Objectives

- Understand the physiologic changes of pregnancy and how they can affect the diagnosis and management of critically ill patients.
- Anticipate and predict the effects of pregnancy on underlying medical conditions.

I. INTRODUCTION

During pregnancy, numerous changes to all maternal body systems (anatomic, physiologic, and biochemical) allow for fetal development and protection, prepare for labor, and compensate for new demands. Variables such as maternal age, multiple gestations, existing health conditions, and genetic factors can affect the mother’s ability to adapt to the demands of pregnancy. Pregnancy can mask or aggravate preexisting diseases in women who, thanks to advances in medical care, are able to conceive with comorbidities that make their obstetric care more complex or place their health at risk. Healthcare personnel must understand the normal physiology of pregnancy to optimize the outcome for these patients.

II. CARDIOVASCULAR SYSTEM

Cardiovascular adaptations are pronounced. While most are tolerated by the mother with little difficulty, coexisting cardiac disease can pose significant problems. For example, underlying conditions that are sensitive to increased blood volume or heart rate may decompensate during pregnancy.
A. Blood Volume

Blood volume increases by 10% as early as the seventh week of gestation, reaching a plateau around 32 weeks. This corresponds to an increase of 45% to 50% (1500-1600 mL, or 1200-1300 for plasma volume) compared to nonpregnant norms. Blood volume in pregnant women ranges between 73 and 96 mL/kg (compared to the nonpregnant value of about 60 mL/kg), and the increase is more pronounced in multiple pregnancies (e.g., twins, triplets). Expansion of plasma volume allows for adequate perfusion of the vital organs, including the uteroplacental unit and fetus. Additionally, total body water increases by 6.5 to 8.5 L by the end of gestation. The water present in the fetus, placenta, and amniotic fluid accounts for approximately 3.5 L of total body water. This expansion in volume also allows for expected blood loss at delivery. In fact, a pregnant woman can lose as much as 2000 mL of blood before showing changes in heart rate and blood pressure.

Onotic pressure depends to a large extent on albumin, which is reduced in pregnant women, even more so in those with preeclampsia.

Expansion of volume with crystalloids further decreases colloid oncotic pressure and increases edema (including pulmonary edema), so caution must be used, especially in cases of preeclampsia.

Normal physiologic changes during pregnancy of plasma proteins and electrolytes can directly affect osmolarity and hydrostatic pressure. These changes are outlined in Table 1-1.

<table>
<thead>
<tr>
<th>Table 1-1</th>
<th>Changes in Serum Parameters</th>
</tr>
</thead>
</table>
| **Plasma proteins** | • Decrease in albumin through hemodilution (20%-40%)  
• Decrease in onotic pressure → edema  
• Decrease in total calcium (increased calcium clearance; no change in ionized calcium)  
• α- and β-globulins increase, facilitating transport of carbohydrates and lipids from placenta to fetus  
• Increase in fibrinogen (50%-80%)  
• Increase in binding globulins → decreased free drug levels |
| **Electrolytes and iron** | • Decrease in osmolarity  
• Decreased HCO₃⁻  
• Decrease in ferritin (30%)  
• Increase in transferrin (70%)  
• Decrease in iron-binding capacity (15%) |
| **Plasma lipids** | • Increase in cholesterol, an estrogen and progesterone precursor (40%)  
• Increase in phospholipids essential for growth (37%) |
B. Anatomic Changes

The heart changes its position, moving toward the head and rotating to the left, as a result of uterine growth and elevation of the diaphragm. There is growth in the four heart chambers, particularly in the left atrium and left ventricle, with thickening of their walls. Mild pulmonary and tricuspid regurgitation occurs in 90% of healthy pregnant women, with more than one-third of these women presenting with insignificant mitral regurgitation. No changes occur in left ventricular function or the ejection fraction. A nonpathologic systolic murmur develops in 90% to 95% of patients. Small Q waves, P- and T-wave inversion (lead III), and changes in ST elevation may be seen on electrocardiography.

