Hemodynamic Monitoring in High-Risk Obstetrics Patients, I
Expected Hemodynamic Changes in Pregnancy

Lt Col Elizabeth J. Bridges, USAF NC
Capt Shannon Womble, USAF NC
Capt Marlene Wallace, USAF NC
Capt Jerry McCartney, USAF NC

Critically ill obstetrics patients may require care in an intensive care unit, or critical care nurses may assist in the management of these patients in the labor and delivery rooms. Critical care nurses are often called on because of their cardiopulmonary expertise, particularly in hemodynamic monitoring. Care of high-risk obstetrics patients is complex because clinical data must be interpreted within the context of the physiological changes that occur during pregnancy.

This article provides an overview of the expected cardiovascular changes associated with pregnancy and the pathological changes that occur with some of the more common conditions that require critical care, such as postpartum hemorrhage and cardiac disease. The discussion includes recommendations on how to interpret cardiovascular findings and hemodynamic data in light of the physiological changes associated with pregnancy. A subsequent article addresses hemodynamic monitoring during preeclampsia.

Expected Hemodynamic Changes in Pregnancy

Recognition of the expected hemodynamic changes associated with various phases of pregnancy is important in the interpretation of patients’ hemodynamic profiles and clinical findings. This section provides an overview of the expected hemodynamic changes that occur during pregnancy, at the time of delivery, and during the postpartum phase (Table 1).

Blood Volume

During pregnancy, total body water increases by approximately 6.5 to 8.5 L. Part of the increase reflects the water content of the fetus, placenta, and amniotic fluid (3500 mL) and an increase in maternal blood volume (1000-2000 mL); the remainder is due primarily to an expansion in plasma volume (1200-1300 mL) and an increase in red blood cell mass (300-400 mL). Plasma volume increases by as much as 11% at the seventh week of pregnancy and peaks at approximately 32 weeks, with a 40% to 50% increase in plasma volume compared with nonpregnant levels (Figure 1). Red blood cell mass increases by approximately 20% throughout pregnancy, depending on whether the mother is receiving iron supplementation. The increase in blood volume is protective, as an uncomplicated vaginal delivery causes 500 to 600 mL of blood loss and the mean blood loss with a cesarean delivery is 1000 mL.

The disparity between increased plasma volume and the smaller increase in red cell mass results in a physiological or dilutional anemia. For example, the hematocrit decreases from a normal nonpregnant value of 0.41 to a minimum of
0.31 to 0.37 in late pregnancy, and the hemoglobin level decreases by approximately 9%. The actual decrease depends on iron supplementation.11

**Blood Pressure**

Arterial blood pressure decreases early in pregnancy; mean arterial pressure has decreased by approximately 10% at the eighth week. This decrease is primarily due to a decrease in diastolic pressure. The blood pressure continues to decrease, reaching its nadir or lowest point (6 mm Hg [SD 1 mm Hg] less than pre-pregnancy value) at approximately 16 to 24 weeks.10-24,25 After the 16th week, the blood pressure progressively increases back to baseline (non-pregnant blood pressure) at term.16

Two factors should be considered when blood pressure is evaluated during pregnancy. First, blood pressure is affected by the pregnant

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**Table 1** Hemodynamic changes during pregnancy6-16

