OBG MANAGEMENT

Management of obstetric hypertensive emergencies

A strategy based on the triad of diagnosis, stabilization, and delivery can minimize risk of catastrophic end-organ damage.

John T. Repke, MD
OBG MANAGEMENT Board of Editors
Professor and Chairman, Department of Obstetrics and Gynecology, Penn State College of Medicine—Milton S. Hershey Medical Center

Life-threatening obstetric hypertensive emergencies cannot be entirely prevented, but the risk of serious complications can be minimized.

The spectrum of hypertensive disease that can complicate pregnancy is broad—ranging from so-called "white coat" hypertension to gestational hypertension, chronic hypertension, chronic hypertension with superimposed preeclampsia, to preeclampsia.

Particularly challenging, however, is hypertension in pregnancy that becomes severe enough to qualify as a hypertensive crisis, bringing on immediate risk to both fetus and mother.

Risk may evolve over days—or hours—and may present as worsening blood pressure culminating in hypertensive crisis. Fetal morbidity and mortality, including placental abruption and acute fetal distress, are often directly linked to the maternal risks of hypertensive encephalopathy and cerebrovascular accident.

Placental abruption and fetal distress are common with severe hypertension even without encephalopathy and cerebrovascular accident. Abruption is unpredictable and potentially catastrophic, even with intense monitoring.

Aggressive BP control, while fundamental, needs to be balanced against the risks to both mother and fetus of overcorrection and undercorrection.

Defining a crisis

What truly defines hypertensive obstetric emergency is a matter of some debate.

Persistent blood pressures above 200 mm Hg systolic and/or 115 mm Hg diastolic qualify, but some have advocated 160/110 mm Hg as the threshold for emergent treatment of blood pressure. Others suggest that the rate of change in blood pressure is what precipitates the crisis, as opposed to the absolute blood pressure readings.

Why BP control is critical

The true pathophysiology of hypertensive crisis in pregnancy is obscure, but undoubtedly shares characteristics seen in the nonpregnant adult. Diagnosing a hypertensive emergency in the nonpregnant adult, in contrast to diagnosis of an obstetric hypertensive emergency, relies more on clinical manifestations of hypertension than on absolute blood pressure level.[1]

Pathophysiology

In the nonpregnant adult, 2 independent processes are thought to be necessary for the full-blown encephalopathic picture: dilation of the cerebral vasculature and fibrinoid necrosis. In the initial phases of severe hypertension, the cerebral vessels constrict to maintain cerebral perfusion pressure in the face of increased systemic