C. Blood Pressure

Blood pressure (BP) is a result of cardiac output (CO) and systemic vascular resistance (SVR). Blood pressure decreases by approximately 10% around the seventh week of gestation, reaching a nadir at the midpoint of the pregnancy (28 weeks) and returning to its prepregnancy values around term. This drop in BP is attributed to systemic vasodilation resulting from progesterone.

The BP can vary depending on the patient’s position (Figure 1-1). Both systolic and diastolic pressures increase with uterine contractions. The maximum increase appears in the second stage of labor.

**Figure 1-1. Postural effect on blood pressure.**

Abbreviation: PP, postpartum

D. Heart Rate

Maternal heart rate increases, as early as 5 weeks' gestation; in the third trimester, it is approximately 20% higher than the nonpregnant norm.

E. Cardiac Output

Cardiac output (CO) starts increasing at 10 weeks' gestation, reaching its peak of 30% to 50% above baseline (4-6 L/min) between 25 and 30 weeks. Stroke volume increases by 20% to 35%, starting at 5 weeks, reaching a maximum at about 32 weeks with a slight decline thereafter. Heart rate also affects the increase in CO.

Output is further increased during labor. Each uterine contraction expels 300 to 500 mL of blood into the mother's circulation, increasing venous return and CO by another 30%. Further CO augmentation occurs immediately after delivery of the infant and placenta, when large quantities of fluids (500 mL) enter the central circulation because of the abolition of the low-resistance placental circuit, relief of inferior vena cava compression, and the mobilization of extravascular fluid into the intravascular space. A further increase in cardiac output is expected in the first few hours after delivery of the placenta: see section G, below.

A CO decrease of 20% to 25% may occur when the pregnant woman is placed in a supine position. The uterus compresses the inferior vena cava, decreasing venous return. This is known as the supine hypotensive syndrome, which appears in 0.5% to 11.2% of pregnant women. The syndrome results in tachycardia, diaphoresis, lightheadedness, nausea, vomiting, pallor, and weakness. Women who do not demonstrate this syndrome have compensatory increases in collateral flow through paravertebral and azygos systems leading to increased preload leading to maintenance of blood pressure.

F. Systemic Vascular Resistance

Systemic vascular resistance (SVR) starts decreasing as early as 5 weeks' gestation (10%), reaching its nadir of approximately 35% (980 dynes/sec/cm\(^5\)) below baseline at 14 to 24 weeks. Vasodilation coupled with the low-resistance placental circuit causes a decrease in SVR. A drop in pulmonary vascular resistance also appears but with no changes in pulmonary artery pressure. SVR remains constant until week 32, subsequently increasing until it reaches prepregnancy normal values at term. Normal physiologic cardiovascular and hemodynamic changes seen in pregnancy based on gestational week are described in Table 1-2.
Physiologic Changes During Pregnancy

G. Effects of Labor and Childbirth on Hemodynamics

Each uterine contraction returns between 300 and 500 mL of blood to the central circulation and therefore augments CO. The increase in venous return during uterine contractions leads to transitory maternal bradycardia. In the second stage of labor, pushing increases CO by more than 50%. Heart rate is influenced by maternal position, pain, and individual variations in uterine contractions. Increases in systolic and diastolic arterial pressures precede contractions by 8 seconds; this is attributable to the increase in CO. The changes are less pronounced when the patient is in the left lateral decubitus position. Oxygen consumption increases approximately 3-fold during contractions.

In the immediate postnatal period, CO increases from 60% to 80%, stabilizing in the first hour postpartum. This is due to the autotransfusion phenomenon: uterine blood volume decreases, and the uterus no longer compresses the vena cava.

In the postpartum period, diuresis increases; this starts between days 2 and 5, allowing for the release of excess fluids accumulated during pregnancy. Inadequate diuresis results in acute pulmonary edema and hypertension.

