

Hypertensive Disorders of Pregnancy

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Hypertensive disorders occur in approximately 8% of all pregnancies. Nearly one-third of these women receive the diagnosis of preeclampsia. Hypertensive disorders of pregnancy are the leading cause of morbidity and mortality worldwide. In the United States, these disorders account for approximately 19% of maternal deaths.¹ In addition, it is the most common reason for peripartum admission to the intensive care unit. Following this lecture, participants will be able to discuss the peripartum diagnosis and anesthetic management of hypertensive disorders of pregnancy.

Definitions²:

Chronic Hypertension: Blood pressure \geq 140/90 mm Hg diagnosed prior to 20 weeks gestation or hypertension diagnosed for the first time during pregnancy that does not resolve postpartum.

Preeclampsia-eclampsia: Blood pressure \geq 140/90 mm Hg diagnosed after 20 weeks gestation and accompanied by proteinuria ($>$ 300mg/24 hr). Preeclampsia may be mild or severe. The diagnosis of eclampsia is made with the onset of seizures in a woman with preeclampsia that cannot be attributed to another cause.

Preeclampsia superimposed on Chronic Hypertension: New onset proteinuria or, in women with hypertension and proteinuria prior to 20 weeks gestation: sudden increases in proteinuria, sudden worsening of hypertension, thrombocytopenia, or abnormal elevations of alanine aminotransferase or aspartate aminotransferase.

Gestational Hypertension: Blood pressure \geq 140/90 mm Hg first detected after mid-pregnancy without proteinuria.

CASE I: A 20 y/o G₃P₀ with obesity and Class D diabetes is admitted at 34 weeks gestation with a blood pressure of 160/90 mm Hg and proteinuria. Her blood pressure improves and she is induced at 36 weeks gestation. The preanesthesia evaluation reveals generalized edema and a Mallampati III airway. She refuses early epidural analgesia. After a lengthy induction, the obstetricians declare “failure to progress” and preparations are made for cesarean delivery.

This discussion will focus on the choice of anesthesia for cesarean delivery in patients with preeclampsia. Even when regional anesthesia is planned for cesarean delivery, preoperative evaluation requires careful airway examination. Although the possibility of difficult airway can be minimized with airway assessment (including listening for hoarseness or stridor), airway edema may not become apparent until laryngoscopy. Although early epidural catheter placement is beneficial for several reasons (e.g. patient comfort, attenuated hemodynamic responses to pain, improved uteroplacental blood flow, avoidance of general anesthesia, possible decreasing platelet counts), some patients are not candidates for regional anesthesia or will refuse these techniques. Regional anesthesia is preferred to general anesthesia because of increased maternal mortality associated with general anesthesia.³ In preeclamptic patients, regional anesthesia is clearly preferable when circumstances permit its use.

Prior to initiation of regional anesthesia, evaluation of coagulation status is recommended. Because thrombocytopenia is the most common coagulopathy in preeclampsia, a screening platelet count is recommended in all patients with preeclampsia.⁴ In most patients, it is safe to monitor only the platelet count. One of the most frequently asked questions of obstetric anesthesiologists is, “What is the lowest

acceptable platelet count for spinal or epidural anesthesia without risking an epidural hematoma?” Although an arbitrary level of $100,000/\text{mm}^3$ is often suggested, there are no supporting outcome data. Despite concerns about abnormal platelet counts and function in preeclamptic patients, there are no reports of epidural hematoma in these patients following epidural anesthesia. The actual platelet count safe for spinal or epidural placement is unknown. The widespread belief that the risk of epidural hematoma is increased with platelet counts less than $100,000/\text{mm}^3$ is based on a study correlating platelet counts with bleeding times.⁵ However, the bleeding time has been abandoned in many institutions because of its failure to predict surgical bleeding. Reviews of the literature demonstrate that there are no existing data to support its ability to predict adequacy of hemostasis.⁶ In addition, bleeding from the skin does not predict bleeding from other parts of the body. The platelet count is just one indicator of a patient’s coagulation status. If there is obvious evidence of clinical bleeding (e.g. petechiae, ecchymoses), other coagulation tests may be indicated to exclude disseminated intravascular coagulation. If the platelet count is $>100,000/\text{mm}^3$ and there is no evidence of clinical bleeding, it is unlikely that the PT or aPPT will be prolonged. In a study of preeclamptic patients without evidence of hemorrhage or abruptio but with platelet counts less than $100,000/\text{mm}^3$, there were no other abnormal coagulation tests (i.e. PT, PTT, fibrinogen).⁷ Thromboelastography (TEG) has also been used to rapidly provide information about the adequacy of platelet function and other coagulation factors. Although some anesthesiologists use TEG to assess hemostasis before administration of epidural anesthesia in parturients with thrombocytopenia, further studies are needed to determine the ability of the TEG to predict the risk of epidural hematoma in such patients.