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before pregnancy</th>
<th>Third trimester, °C % change (laboratory value)</th>
<th>Labor† (during contractions), °C % change</th>
<th>After delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood volume, L</td>
<td>4.2</td>
<td>↑ 48% (6.2 L)</td>
<td>No change</td>
<td>↓ 0.5-1.0 L (10%-20%)</td>
</tr>
<tr>
<td>Plasma volume, L</td>
<td>2.4</td>
<td>↑ 40-50% (3.4-4.6 L)</td>
<td>No change</td>
<td>↓ 10% by day 5</td>
</tr>
<tr>
<td>Hemoglobin, g/L</td>
<td>120-160</td>
<td>↓ 9% (110-120 g/L)</td>
<td>No change</td>
<td>Total decrease in blood volume (cells + plasma) = 20%</td>
</tr>
<tr>
<td>Hematocrit, proportion of 1.0</td>
<td>0.41</td>
<td>↓ 10-25% (0.31-0.37)</td>
<td>No change</td>
<td>Stable, then ↓ 6% by day 5</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>108</td>
<td>↑ 5-10% (decreased through week 24)</td>
<td>Increases</td>
<td>Returns to prelabor values at 24 hours</td>
</tr>
<tr>
<td>Diastolic</td>
<td>67</td>
<td>↑ 0-10% (decreased through week 24)</td>
<td>Increases</td>
<td>Returns to prelabor values at 24 hours</td>
</tr>
<tr>
<td>Cardiac output, L/min</td>
<td>4.9</td>
<td>↑ 27-50% (7.3 L/min)</td>
<td>↑ 11</td>
<td>Returns to prelabor values at 1 hour and to pre-pregnancy values at 10-14 days</td>
</tr>
<tr>
<td>Stroke volume, mL per beat</td>
<td>65</td>
<td>↑ 21-30% (79-85 mL per beat)</td>
<td>↑ 11</td>
<td>Returns to prelabor values at 24 hours and to pre-pregnancy values at 12 weeks to 1 year</td>
</tr>
<tr>
<td>Heart rate, beats per minute</td>
<td>75</td>
<td>↑ 16-29% (87-95 beats per minute)</td>
<td>↑ 0-20</td>
<td>Returns to prelabor values at 1 hour and to pre-pregnancy values at 6 to 12 weeks</td>
</tr>
<tr>
<td>Systemic vascular resistance, dyn · s · cm⁻⁵</td>
<td>800-1200</td>
<td>↓ 0-20% (34% to nadir at 14-24 weeks)</td>
<td>No change</td>
<td>Returns to pre-pregnancy values at 12 weeks to 1 year</td>
</tr>
<tr>
<td>Central venous pressure, mm Hg</td>
<td>2-6</td>
<td>No change</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Pulmonary artery pressures, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>15-25</td>
<td>No change</td>
<td>ND</td>
<td>Unchanged throughout pregnancy</td>
</tr>
<tr>
<td>End-diastolic</td>
<td>8-12</td>
<td>No change</td>
<td>ND</td>
<td>Unchanged throughout pregnancy</td>
</tr>
<tr>
<td>Wedge</td>
<td>6-12</td>
<td>No change</td>
<td>ND</td>
<td>Unchanged throughout pregnancy</td>
</tr>
<tr>
<td>Pulmonary vascular resistance, dyn · s · cm⁻⁵</td>
<td>220</td>
<td>↓ 25% (175 dyn · s · cm⁻⁵)</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Colloid oncotic pressure, mm Hg</td>
<td>20</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
</tbody>
</table>

*Compared with value before pregnancy.†Compared with third trimester/prelabor values.↑ indicates increase; ↓ decrease; ND, no data.
woman’s body position. Early in pregnancy, blood pressure is highest in the upright or sitting position, intermediate in the supine position, and lowest in the side-lying position. In the third trimester, the effect of body position on blood pressure is variable, depending on the relative degree of compression of the inferior vena cava and the aorta by the gravid uterus (Table 2).