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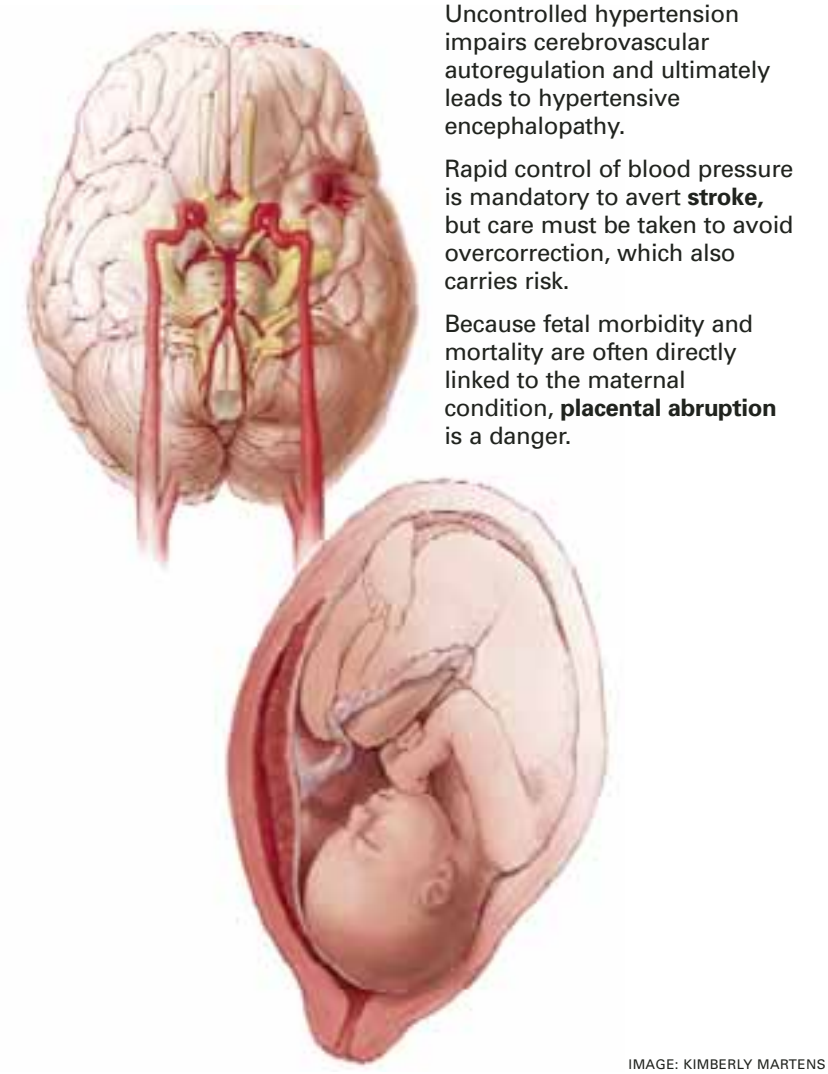
Why hypertensive emergencies endanger both mother and fetus



Uncontrolled hypertension impairs cerebrovascular autoregulation and ultimately leads to hypertensive encephalopathy.

Rapid control of blood pressure is mandatory to avert **stroke,** but care must be taken to avoid overcorrection, which also carries risk.

Because fetal morbidity and mortality are often directly linked to the maternal condition, **placental abruption** is a danger.

IMAGE: KIMBERLY MARTENS

arterial pressure. Once the limits of autoregulation are exceeded, reflex cerebral dilatation and resultant overperfusion lead to microvascular damage, exudation, microthrombus formation, and increased intracranial pressure, which in turn result in the encephalopathic picture.

In pregnancy, a prominent feature seems to be loss of cerebrovascular autoregulation, resulting in hypertensive encephalopathy once the upper limits of cerebral perfusion pressures are exceeded.[2] Rapid control of blood pressure is needed even more because of the risks of placental abruption and stroke (see *above*). Stroke is of special concern in the setting of thrombocytopenia or HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome. Cerebral edema may be more closely associated with endothelial cell injury than with blood pressure,[3] although control of blood pressure may help minimize the endothelial cell injury.

Minimizing organ damage

First, restore normal BP

The most important clinical objective for treatment of acute hypertensive crisis in the nonpregnant adult is to minimize end organ damage, especially to the brain[4]; in obstetric cases, the major morbidity and mortality result from cardiac and renal, as well as cerebrovascular damage. Fetal morbidity and mortality, although not inconsequential, is often directly linked to the maternal condition, and therefore management is based on the triad of diagnosis, stabilization, and delivery.

The physiological dysfunctions described above are best tended to by aggressively controlling blood pressure. With restoration of acceptable blood pressures, generally in the range of 140 to 150 mm Hg systolic and 90 to 100 mm Hg diastolic, cardiac dysfunction begins to reverse, renal function tends to improve, and the restoration of cerebral autoregulatory capability lessens (but does not eliminate) the likelihood of stroke.

Rule out other causes

The foremost goal of therapy for malignant hypertension is to restore normal blood pressure, which depends on correct diagnosis so that appropriate pharmacotherapy may be initiated. For example, clinical situations that could cause malignant hypertension include such disparate entities as acute aortic dissection, acute left ventricular failure, pheochromocytoma, monoamine oxidase inhibitor–food (tyramine) interactions, eclampsia, and acute cocaine intoxication, to name but a few.