A more difficult situation is the patient who has a significant decrease in the platelet count after a short period of time (i.e. $150,000/\text{mm}^3$ to $100,000/\text{mm}^3$). Administration of regional anesthesia in these patients is dependent on clinical judgment. The remote

risk of epidural hematoma must be weighed against the benefits of regional anesthesia.

If regional anesthesia is not contraindicated, either spinal or epidural anesthesia can be administered safely in most patients with preeclampsia. Spinal anesthesia has been relatively contraindicated in patients with severe preeclampsia because of the potential risk of sudden hypotension with rapid onset of sympathetic blockade. However, there is growing support for the administration of spinal anesthesia for cesarean delivery in preeclamptic parturients. Advantages of spinal anesthesia include: 1) avoidance of general anesthesia (i.e. risks of airway misadventure and hypertension associated with laryngoscopy and endotracheal intubation); 2) rapid onset; 3) increased reliability; 4) less risk of trauma in the epidural space. A small prospective randomized study comparing spinal versus epidural anesthesia during cesarean delivery did not find significant differences in blood pressures between groups.⁸ In addition, a retrospective evaluation of spinal versus epidural anesthesia in patients with severe preeclampsia found no difference in the incidence of hypotension.⁹ However, fluid requirements were increased in patients receiving spinal anesthesia. More recently, a prospective cohort study compared the incidence and severity of spinal anesthesia-associated hypotension in severely preeclamptic versus healthy parturients.¹⁰ Despite study limitations¹¹, the authors demonstrated that severely preeclamptic women receiving spinal anesthesia for cesarean delivery are at no greater risk of catastrophic hypotension than normotensive women when using standard spinal doses.¹⁰ Although there are no large randomized trials that support the administration of spinal anesthesia in parturients with severe preeclampsia, spinal anesthesia may be preferable to general anesthesia when the patient does not have an existing epidural catheter or there is insufficient time because of non-reassuring fetal heart rate patterns.

CASE II: A 40 y/o G₂P₁ with 35 week gestation is admitted with a blood pressure of 180/112 mm Hg and 4+ proteinuria. Admission labs reveal a platelet count of 50,000/mm³, AST =150, and hematocrit of 20%. Her cervix is 2 cm dilated, 50% effaced, and vertex is –1 station. Induction of labor is planned.

HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome is a complication of preeclampsia that develops in 10 to 20% of these patients. Many patients with severe preeclampsia or eclampsia develop a microangiopathic hemolytic anemia associated with thrombocytopenia and elevated liver enzymes. Ten to twenty percent of these patients will have a normal blood pressure, making the diagnosis sometimes difficult. Initial management resembles the treatment of severe preeclampsia. First, the maternal condition is stabilized, especially blood pressure and coagulation. Although HELLP syndrome is not an indication itself for immediate cesarean delivery, delivery is the only definitive treatment for preeclampsia. Patients developing laboratory evidence of disseminated intravascular coagulation are delivered immediately, regardless of gestational age. This discussion will focus on blood pressure control and options for labor analgesia.

Although the anesthetic care of parturients with mild preeclampsia differs little from routine management, patients with severe disease (i.e. BP \geq 160/90 mm Hg) require careful consideration. Maternal morbidity from pulmonary edema and cerebral hemorrhage should be prevented. In cases of severe disease, blood pressure should not be “normalized.” The National Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy suggests that blood pressures of at least 160/105 mm Hg should be treated with antihypertensive medications² to prevent the increased risk of cerebral vascular accidents. Because uteroplacental perfusion is proportionate to blood pressure, precipitous decreases in blood pressure can cause fetal compromise. Consequently, antihypertensive agents must be titrated.

Labetalol, hydralazine, and sodium nitroprusside are antihypertensive agents administered for blood pressure control. Labetalol is representative of a class of drugs that act as competitive antagonists at both α_1 and β -adrenergic receptors. The potency of the mixture for beta blockade is 5 to 10 fold that for alpha blockade. Labetalol produces decreased heart rate and contractility. Although systemic vascular resistance is reduced, cardiac output is maintained. Labetalol also preserves uteroplacental perfusion. Disadvantages include variable dose requirements and duration. The National Education Program Working Group on Hypertension in Pregnancy 2000 has established guidelines for labetalol administration in patients with hypertensive disorders during pregnancy.² Table 1.