Second, comparison of blood pressure obtained by various techniques (arterial catheter versus automated cuff or auscultation) is not recommended. For example, in postpartum women, measurements of systolic blood pressure obtained by using an automated cuff were significantly lower than measurements obtained by using an arterial catheter, whereas the measurements of diastolic pressures were similar. In patients with preeclampsia, measurements of systolic pressure obtained by using an arterial catheter were 19 mm Hg higher than measurements obtained by using an automated cuff. Conversely, measurements of systolic pressure obtained by using an automated cuff tended to be higher than measurements obtained by using auscultation, and measurements of diastolic pressure tended to be lower (4-8 mm Hg). However, in normotensive patients, average measurements of systolic pressure obtained by using an arterial catheter were 5 mm Hg lower than those obtained by using an automated cuff.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Hemodynamic alterations in response to position change late in the third trimester of pregnancy*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MAP (mm Hg)</td>
</tr>
<tr>
<td>Index</td>
<td>Left lateral</td>
</tr>
<tr>
<td>---------</td>
<td>--------------</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>90 ± 6</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>6.6 ± 1.4</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>82 ± 10</td>
</tr>
<tr>
<td>SVR (dyne · sec · cm⁻²)</td>
<td>1210 ± 266</td>
</tr>
<tr>
<td>PVR (dyne · sec · cm⁻²)</td>
<td>76 ± 16</td>
</tr>
<tr>
<td>PAWP (mm Hg)</td>
<td>8 ± 2</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>3</td>
</tr>
</tbody>
</table>

*Values are means ± SDs. CO indicates cardiac output; CVP, central venous pressure; HR, heart rate; MAP, mean arterial pressure; PAWP, pulmonary artery wedge pressure; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance.
†P < .05 compared with left lateral position.

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Cardiac Output

Cardiac output increases early in pregnancy (13%-20% by 5-11 weeks). The early changes are attributed to an increase in stroke volume and are independent of metabolic rate and pregnancy-induced increases in blood volume (Figure 2). The early increase in cardiac output may be triggered by a decrease in systemic vascular resistance (SVR). Cardiac output continues to increase throughout pregnancy; peak values (27%-50% higher than baseline) occur late in the second trimester (eg, an increase from a prenatal value of 4.9 L/min to 7.3 L/min). The later increase is attributed primarily to an increase in heart rate. During the third trimester, the change in cardiac output is highly variable. Grouped data indicate a decrease; however, data on individual women indicate an increase, a decrease, or no change. The variability may reflect changes related to body position (eg, a decrease in stroke volume due to compression of the inferior vena cava when the pregnant woman is supine) or to the technique used to measure the output.

Two important points about measurement of cardiac output are as follows: First, throughout pregnancy, cardiac output should be measured with the pregnant woman in the same position (eg, left lateral decubitus) because output is lower in supine and standing positions (Table 2). Second, recommendations for the use of cardiac index are equivocal because the correlation between cardiac output and body surface area during pregnancy is poor.

Stroke Volume

Stroke volume begins to increase (approximately 6-10 mL per beat) early in pregnancy (weeks 5 to 8) and continues to increase throughout pregnancy. Maximal stroke volume (32% greater than nonpregnant levels) occurs at 16 to 24 weeks. After the peak at 24 weeks, the stroke volume remains the same or decreases slightly throughout the remainder of the pregnancy.

Heart Rate

Heart rate increases by approximately 8/min early in pregnancy. This early increase is a compensatory response to the decrease in SVR. The heart rate continues to increase to a peak (15-20/min greater than prepregnant value) at term. The decrease in SVR is thought to be due to vasodilatation mediated by gestational hormones, prostaglandins, and creation of low-resistance vascular beds in the uterus and placenta.

Systemic Vascular Resistance

SVR begins decreasing early in pregnancy (30% less than prepregnant value at 8 weeks) and continues to decrease, reaching its lowest point (nadir 34% less than prepregnant value) between approximately the 14th to 24th weeks. After reaching the nadir, the SVR progressively increases toward non-pregnant values at term. The decrease in SVR is thought to be due to vasodilatation mediated by gestational hormones, prostaglandins, and creation of low-resistance vascular beds in the uterus and placenta.

