Hemodynamic Monitoring in High-Risk Obstetrics Patients, II
Pregnancy-Induced Hypertension and Preeclampsia

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

Critical care nurses are called upon to assist with the care of critically ill obstetrics patients. Some of the most complex care is required for patients with pregnancy-induced hypertension or preeclampsia. This article provides an overview of the pathological changes and expected hemodynamic changes associated with pregnancy-induced hypertension. A previous article addressed the changes expected during a normal pregnancy and the changes associated with cardiovascular disease or hemorrhage. The clinical manifestations and medical and nursing management are summarized in a case study.

Expected Hemodynamic Changes in Pregnancy

Interpreting the pathophysiological changes that occur with pregnancy-induced hypertension requires a review of the hemodynamic changes expected during pregnancy, particularly during the third trimester. In summary (Table 1), increases in systolic and diastolic blood pressure of up to 10% of baseline are expected.2-4 These increases in blood pressure reflect an increase in stroke volume and cardiac output, despite a decrease in systemic vascular resistance (SVR). Additionally, despite the marked increase in blood volume (as indicated by an increase in left ventricular end-diastolic volume) and pulmonary blood flow, pulmonary artery pressure and pulmonary artery wedge pressure (PAWP) remain at baseline levels throughout pregnancy.5,15,16 Recognition of these expected changes is important. In patients with preeclampsia, SVR may increase dramatically, with a resultant decrease in cardiac output, or a hyperdynamic profile (high cardiac output and low SVR) may persist.8,17-19 Additionally, remembering that pulmonary artery pressure and PAWP generally remain at baseline levels throughout pregnancy is useful in the differential diagnosis of refractory oliguria that may accompany preeclampsia.20,21

Hemodynamic Alterations Associated With Pregnancy-Induced Hypertension, Preeclampsia, and Eclampsia

Pregnancy-induced hypertension is hypertension that develops as a consequence of pregnancy and regresses after delivery. Pregnancy-induced hypertension can be differentiated from chronic hypertension, which appears before 20 weeks’ gestation or continues for a long period after delivery.22 Preeclampsia, which is a type of pregnancy-induced hypertension characterized by progressive hypertension and pathological edema, is clinically defined as a blood pressure greater than 140/90 mm Hg after 20 weeks’ gestation plus proteinuria (300 mg/24 hours or greater than 1+ protein on a dipstick sample of urine collected at random).23 Eclampsia is the occurrence of con-
vulsions or coma unrelated to other cerebral conditions with signs and symptoms of preeclampsia. Preeclampsia and eclampsia may be complicated by the onset of the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count). Patients with HELLP syndrome are a subset of those with severe pregnancy-induced hypertension who are at increased risk for multiple organ system dysfunction. Maternal complications associated with HELLP include a coagulopathy (specifically, microangiopathic hemolytic anemia) due to liver failure and thrombocytopenia, acute respiratory distress syndrome, and acute renal failure; all of these may require hemodynamic monitoring to guide therapy.

The hemodynamic profile of a patient with preeclampsia varies

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before pregnancy</th>
<th>Third trimester, % change (laboratory value)</th>
<th>Labor, % change (during contractions)</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 prepregnancy 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 prepregnancy 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 prepregnancy 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 prepregnancy 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.
Reprinted from Bridges et al., with permission.
depending on the stage of the disease. During the preclinical or latent phase of preeclampsia, the hemodynamic profile is characterized as hyperdynamic, that is, increased cardiac output with normal vascular resistance. With the onset of preeclampsia, the hemodynamic profile varies. In one longitudinal study, women in whom preeclampsia developed crossed over from a high-output state to a high-resistance state, with a dramatic decrease in cardiac output (ie, greater than 3 L/min) and an increase in vascular resistance. However, in other studies, many women had an unchanged profile (high-output or high-resistance states). These differences may be due to variability in treatment among different study populations or to the presence of more than a single hemodynamic profile for preeclampsia. These findings highlight the importance of individualizing the treatment of patients who have preeclampsia.

Hemodynamic monitoring may be required for 3 subsets of patients with preeclampsia, specifically patients with refractory oliguria, pulmonary edema, or refractory hypertension. In patients with preeclampsia, central venous pressures correlate poorly with PAWP; thus if hemodynamic monitoring is needed, a pulmonary artery catheter is generally required.