Hydralazine, an arteriolar vasodilator, is also administered for blood pressure control. It remains a popular drug for patients with preeclampsia because it increases renal and uterine artery blood flow. Although 5 to 10mg are administered intravenously every 20 minutes, the onset and duration are often unpredictable. Side-effects include reflex tachycardia and ventricular dysrhythmias. Results of a recent meta-analysis comparing hydralazine, labetalol, and nifedipine suggest that hydralazine is not first-line treatment for severe hypertension. The authors concluded that more clinical trials are needed with labetalol and nifedipine showing the most promise.

Sodium nitroprusside is another alternative antihypertensive most often administered to patients with refractory hypertension and severe preeclampsia. Sodium nitroprusside administration requires placement of an arterial catheter to facilitate continuous assessment of maternal blood pressure. Advantages include a fast onset and short duration. It also preserves uteroplacental perfusion. Side-effects include reflex tachycardia, cerebral vasodilation, and potential cyanide toxicity. Table 2.

One retrospective report suggests that spinal or epidural anesthesia can be performed in patients with platelet counts of 69,000-98,000/mm³ without an increased risk of complications.¹² Most obstetric anesthesiologists will place epidural catheters in patients with platelet counts of 70,000-75,000mm³. In the event of decreasing platelet counts and in the absence of established labor, an epidural catheter can be placed before the situation worsens. Saline can be infused to keep the catheter patent until labor is established and pain relief is required. One recent study also suggests that parturients with antepartum HELLP syndrome may benefit from antepartum steroid administration to increase platelet counts and permit administration of regional anesthesia.¹³ For each patient, the risk of epidural hematoma must be compared with the potential risk of general anesthesia. In patients who develop thrombocytopenia or other coagulopathy with an epidural catheter in place, questions arise about time of catheter removal. Although there are no definite guidelines for management, it seems reasonable to wait until the platelet count normalizes. Unfortunately, platelet counts may not return to normal until 5-6 days post delivery. Most clinicians are uncomfortable with leaving catheters in place for such a time period. If a clinician removes an epidural catheter while a patient remains thrombocytopenic, frequent neurological examinations should be performed to detect potential epidural hematoma formation.

In patients that do not respond to steroids and the platelet count remains low, patient controlled intravenous analgesia (PCIA) is an analgesic alternative.

Although fentanyl, meperidine, and remifentanyl have been utilized for PCIA administration, fentanyl has several advantages: 1) familiarity of fentanyl and PCIA; 2) safety profile of both PCIA and fentanyl; 3) proven efficacy; 4) readily available delivery systems. Despite concern of neonatal respiratory depression at time of delivery following fentanyl administration, neonatal depression is less common than with meperidine and easily treated with naloxone. Other disadvantages of meperidine

include increased placental transfer, fetal plasma levels, and active metabolites when compared with fentanyl. In addition, renarcotization of the neonate following an initial dose of naloxone has been observed following meperidine administration.

Remifentanyl has recently been identified as a potential intravenous parenteral labor analgesic. Although remifentanyl has several advantages including, rapid onset, short duration, and no active metabolites, the therapeutic ratio of remifentanyl is very narrow. Such a therapeutic ratio requires vigilant observation for respiratory depression and a delivery device capable of delivering lockout intervals of 1 to 2 minutes. Currently, PCIA fentanyl provides the best option for parturients presenting with a contraindication to epidural analgesia or placement that is technically impossible.

Table 3.

CASE III: 39 y/o G₃P₂ undergoes induction of labor at 42 weeks gestational age. Her admission blood pressure is 130/80 mm Hg and urine is negative for protein. An epidural catheter is inserted. At 4 cm cervical dilation, the patient develops tonic-clonic seizure activity. She is unresponsive and the fetal heart rate (FHR) is 80 bpm. After oxygen administration, the FHR returns to 120 bpm. The patient becomes responsive 10 minutes following the seizure activity.

This discussion will focus on eclampsia and the evaluation of peripartum seizure activity. Eclampsia is defined as convulsions and/or coma not caused by coincident neurologic disease which occur during the peripartum period. Eclampsia is usually preceded by preeclampsia. Symptoms of headache, visual disturbance, and epigastric pain often precede seizure activity. Eclamptic seizures rarely develop without a premonitory warning. Seizure activity often begins with facial twitching and generalized tonic-clonic seizures developing within seconds. Seizures are generally followed by others and the duration of unresponsiveness is variable. Seizure activity often results in maternal hypoxemia and acidosis. Fetal bradycardia is also common,