### Table 1-2 Circulatory Changes of Pregnancy

<table>
<thead>
<tr>
<th>Parameters</th>
<th>5 w</th>
<th>12 w</th>
<th>20 w</th>
<th>24 w</th>
<th>32 w</th>
<th>38 w</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR↑</td>
<td>&lt;5%</td>
<td>11%-15%</td>
<td>11%-15%</td>
<td>11%-15%</td>
<td>16%-20%</td>
<td>16%-20%</td>
</tr>
<tr>
<td>SBP</td>
<td>↓&lt;5%</td>
<td>↓&lt;5%</td>
<td>↓&lt;5%</td>
<td>↑&lt;5%</td>
<td>↑6%-10%</td>
<td>6%-10%</td>
</tr>
<tr>
<td>DBP</td>
<td>↓&lt;5%</td>
<td>6%-10%</td>
<td>↓5%</td>
<td>↑6%-10%</td>
<td>6%-10%</td>
<td>6%-10%</td>
</tr>
<tr>
<td>SV↑</td>
<td>5%</td>
<td>21%-30%</td>
<td>&gt;30%</td>
<td>&gt;30%</td>
<td>21%-30%</td>
<td>21%-30%</td>
</tr>
<tr>
<td>CO↑</td>
<td>6%-10%</td>
<td>&gt;30%</td>
<td>&gt;40%</td>
<td>&gt;40%</td>
<td>&gt;40%</td>
<td>&gt;40%</td>
</tr>
<tr>
<td>SVR↓</td>
<td>6%-10%</td>
<td>21%-30%</td>
<td>&gt;30%</td>
<td>&gt;30%</td>
<td>&gt;30%</td>
<td>21%-30%</td>
</tr>
<tr>
<td>LVEF↑</td>
<td>5%</td>
<td>6%-10%</td>
<td>6%-10%</td>
<td>6%-10%</td>
<td>5%</td>
<td>5%</td>
</tr>
</tbody>
</table>

Abbreviations: W, weeks; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; SV, stroke volume; CO, cardiac output; SVR, systemic vascular resistance; LVEF, left ventricular ejection fraction.
III. RESPIRATORY SYSTEM

In the upper respiratory tract, hyperemia, edema, and glandular hypersecretion cause an increase in nasal congestion, rhinitis, and epistaxis during pregnancy. This congestion and edema complicate air intake, obstruct the airflow, and make intubation more difficult (Chapter 2).

Tidal volume and minute ventilation are increased. Functional residual capacity decreases, beginning between 16 and 24 weeks’ gestation and continuing until term. Spirometry studies in pregnant women have not shown any changes in forced expiratory volume in the first second of expiration or in its ratio with functional vital capacity. There are no changes in expiratory flow resistance (Table 1-3).

<table>
<thead>
<tr>
<th>Table 1-3</th>
<th>Changes in Pulmonary Volumes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total lung capacity</td>
<td>Decrease of 200-400 mL (-4%)</td>
</tr>
<tr>
<td>Residual functional capacity</td>
<td>Decrease of 300-500 mL (-17% to -20%)</td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>Decrease of 100-300 mL (-5% to -15%)</td>
</tr>
<tr>
<td>Residual volume</td>
<td>Decrease of 200-300 mL (-20% to -25%)</td>
</tr>
<tr>
<td>Inspiratory capacity</td>
<td>Increase of 100-300 mL (5% to 10%)</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>No changes</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>Increase ≈150 mL (33%)</td>
</tr>
</tbody>
</table>

Minute ventilation is the tidal volume multiplied by respiratory rate. Respiratory rate increases little, if at all; most of the change in minute ventilation (30%-50%) is attributed to an increase in tidal volume to approximately 600 mL. The ratio of dead space to tidal volume changes because of an increase in the non-anatomic alveolar dead space, for which the mechanism is unknown.