**Refractory Oliguria**

Three different hemodynamic subsets of patients with preeclampsia with persistent oliguria have been described (Table 2). Subset 1 (low PAWP, hyperdynamic left ventricular function, and normal or increased SVR) is consistent with intravascular volume depletion. Patients in this subset respond to volume resuscitation. Subset 2, which is characterized by normal or increased PAWP, normal cardiac output, and normal SVR, is thought to be caused by renal arteriospasm. Treatment for patients in this subset is focused on reducing arteriospasm by administration of low-dose dopamine. In subset 3, the hemodynamic profile is consistent with systemic vasoconstriction (increased PAWP, increased SVR, decreased cardiac output). Treatment for patients in this subset is focused on afterload reduction and diuresis to improve ventricular function.

**Pulmonary Edema**

The risk of pulmonary edema is increased in patients with pregnancy-induced hypertension and preeclampsia because of a decrease in colloid osmotic pressure (COP). COP, which is approximately 16 to 20 mm Hg in a healthy person lying supine, offsets the pressure (capillary hydrostatic pressure) that forces fluid out of the capillary. If the COP is decreased, net movement of fluid into the interstitium is increased, resulting in pulmonary edema. The risk of pulmonary edema is especially high during the postpartum period when a further decrease in COP occurs. The decreased COP is particularly problematic if large volumes of crystalloids are used in resuscitation. Other causes of pulmonary edema, including cardiogenic pulmonary edema (eg, iatrogenic volume overload) and alteration in pulmonary capillary permeability must also be ruled out.

**Refractory Hypertension**

Placement of a pulmonary artery catheter may be useful in treating patients who are refractory to standard hypertensive therapy (eg, hydralazine or labetolol). Monitoring via a pulmonary artery catheter may help differentiate the cause of increased blood pressure, such as increased blood pressure due to increased vascular resistance.

---

**Table 2** Hemodynamic subsets in preeclampsia with oliguria

<table>
<thead>
<tr>
<th>Subset</th>
<th>Causal mechanism</th>
<th>Pulmonary artery wedge pressure</th>
<th>Systemic vascular resistance</th>
<th>Left ventricular function</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Intravascular volume depletion</td>
<td>Low or low normal</td>
<td>Moderately increased</td>
<td>Hyperdynamic</td>
<td>Volume resuscitation</td>
</tr>
<tr>
<td>2</td>
<td>Renal arteriospasm</td>
<td>Increased</td>
<td>Normal</td>
<td>Increased</td>
<td>Volume resuscitation</td>
</tr>
<tr>
<td>3</td>
<td>Generalized vasospasm</td>
<td>Increased</td>
<td>Increased</td>
<td>Suppressed</td>
<td>Afterload reduction</td>
</tr>
</tbody>
</table>

---
**Case Study**

The following case study describes the hemodynamic changes and medical and nursing care for a patient with complicated preeclampsia.

**Findings at Admission**

A 20-year-old, gravida 2, para 0 woman was admitted at 30 weeks’ gestation because of pregnancy-induced hypertension. She was having irregular contractions. She had no history of hypertension or other medical conditions.

Her vital signs were as follows:
- Heart rate: 108/min
- Blood pressure: 160/110 mm Hg
- Respiration: 26/min
- Temperature: 37.4°C (99.2°F)
- Arterial oxygen saturation: 94%

**Nursing Note**

All blood pressure measurements should be done with the patient in the same position (blood pressure is highest in the sitting position and lowest in the side-lying position) and by using the same technique (ie, do not compare a measurement obtained via an arterial catheter with a measurement obtained by using an automated blood pressure cuff).

The assessment findings were as follows:
- Renal system: 4+ proteinuria with a urine output of approximately 12 mL/h
- Generalized peripheral edema
- Respiratory system: lung sounds clear
- Fetal heart tones: heart rate = 140/min, with accelerations and occasional variable decelerations. (Variable decelerations occur in approximately 50% of all fetuses during labor, are usually temporary, and may change with the mother’s body position.)

**Nursing Note**

Despite clear lung sounds, this patient is at increased risk for pulmonary edema, particularly during the post-partum period. Continued close assessment of pulmonary status is warranted. The oliguria may reflect intravascular volume deficit, renal arterial vasospasm, or systemic vasoconstriction. Evaluation of the patient’s response to a bolus of fluid and evaluation of her hemodynamic status will aid in the differential diagnosis of the oliguria.