however the FHR usually recovers in 3-5 minutes. Intrauterine resuscitation and maternal airway management are of overriding importance. If fetal bradycardia persists for > 10 minutes, it may be due to other causes (e.g. abruption). The initial management of seizure activity includes: 1) supplemental oxygen; 2) airway support versus intubation; 3) lateral positioning; 4) sodium thiopental 50-100mg (terminates seizure activity); 5) supplemental magnesium sulfate; 6) intrauterine resuscitation. However, not all peripartum seizures are eclamptic seizures. Other causes of seizures include: 1) seizure disorder; 2) arteriovenous malformation; 3) ruptured aneurysm/subarachnoid hemorrhage; 4) sagittal sinus thrombosis; 5) tumor; 6) cerebral vascular accident. Criteria for CT scan/MRI include: 1) recurrent seizures despite therapeutic magnesium sulfate; 2) focal seizure activity; 3) decreasing level of consciousness.

Case IV: A 25 y/o G₁P₀ with a 26 week gestation intrauterine pregnancy is admitted to her local hospital with 18 hours of anuria, headache, nausea/vomiting. Her blood pressure is 120/70 mm Hg. Urine output fails to respond to 2 liters of crystalloid. Admission labs reveal a BUN of 36, Cr = 3.0mg/dl, AST = 627, and ALT = 862. The patient is transferred to a tertiary care center. On admission, blood pressure is 90/60 mm Hg. Fetal status is reassuring. However, admission laboratories reveal a Cr = 3.6mg/dl, PT =19.2 sec, PTT =31.7 sec, AST = 425, ALT = 826. Although a total of 500mL Hetastarch is administered after admission, the patient remains anuric.

This case will focus on fluid management and hemodynamic monitoring in preeclampsia. Because excessive administration of crystalloid or colloid may result in pulmonary or cerebral edema, evaluation of the patient's fluid balance is extremely important. The goal of fluid therapy is to: 1) provide an adequate volume to meet daily maintenance requirements and compensate for insensible losses; 2) maintain adequate urine output; 3) provide a carrier for administration of therapeutic agents

(e.g. magnesium sulfate); 4) compensate for any reduction in preload and afterload during the administration of epidural anesthesia. During fluid administration, it is important to remember that the “vascular tree” is contracted and porous but generally not underfilled. For regional anesthesia, careful preloading and fluid titration are imperative. Small volumes (250mL) should be infused incrementally with careful monitoring of blood pressure and fetal status. Attempts to rapidly expand the “vascular tree” may lead to life-threatening pulmonary edema. Lactated ringers and saline readily leak out. While hetastarch and albumin also leak out to a lesser degree, only packed red blood cells remain intravascularly. These women are also more susceptible to blood loss because of minimal blood volume expansion. In the postpartum period, acute blood loss readily produces acute hypotension.

In this case, the blood pressure and urine output remain low despite fluid administration. Why? Does the patient have renal or cardiac failure, or does the patient have low intravascular volume? Is invasive central monitoring necessary? In severe preeclampsia, there is no proven benefit of invasive hemodynamic monitoring. For each patient, benefit and risk must be assessed. Although the indications for invasive central monitoring remain controversial, most agree that the accepted indications in preeclamptic patients include: 1) refractory hypertension; 2) pulmonary edema; 3) refractory oliguria unresponsive to fluid challenge; 4) severe cardiopulmonary disease. Once the decision has been made to place invasive monitoring, the choice of a central venous catheter (CVP) versus a pulmonary artery (PA) catheter is also controversial. When the CVP is greater than 6 cm H₂O, the correlation between the CVP and pulmonary artery catheter is unreliable.¹⁴ Although in most cases, CVP monitoring is adequate, some favor PA catheter monitoring for the additional information obtained. Recent American Society of Anesthesiologists Practice Guidelines for Pulmonary Artery Catheterization suggest that evidence regarding the effectiveness of PA catheterization in obstetrics is lacking and controlled

outcome studies have not been reported.¹⁵ These guidelines state that the “appropriateness of routine PA catheterization depends on the combination of risks associated with the (a) patient, (b) surgery, and (c) practice setting. Routine catheterization is generally inappropriate for low- or moderate-risk patients. Low-risk patients include those with ASA physical status of 1 or 2 or with hemodynamic disturbances unlikely to cause organ dysfunction. Those at moderate-risk are in category ASA 3 or have hemodynamic disturbances that occasionally cause organ dysfunction. Those at high-risk are in category ASA 4 or 5 and have hemodynamic disturbances with a great chance of causing organ dysfunction or death. Although the incidence of insertion-related complications is similar with both methods of central monitoring, there are additional risks with PA catheter placement including pulmonary infarction, pulmonary artery rupture, balloon rupture, thrombosis. Because volume resuscitation can be hazardous in these patients, some will benefit from invasive hemodynamic monitoring but the management must be individualized. Patient status, facilities, and monitoring risk/benefit must be considered. There is little doubt that placement of invasive hemodynamic monitors can be both difficult and dangerous without adequate nursing support and knowledgeable management. Table 4.