Pulmonary Artery Pressures

Despite an approximate increase of 32% to 47% in pulmonary blood flow during pregnancy, pulmonary...
Left Ventricular Wall Thickness

Left ventricular mass increases during pregnancy because of an increase in the thickness of the left ventricular wall, although this change is not generally detected on electrocardiograms. The increase in wall thickness is greater than the increase in end-diastolic radius, resulting in concentric hypertrophy.30,35,33,47

Oxygen Delivery

Early in pregnancy, oxygen delivery exceeds oxygen consumption, primarily because of the increase in cardiac output, resulting in a decrease in the arteriovenous oxygen difference.48 However, oxygen consumption progressively increases throughout pregnancy, peaking at term (20%-30% greater than prepregnant levels).49 During the third trimester, the progressive increase in oxygen consumption relative to oxygen delivery results in a return of the arteriovenous oxygen difference back to prepregnant levels.

One factor to be considered when interpreting changes in oxygen delivery is that in the third trimester, arterial oxygen content (1.34 × hemoglobin level × arterial oxygen saturation) and mixed-venous oxygen content (1.34 × hemoglobin level × venous oxygen saturation) are decreased. The decrease is due to the physiological anemia of pregnancy.49 The implication of this decrease in oxygen content is that although cardiac output may increase, no significant increase in oxygen delivery occurs because of the offsetting effect of the decreased oxygen content (oxygen delivery = cardiac output × oxygen content).

Hemodynamic Changes During Labor

During labor, hemodynamic changes depend on the pregnant woman’s body position (greater increase in cardiac output in the supine position than in the left lateral decubitus position),50,52 phase of labor,51 and the type of anaesthetics/analgescs used.54 Pain and anxiety associated with labor may contribute to as much as 50% to 60% of the increase in labor-related changes in cardiac output.52,55 In addition, uterine contractions, which translocate approximately 300 to 500 mL of blood into the central circulation, further increase the cardiac output by augmenting the stroke volume.56 For example, in term normotensive pregnant women, uterine contractions increased left ventricular stroke volume by 16% (75 ± 15 mL per beat to 89 ± 17 mL per beat, P < .001) and were associated with an 11% increase in cardiac output (6.31 ± 1.79 L/min to 7.12 ± 1.93 L/min, P < .001).54 The contraction-induced increase in cardiac output is also related to the phase of labor; values at the time of full cervical dilatation are 1 to 2 L/min greater than prelabor values.51,57 Oxygen consumption also increases approximately 23% during labor.58

The hemodynamic effects of epidural and spinal anesthesia depend on the level of the block. For example, “low-block” epidural anesthesia extending to the 10th thoracic dermatome does not markedly affect the hemodynamic response to labor. However, regional anesthesia can cause a decrease in preload because of venodilatation with resultant hypotension.54,59

Hemodynamic Changes After Delivery

After an uncomplicated delivery, the heart rate, stroke volume, mean arterial pressure, and cardiac output remain increased during the immediate postpartum period. Heart rate and cardiac output return to prelabor values at 1 hour postpartum, and the mean arterial pressure and stroke volume return to prelabor values by 24 hours.55 Beginning approximately 24 hours after birth, the cardiac output progressively decreases to nonpregnant values during the next 10 to 14 days.5,10,12,13,17,60 The heart rate returns to prepregnant levels at 6 to 12 weeks.15,36 However, at 6 to 12 weeks postpartum, stroke volume and left ventricular end-diastolic volume remain higher than preconception values, and SVR remains lower than preconception values.7 Even at 1 year, hemodynamic
values may remain altered from preconception values, with greater changes in multiparous women. During the postpartum phase, an acute decrease in blood volume occurs within the first hour after delivery (10% for vaginal delivery and 17% for cesarean delivery). Blood volume continues to decrease until day 5 for both types of delivery. The acute decrease reflects the delivery-related blood loss; the later changes reflect diuresis and a decrease in plasma volume. Despite the decrease in blood volume, for vaginal deliveries, the hematocrit remains relatively stable and may even increase, reflecting the predominant loss of plasma. In women who have cesarean delivery, the hematocrit is relatively stable during the first 24 hours after delivery and then progressively decreases (about 6%) by day 5.