Because minute ventilation is increased, respiratory alkalosis occurs. $P_{aCO_2}$ is reduced; the normal value in pregnancy is between 28 and 32 mm Hg (3.8-4.3 kPa). Values in the nonpregnant normal range ($≈40$ mm Hg) signify ventilatory failure in pregnancy. Increases in maternal $P_{aCO_2}$ also decrease transfer of $CO_2$ down the fetal-maternal $CO_2$ transplacental gradient, so fetal acidemia may result. Fetal pH is generally 0.1 units lower than the maternal pH (Table 1-4). Renal compensation for chronic respiratory alkalosis results in a decrease in the bicarbonate levels (18 to 21 mEq/L).

Dyspnea is common during pregnancy, occurring in 60% to 70% of healthy pregnant women.
Oxygen consumption increases 30 to 50 mL/min, two-thirds of which covers the mother’s additional requirements (principally the kidney) and one-third to the placenta and fetus. The combination of increased oxygen consumption and decreased functional residual capacity makes the pregnant woman particularly susceptible to developing hypoxemia during periods of apnea and endotracheal intubation attempts.

### Table 1-4 Arterial Blood Gas Values in the Nonpregnant and Pregnant Patient

<table>
<thead>
<tr>
<th></th>
<th>Not pregnant</th>
<th>1st trimester</th>
<th>3rd trimester</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35-7.45</td>
<td>7.42-7.46</td>
<td>7.44</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>90-100 (12-13.3 kPa)</td>
<td>106 (14.13 kPa)</td>
<td>101-104 (13.46-13.86 kPa)</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>35-45 (4.66-6.0 kPa)</td>
<td>28-29 (3.73-3.87 kPa)</td>
<td>27-32 (3.6-4.27 kPa)</td>
</tr>
<tr>
<td>HCO₃⁻, mEq/L</td>
<td>22-26 (22-26 mmol/L)</td>
<td>18 (18 mmol/L)</td>
<td>18-21 (18-21 mmol/L)</td>
</tr>
</tbody>
</table>

**IV. RENAL SYSTEM**

The collecting systems are dilated as early as the first trimester. Mechanical obstruction is also a factor as the uterus enlarges. The degree of urinary tract dilation is more pronounced on the right than on the left, presumably because the uterus is rotated to the right. Ureteral compression results in urinary stasis that predisposes the pregnant woman to develop infections, nephrolithiasis, and pyelonephritis.

Changes in renal function are due to an increase in renal blood flow (from 35% to 60%), which causes a 40% to 50% increase in the glomerular filtration rate, reaching a peak at ≈180 mL/min. Normal serum creatinine in pregnancy is about half of the normal nonpregnant value: a level of >0.8 mg/dL (70.72 μmol/L) indicates impaired renal function.

Glycosuria is common in pregnancy because of increased glomerular filtration rate and impaired reabsorption of glucose in the loop of Henle and collecting tubule. It does not necessarily indicate diabetes.

**V. GASTROINTESTINAL SYSTEM**

The increase in the size of the uterus causes extrinsic compression of the stomach. Added to the decrease in tone of the lower esophageal sphincter, this increases gastric acidity and reflux. The risk of aspiration thus is high in the pregnant woman, for example, during induction of anesthesia and endotracheal intubation. Commonly measured liver function tests undergo changes during pregnancy as well (Table 1-5).
VI. HEMATOLOGIC SYSTEM

The erythrocyte mass increases by approximately 30%, along with leukocyte levels that rise from 5,000/µL to 12,000/µL, reaching values of between 20,000/µL and 30,000/µL during labor and the puerperium. The platelet count is decreased due to the initial dilutional effect but also to an increase in consumption. Other changes include increases in factors XII, X, IX, VII, and VIII, von Willebrand factors, and fibrinogen, and decreases in factor XI and protein S. The result is a predisposition to thrombosis during pregnancy and the puerperium (Chapter 6).

Key Points

- The physiological changes of pregnancy occur in all maternal systems, though the extent varies by trimester and across individuals.

- An awareness of the normal physiological changes of pregnancy will allow the clinician to predict and anticipate possible complications during the care of critically ill obstetric patients.
Suggested Readings