Case V: A 35-year-old female presents to the emergency room for treatment of a non-postural headache associated with visual changes. She is without focal neurological symptoms or mental status changes but her blood pressure is 160/95 mm Hg. She received epidural labor analgesia five days ago for an uncomplicated vaginal delivery. The emergency room physician administered 500mg caffeine intravenously. Despite the caffeine, the headache persists and you are consulted about performing an epidural blood patch.

Headache is a common postpartum complaint that affects nearly 40% of women in the week following delivery. Risk factors for the development of postpartum headache include a history of migraine and/or preeclampsia. Although women who develop headache 48 hours or more after delivery are generally evaluated in the emergency department, anesthesiologists are often consulted because postdural puncture headache is the most common postoperative complication of regional anesthesia in obstetric patients. A careful history, including symptoms past and present, a physical examination, and laboratory testing if indicated are essential in guiding proper diagnosis and treatment. Difficult diagnostic problems may require consultation by a neurologist. In addition, reassessment for admission by obstetricians may be necessary. Anesthesiologists should be aware that dural puncture is only one of many causes of postpartum headache. Table 5.

Current obstetric treatment in the United States has resulted in a shift of preeclampsia/eclampsia to the postpartum period.¹⁶ Postpartum preeclampsia or eclampsia may present as a severe headache associated with hypertension up to 14 days after delivery. Although the headache is not related to patient position or posture and is without focal neurological deficits, it can be associated with visual changes and nausea and vomiting. Eclampsia is a form of hypertensive encephalopathy that can manifest as seizures, stupor, and sometimes coma. Eclampsia is a life-threatening emergency that occurs suddenly. Seizures may occur even when the hypertension is not severe, however headache is often a serious premonitory sign. Although postpartum headaches are common, careful evaluation is imperative.

**Table 1. Working Group 2000:
Guidelines for Labetalol Administration During Pregnancy²**

1. 20mg bolus, wait 10 minutes
2. If not effective, then administer 40mg
3. If not effective, then administer 80mg q 10 minutes
4. Do not exceed 220mg (total dose)

Table 2. Blood Pressure Control: Others

Nifedipine	Rapid smooth decrease in BP No parenteral form Interacts with MgSO ₄
Esmolol	Decreased fetal pO ₂ Prolonged fetal β-blockade
Clonidine	Fetal hypoxemia Decreased uterine blood flow
ACE Inhibitors	Neonatal hypotension Neonatal renal failure Teratogenicity

Table 3. PCIA Fentanyl Protocol¹⁷

1. Resuscitation equipment and drugs immediately available (maternal and neonatal)
2. Pulse oximetry
3. One-on-one nursing
4. Ongoing parturient education regarding utilization of PCIA
5. Assurance that support providers understand importance and implications of not assisting parturient with PCIA administration
6. Anesthesiologist administered intravenous fentanyl loading dose 2-3 mcg/kg (150-200 mcg)
7. Initial PCIA parameters: Bolus: 50 mcg
Lockout: 10 minutes
4-hour limit: NIL
No continuous (background) IV infusion of fentanyl
8. As requirements increase as labor progresses the following titration regime is followed:
 - Ensure proper utilization of PCIA
 - Ensure demands produce delivery (e.g. disconnect)
 - Decrease lockout: 10 min to 5 min (i.e. doubles available dose)
 - Reassess in 1-2 hours if requirements are not met
 - Increase bolus: 50-75 mcg
 - Reassess in 1-2 hours
9. This regime usually produces VAS pain scores of 3-4/10 during contractions

(Adapted from: Campbell DC. Parenteral Opioids for Labor Analgesia. *Clin Obstet Gynecol* 2003;46:616-22.)

Table 4. Invasive Hemodynamic Monitoring

Cardiac Index (CI)	High	Normal	Low
Afterload (SVR)	Low	High	High
Volume (PCWP)	Normal	Low	High
Therapy	β -blocker	Volume/ Vasodilator	Vasodilator/ Inotrope

Table 5. Differential diagnosis of postpartum headache

Postdural puncture headache
Nonspecific headache
Migraine headache
Caffeine withdrawal headache
Preeclampsia
Hypertension
Subdural hematoma
Subarachnoid hemorrhage
Cortical vein thrombosis
Tumor
Sinusitis
Meningitis
Pneumocephalus
Drugs
Lactation headache
Hypoglycemia

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