Frequently, chronic hypertension or

Clip-and-save chart

Stepwise drug therapy for obstetric hypertensive crisis

INDICATIONS	MEDICATION AND DOSAGE	REMARKS
STEP 1 HTN persisting >60 minutes: SBP >160 mm Hg DBP >110 mm Hg	**Labetalol:** Initiate 5 to 10 mg IV, then double the dose every 15 to 20 minutes to a cumulative maximum dose of 300 mg	**Contraindications:** Heart failure, asthma, chronic obstructive pulmonary disease, heart block **Should be compatible** with Labor and Delivery Unit management policies and with capability of critical care monitoring
STEP 2 If above regimen is inadequate to consistently maintain BP <160/110 mm Hg	**Hydralazine:** Initiate 10 mg IV, then double the dose every 10 to 15 minutes, to a cumulative maximum dose of 300 mg or **Nifedipine:** Give 10 mg by mouth every 15 to 20 minutes, to a cumulative maximum dose of 90 mg	**Contraindications:** Inability to tolerate vasodilation **Contraindications:** Patient contraindications to calcium channel blockers, CHF, heart block failure, heart block
STEP 3 If above regimen is inadequate to consistently maintain BP <160/110 mm Hg	**Nitroprusside:** 0.25 µg/kg/minute	**Manage in ICU,** using personnel experienced in use of nitroprusside **Consider stabilization and delivery**
THERAPY TO PREVENT PROGRESSION TO ECLAMPSIA		
Begin as soon as possible, unless diagnosis of preeclampsia can be excluded with certainty	**Magnesium sulfate:** Loading dose of 4 to 6 g as a 20% solution over 15 to 20 minutes. Then, continuous infusion via pump at a rate that depends on the patient's renal function	In a patient with normal renal function, a rate of 2 g/h is appropriate, but may need to be reduced if acute renal failure ensues. Continue in patients with superimposed preeclampsia until obvious signs of disease resolution and for at least 24 hours postpartum
POSTPARTUM THROMBOCYTOPENIA OR CHF		
HTN persistent for >60 minutes: SBP >160 mm Hg DBP >105 mm Hg MAP >125 mm Hg	**Same as above**	Consider use of **furosemide** as an adjunct to the listed antihypertensive regimens. Volume overload not infrequently accompanies these cases, and such diuretic therapy is essential when pulmonary edema has complicated the clinical picture

severe preeclampsia defines the underlying "cause" of the severe hypertension, but consideration of other diagnoses, such as uncontrolled hyperthyroidism or pheochromocytoma, should not be overlooked. For example, in pheochromocytoma blood pressures tend to be paroxysmal with wide fluctuations. In hyperthyroidism, clinical signs or symptoms would be expected to accompany the clinical picture, such as the presence of proptosis, exophthalmos, lid lag, tremor, elevated temperature, and a wide pulse pressure, to name but a few.

Regimens to lower BP safely

It is imperative that blood pressure be lowered in a measured and safe manner, not to exceed a drop of 25% to 30% in the first 60 minutes, and not to drop below 150/95.[4] Medications available for blood pressure reduction are listed in the *Clip-and-save chart* above.[5]

Every effort must be made to *not overcorrect* the hypertension. Too swift or too dramatic a reduction in blood pressure can have untoward consequences for both mother and fetus, including but not limited

to acute fetal distress secondary to uteroplacental underperfusion, and the possibility of maternal myocardial or cerebral infarction. For these reasons, short-acting intravenous agents are recommended to treat hypertensive emergencies, and oral or sublingual compounds are to be avoided because they are more likely to cause precipitous and erratic drops in blood pressure.[6]

Pulmonary edema is not uncommon, due to capillary leakage and myocardial dysfunction. Aggressive use of furosemide along with a rapidly acting antihypertensive drug will best allow for improvement of the clinical picture in a timely manner.

Acute management steps

Critical care facilities required. During the acute management phase, patients should be cared for in an intensive care unit (or a labor and delivery unit with critical care capabilities) under the direction of physicians skilled in managing critically ill patients. In most institutions, such management will include participation of anesthesiologists, maternal-fetal medicine specialists, and nurses with critical care expertise.