**Findings 4 Hours After Initial Treatment**

Four hours after treatment, the patient’s vital signs were as follows:
- Heart rate: 120/min
- Blood pressure: 169/104 mm Hg
- Respiration: 28/min
- Arterial oxygen saturation: 87%, with oxygen given at a rate of 10 L/min

Assessment findings were as follows:
- Renal system: urine output 30 to 50 mL/h
- Respiratory system: crackles in lower lobes
- Fetal heart tones: tracing worsened to deceleration with no variability (indicative of fetal compromise)

**Treatment**

The patient was given a 4-g bolus dose of magnesium sulfate and then a maintenance dose of 2 g. Isotonic sodium chloride solution was infused at a rate of 125 mL/h after intravenous administration of a bolus dose of 500 mL. Pitocin was administered to induce labor.

**Nursing Note**

Continued close monitoring of neurological status is warranted to detect signs and symptoms of hypoxemia, impending seizure activity, increased intracranial pressure, or toxic effects of magnesium.

Calcium gluconate, the antidote for magnesium poisoning, should be kept at the bedside; the usual dose is a 1-g intravenous bolus. Administration of crystalloid solution must be performed with caution because the combination of increased volume resuscitation and decreased COP increases the risk for pulmonary edema.

**Findings at Admission**

The patient was given a 4-g bolus dose of magnesium sulfate and then a maintenance dose of 2 g. Isotonic sodium chloride solution was infused at a rate of 125 mL/h after intravenous administration of a bolus dose of 500 mL. Pitocin was administered to induce labor.

**Nursing Note**

Continued close monitoring of neurological status is warranted to detect signs and symptoms of hypoxemia, impending seizure activity, increased intracranial pressure, or toxic effects of magnesium.

Calcium gluconate, the antidote for magnesium poisoning, should be kept at the bedside; the usual dose is a 1-g intravenous bolus. Administration of crystalloid solution must be performed with caution because the combination of increased volume resuscitation and decreased COP increases the risk for pulmonary edema.

**Discussion**

This patient had the classic signs and symptoms of preeclampsia (hypertension, proteinuria, edema) along with oliguria. The oliguria was initially treated with crystalloids. Four hours later, her condition had deteriorated and was consistent with uncontrolled hypertension and pulmonary edema (bilateral crackles and decreased arterial oxygen saturation). After placement of the pulmonary artery catheter, the hemodynamic profile reflected a hyperdynamic state (increased cardiac output and decreased SVR). The pulmonary edema most likely was due to decreased COP and was exacerbated by the slight increase in PAWP associated with the crystalloid administration. (Note: pulmonary edema does not generally become apparent until the PAWP exceeds 18 mm Hg.) As in this case, the treatment for preeclampsia is delivery if possible. The pulmonary edema was treated in a standard manner with diuretics and reduction of intravenous fluids.
patients with increased blood pressure due to increased vascular resistance, when the vascular resistance is decreased, the cardiac output increases without a change in blood pressure. The other less common cause of increased blood pressure is increased cardiac output, which can be diagnosed via pulmonary artery pressure monitoring.24,26,29

Other Clinical Concerns in Preeclampsia

Other factors to consider in the care of patients with preeclampsia are that compared with women who have a normal pregnancy, women with severe preeclampsia have decreased oxygen consumption, oxygen delivery, and oxygen extraction ratio.27,25,28,29,30 The altered oxygen balance may be particularly important in patients with compromised cardiac output. Additionally, in patients with preeclampsia or eclampsia, the blood volume does not increase as much as in a normal pregnancy, making these patients with complicated preeclampsia less tolerant to peripartum blood loss.22

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

Hemodynamic Monitoring in High-Risk Obstetrics Patients, II: Pregnancy-Induced Hypertension and Preeclampsia
Elizabeth J. Bridges, Shannon Womble, Marlene Wallace and Jerry McCartney

Crit Care Nurse 2003;23 52-57
Copyright © 2003 by the American Association of Critical-Care Nurses
Published online http://ccn.aacnjournals.org/

Personal use only. For copyright permission information:
http://ccn.aacnjournals.org/cgi/external_ref?link_type=PERMISSIONDIRECT

Subscription Information
http://ccn.aacnjournals.org/subscriptions/

Information for authors
http://ccn.aacnjournals.org/misc/ifora.xhtml

Submit a manuscript
http://www.editorialmanager.com/ccn

Email alerts
http://ccn.aacnjournals.org/subscriptions/etoc.xhtml