Knowledge of the expected postpartum changes in hemodynamic variables, blood volume, and hematocrit is important to detect signs of postpartum hemorrhage. Because of the increased blood volume during pregnancy, a delivery-related loss of up to 1000 mL may be well tolerated and not cause a compensatory cardiovascular response (eg, increased heart rate compared with predelivery values). However, an acute decrease in hematocrit or indications of cardiovascular compensation, such as increased heart rate and decreased stroke volume compared with predelivery values, are not expected findings and may indicate severe hemorrhage (Figure 3).

An important assessment point is that in most instances of postpartum hemorrhage, blood pressure and cardiac output remain increased because of the compensatory increase in heart rate. Therefore, a decrease in blood pressure and cardiac output are not expected changes in the immediate postpartum period and warrant rapid assessment to detect the cause. Use of a pulmonary artery catheter is not generally required in cases of postpartum hemorrhage unless the patient has additional complications or comorbid conditions, such as acute lung injury or underlying cardiopulmonary disease.

**Indications for Hemodynamic Monitoring in Pregnancy**

The indications for invasive hemodynamic monitoring in pregnant women are similar to those for...
nonpregnant persons, with a limited number of additional pregnancy-specific indications (Table 3). The following section focuses on 1 of the 2 most common indications for invasive hemodynamic monitoring in pregnancy: cardiovascular disease. A separate article focuses on the other common indication for hemodynamic monitoring: preeclampsia.

**Cardiovascular Disease**

Although a relatively small number of pregnancies (1%-2%) involve women with cardiac disease, cardiovascular disease is the leading cause of indirect maternal death during pregnancy. The risk of morbidity and mortality with cardiovascular disease depends on the type of disease, the presence of pulmonary hypertension or cyanosis, ventricular function, functional capacity, and history of cardiac surgery. Maternal mortality increases as the New York Heart Association functional class increases (Table 4).

Use of a pulmonary catheter is recommended only when measurements of pressure and cardiac output of the left side of the heart are required to guide therapy, for example, in patients with complicated mitral or aortic stenosis or pulmonary hypertension or patients with impaired functional status (eg, New York Heart Association functional class III or IV). However, use of a pulmonary artery catheter is not generally recommended for patients with right ventricular outflow obstruction, low pulmonary artery pressure, or right-to-left shunt. Additionally, the risk-to-benefit ratio of pulmonary artery catheterization should be weighed in conditions associated with an increased prevalence of catheter-related complications (eg, Eisenmenger syndrome).

**Challenges in the Diagnosis of Heart Disease**

Physical assessment of pregnant patients who have cardiac disease is challenging because of the normal

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**Table 3** Indications for hemodynamic monitoring in pregnancy

<table>
<thead>
<tr>
<th>Minimal risk (0%-1% mortality)</th>
<th>Moderate risk (5%-15% mortality)</th>
<th>High risk (25%-50% mortality)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial septal defect</td>
<td>Mitral stenosis, NYHA class III/IV</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>Aortic stenosis</td>
<td>Aortic coarctation with valvular involvement</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>Aortic coarctation without valvular involvement</td>
<td>Marfan syndrome with aortic involvement</td>
</tr>
<tr>
<td>Pulmonic valve disease</td>
<td>Tetralogy of Fallot, uncorrected</td>
<td></td>
</tr>
<tr>
<td>Tricuspid valve disease</td>
<td>Previous myocardial infarction</td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot, corrected</td>
<td>Marfan syndrome, normal aorta</td>
<td></td>
</tr>
<tr>
<td>Bioprosthetic valve</td>
<td>Mitral stenosis with atrial fibrillation</td>
<td></td>
</tr>
<tr>
<td>Mitral stenosis, NYHA class I/II</td>
<td>Artificial valve</td>
<td></td>
</tr>
</tbody>
</table>

*NYHA indicates New York Heart Association functional classification: I, Patients with cardiac disease but without resulting limitation of physical activity; II, Patients with cardiac disease resulting in slight limitation of physical activity; III, Patients with cardiac disease resulting in marked limitation of physical activity; IV, Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort.