Delivery considerations. During initial management, the patient should have continuous fetal heart rate monitoring. Under such extreme circumstances, it is often not possible to prolong a pregnancy that is remote from term. Delivery decisions will need to balance prematurity risks against maternal risks of continuing the pregnancy.

Hypertension is not a contraindication to glucocorticoids for accelerating lung maturation in the fetus and minimizing neonatal risk of intracranial hemorrhage and necrotizing enterocolitis.[7] Adjusting for gestational age, neonates of preeclamptic mothers are afforded no additional maturity compared with neonates born prematurely for other reasons. Delay of delivery for 48 to 72 hours may not be possible in many cases, however. Once the patient is stabilized, delivery must be considered.

FAST TRACK

I prefer a loading dose of 6 g magnesium sulfate for preventing progression to eclampsia

Start magnesium sulfate, continue antihypertensives

At this point, it is prudent to start magnesium sulfate to prevent eclampsia. In most cases, however, excluding a diagnosis of preeclampsia in a timely manner is nearly impossible. Under these circumstances, magnesium sulfate is recommended, in addition to continued antihypertensive medications, to maintain BP control.

Magnesium sulfate is best administered intravenously, preferably through an infusion pump apparatus. A loading dose of 4 to 6 g (I prefer 6 g) is given as a 20% solution over 15 to 20 minutes, and then a continuous infusion may be initiated at a rate that depends on the patient's renal function. In a patient with normal renal function, a rate of 2 g per hour is appropriate, but may need to be reduced if acute renal failure ensues.

In a report of 3 recent cases, investigators found magnesium sulfate was beneficial for controlling the clinical symptoms of pheochromocytoma when conventional therapy was unsuccessful. The presenting symptoms of these *nonpregnant* patients included hypertensive encephalopathy (2 patients) and catecholamine-induced cardiomyopathy (1 patient).[6]

In general, however, the role of magnesium sulfate should be for preventing progression to eclampsia, and not for acute blood pressure control.

Delivery decisions

Vaginal delivery is often less hemodynamically stressful for the mother, but not always practical. Many cases are remote from term with the fetus in a nonvertex presentation, or the uterine cervix is unfavorable for induction, or a protracted attempt at labor induction may not be prudent.

Under such circumstances, cesarean delivery must be considered and may be preferable. The reasons relate to the underlying maternal condition that often includes some degree of uteroplacental

insufficiency. Altered placental function, combined with extreme prematurity, often results in the fetus being unable to tolerate labor for very long, necessitating emergent cesarean under potentially less controlled circumstances. The anesthesiologist and others on the critical care team must review the optimal anesthesia technique.

In most circumstances, and in the absence of coagulopathy, regional anesthesia affords the best results. When regional anesthesia is not an option, balanced general endotracheal anesthesia with antihypertensive premedication using a short-acting agent may be the safest alternative.

Maintain postpartum vigil

With delivery of the fetus, there may be a temptation to be less rigorous in maintaining blood pressure control during the postpartum period. In patients with chronic hypertension without superimposed preeclampsia, this may be acceptable, as these patients better tolerate higher blood pressures and still maintain appropriate cerebral vascular autoregulation.

For women who were previously normotensive, or who had superimposed preeclampsia, more rigorous control of blood pressure is recommended, especially if they show any degree of thrombocytopenia or pulmonary edema. (See *Clip & save: Stepwise drug therapy for obstetric hypertensive crisis*, page 43.) The rationale relates to cerebral perfusion pressures and risk of stroke in these susceptible women, if thresholds are exceeded, and to the risk of worsening pulmonary edema in the setting of increased capillary hydrostatic pressure and reduced colloid osmotic pressure.

Additionally, continuation of magnesium sulfate is recommended for patients with superimposed preeclampsia until obvious signs of disease resolution, and for a minimum of 24 hours. ■

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