased serum blood urea nitrogen, creatinine, and uric acid  
    - Altered renal excretion of drugs = increased risk of subtherapeutic blood and tissue levels |
| **Renal tubular function** |  
  - Increased reabsorption of solutes  
  - Increased renal excretion of glucose, protein, amino acids, urea, uric acid, water-soluble vitamins, calcium, hydrogen ions, and phosphorus  
  - Retains sodium and water  
  - Potassium excretion is decreased  
  - Results in:  
    - Homeostasis  
    - Glycosuria, proteinuria  
    - Compensatory response for respiratory alkalosis  
    - Increased nutritional needs for calcium and water-soluble vitamins |
| **Renin-angiotensin-aldosterone system** |  
  - All components increase  
  - Resistance to pressor effects of angiotensin II  
  - Results in:  
    - Extracellular volume = homeostasis  
    - Retention of water and sodium  
    - Maintains normal blood pressure |
| **Arginine vasopressin** |  
  - Regulation of osmolarity leads to water retention  
  - Results in:  
    - Increased extracellular and plasma volume  
    - Volume homeostasis |
Can result in fetal tachycardia secondary to a sympathetic response to decreased fetal oxygen. Hypertensive disorders of pregnancy—can lead to infarcts or placental abruption. Placental abruption. Uterine rupture. Diabetes mellitus—increases thickness of placenta leading to increased diffusion distance. Postdate placenta—can lead to calcifications of placenta. Results: decreased placental surface area resulting in decreased uterine perfusion. Anesthesia—decreases intervillous space blood flow because of maternal hypotension. Uterine contractions.

Uterine contractions are produced when electrical activity passes through numerous communication sites (ie, gap junctions) between each uterine cell. At that site there is an exchange of ions inside and outside each uterine cell. This exchange leads to shortening of myometrial fibers, causing the muscular uterine wall to contract. Just like the heart has pacemakers to regulate electrical conduction, the uterus has gap junctions. The majority of these gap junctions are in the fundus (upper portion of the uterus). This is what is called “fundal dominance” and is considered the main pacemaker in the uterus. Fundal dominance aids in the descent of the fetus. When the uterus is at rest, the fetus receives most of its oxygen and nutrient needs as well as eliminates most of the excess carbon dioxide. If the fundus is not the dominant pacemaker, and another portion of the uterus takes over, or if the uterus does not achieve relaxation between contractions, the uterine action becomes dysfunctional and can prevent fetal descent or contract in an abnormal pattern. As a result, uterine contractions decrease blood flow, decreasing the passage of oxygen and carbon dioxide, along with other nutrients, across the placenta.

Knowledge and understanding of maternal physiology is imperative, especially for the perinatal nurse. It is the key to her/his success in understanding, managing, and providing optimal care to his/her patients.

REFERENCES

A Systematic Approach to the Physiologic Adaptations of Pregnancy