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changes that occur during pregnancy. For example, dyspnea, edema, increased heart rate, midsystolic murmur due to pulmonic or tricuspid regurgitation, and shifted apical pulse are all expected findings in pregnancy. Compared with findings on prepregnancy chest radiographs, during pregnancy, the cardiac silhouette appears enlarged with increased lung markings. Although these changes typically indicate increased pulmonary venous pressure due to left ventricular failure or mitral valve disease, they are expected during pregnancy. The apparent enlargement of the heart is due in part to the increased thickness of the left ventricular wall and to the upward displacement of the heart by the gravid uterus. Additionally, an increase in heart size occurs because of increased preload.5,24,48 These changes in radiographic findings complicate the diagnosis in pregnant patients with suspected cardiovascular disease. Of note, all other pulmonary and cardiac radiographic characteristics indicative of cardiovascular disease remain unchanged during pregnancy.

The pregnancy-induced distention of the heart leads to dilatation of the valvular rings and subsequent regurgitant murmurs.5,55 Common findings on auscultation include a loud, split S1 (due to delayed mitral valve closure) and early to midsystolic ejection murmurs (eg, tricuspid valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve closure) and early to midsystolic, loud, split S1 (due to delayed mitral valve 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Interaction Between Pregnancy-Induced Cardiovascular Changes and Cardiac Disease

Regardless of the disease process, management of pregnant patients who have cardiac disease requires an understanding of the expected physiological changes (increased intravascular volume, cardiac output, and heart rate and decreased vascular resistance), because these changes may exacerbate or minimize cardiovascular compromise.5 For example, increased intravascular volume may be poorly tolerated in women who have mitral regurgitation.5 In contrast, in patients with aortic stenosis with ventricular hypertrophy, increased preload may be beneficial in maintaining stroke volume and cardiac output. In women with aortic stenosis with ventricular hypertrophy, a PAWP that is “normal” may be insufficient to maintain cardiac output and a higher value (eg, 18 mm Hg) may be needed.75

The pregnancy-induced increase in heart rate may cause cardiovascular compromise for patients with valvular dysfunction (eg, mitral stenosis) in whom diastolic filling time is heart-rate dependent. Patients who have mitral stenosis, the most common rheumatic valvular abnormality in pregnant women, may benefit from beta-blockade to control the heart rate and diuresis to minimize pulmonary congestion.5,75 However, the decision to use diuretics must be made with care because PAWP is not an accurate indicator of left ventricular preload in patients with mitral stenosis, and excessive decrease of intravascular volume may compromise uteroplacental circulation.5

The pregnancy-related decrease in vascular resistance may be beneficial for patients with regurgitant disease (eg, mitral or aortic regurgitation), particularly because of the increase in intravascular volume.74 However, cardiovascular compromise may occur in the postpartum period as vascular resistance returns to normal. If cardiovascular compromise occurs, treatment may include vasodilator therapy (hydralazine) if vascular resistance is increased and diuretics for pulmonary edema.73 In contrast, the expected decrease in vascular resistance may worsen the hemodynamic effects of a stenotic lesion (eg, aortic stenosis) or right-to-left shunts.66,77

The potential for cardiovascular compromise is high during labor, delivery, and the immediate postpartum period. In patients at risk for formation or dissection of aortic aneurysm (eg, women with Marfan syndrome), the careful use of analgesics and β-blocker therapy is recommended to minimize the labor-related fluctuations in cardiac output and blood pressure.56,73,78 During the postpartum period, the increase in intravascular volume may cause pulmonary edema in patients who have mitral stenosis.79 In contrast, any factor that decreases intravascular volume or causes hypotension (eg, postpartum hemorrhage) may worsen preload conditions, such as aortic stenosis.

Acknowledgments
The opinions and assertions contained herein are the private views of the authors and are not to be construed as the official policy or position of the US government, the Department of Defense, or the Department of the Air Force.

References


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