Hypertensive Crisis
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KEY POINTS

- Hypertension is a common, serious disease that is often undertreated.
- Hypertensive urgency or emergency is the presence of elevated risk for or actual end-organ dysfunction caused by the elevated blood pressure. Severely elevated blood pressure itself does not create an emergency.
- A hypertension evaluation is, primarily, the assessment of key organ systems.
- Hypertensive emergencies are decompensated processes requiring immediate stabilization.
- Hypertensive urgencies occur in patients with underlying target organ disease and no evidence of current compounded dysfunction but who have higher risk for near-term complications.
- Severely elevated blood pressure alone does not usually require aggressive therapy.
- Therapy should be determined by the underlying pathology.
- Frequently, the most important intervention is establishing good primary care.

EPIDEMIOLOGY

Worldwide, as many as 1 billion people suffer from hypertension, and about 7.5 million deaths per year are attributed to hypertension. Approximately 28.9% of individuals in the United States, or 85 million, are affected by hypertension. Less than two thirds of U.S. adults with hypertension are aware of their condition, less than half are currently undergoing treatment of it, and only 30% have their blood pressure under control, yet of those seen in hypertensive crisis, it has been previously diagnosed in most of them and they have inadequate blood pressure control. Although hypertensive crisis develops in only 1% of patients with hypertension, some studies have found that hypertensive emergencies account for 28% of all patient visits to the emergency department (ED) for medical complaints, 21% of which were hypertensive urgencies and 6.4% were hypertensive emergencies. Pre-eclampsia (pregnancy-induced hypertension with proteinuria) occurs in 7% of pregnancies and most frequently in primigravida. Uninsured populations, who receive a disproportionate amount of their care in EDs, have a higher prevalence and poorer control of elevated blood pressure. In inner-city public EDs, as many as 20% of the adult population have been found to have blood pressure higher than 140/90 mm Hg.

As an important cardiovascular, cerebrovascular, and renal failure modifiable risk factor, a modest 5–mm Hg decrease in the population is estimated to reduce stroke mortality by 9% and cardiovascular deaths by 12%.

PATHOPHYSIOLOGY

Hypertension is multifactorial and includes genetic and environmental causes, and the causes of hypertensive crises are poorly understood. Hypertension coincides with elevated peripheral vascular resistance (PVR) and normal to low cardiac output. The mechanism of the disease is probably an imbalance in autoregulation of the renin-angiotensin system. Malignant-accelerated hypertensive crises are thought to be due to an abrupt increase in PVR caused by humoral vasoconstrictors leading to endothelial injury, vascular permeability, activation of the coagulation cascade, and necrosis of arterioles. Other hypertensive crises occur when the elevated blood pressure of patients with hypertension exacerbates injury to target organs; it often results in a pathologic feedback loop, which further elevates the blood pressure and exacerbates the damage.

PRESENTING SIGNS AND SYMPTOMS

Hypertensive disease occurs in organ systems in which injury to arterioles leads to ischemic damage or hemorrhage. These target organs include the brain, heart, blood vessels, and kidneys. Therefore, the clinical history and physical examination must include an evaluation of these organ systems.
Hypertensive Crisis

Cerebrovascular decompensation is less likely. Hypertensive encephalopathy produces characteristic findings on computed tomography (CT). Scans show a posterior leukoencephalopathy that predominantly affects the white matter of the parietooccipital regions bilaterally. CT is useful in excluding other causes of altered mental status such as intracranial bleeding.

ACCELERATED-MALIGNANT HYPERTENSION

Accelerated-malignant hypertension occurs most commonly in young African American males with underlying renal parenchymal disease or renovascular disease. It is most commonly found in patients with long-standing hypertension and usually occurs without encephalopathy. When endothelial vasodilator responses are overwhelmed, further hypertension and endothelial damage occur and lead to inflammatory vasculopathy. Marked elevation in blood pressure and characteristic eyeground findings make the diagnosis. Flame-shaped hemorrhages develop around the optic disk because of the high intravascular pressure, and soft exudates are caused by ischemic infarction of the nerve fibers secondary to occlusion of the supplying arterioles. Common symptoms include headache (85%), visual blurring (55%), nocturia (38%), and weakness (30%). Laboratory evidence includes azotemia, proteinuria, hematuria, hypokalemia, and metabolic alkalosis. Papilledema is considered the sine qua non of malignant hypertension. Accelerated hypertension is used to describe the same condition (hemorrhages and exudates) without papilledema. Because the absence of papilledema does not connote a different clinical prognosis or therapy, the term accelerated-malignant hypertension is now recommended.

DIFFERENTIAL DIAGNOSIS

Persistently elevated blood pressure can trigger or exacerbate crises in these target organs. Rapid and progressive target organ damage secondary to severely elevated blood pressure defines a hypertensive emergency. Less commonly, hypertension is the primary crisis. Increasing systemic pressure causes an inflammatory endovasculitis; further damage and aggravation as a result of adrenergic stimulation and vasoconstriction accelerate the elevated blood pressure. The multiorgan disease resulting from an overwhelmed autoregulatory function is called malignant-accelerated hypertension. Inflammatory changes in the cerebral vasculature produce a serious alteration in mental status termed hypertensive encephalopathy. Primary and secondary hypertensive emergencies that must be included in the initial differential diagnosis are listed in Box 69.2.

HYPERTENSIVE ENCEPHALOPATHY

The triad of severe hypertension, altered mental status, and (often) papilledema characterizes hypertensive encephalopathy, and it may be accompanied by lethargy, confusion, headache, visual disturbances, and seizures. Somnolence, stupor, and nausea or vomiting may also occur. Retinopathy may or may not be present. The mechanism of the disease is loss of autoregulation as a result of the cerebral overperfusion caused by profound hypertension; when the hypertension is controlled, the patient’s mental status improves. Persistent overperfusion results in vasodilation and increased permeability of cerebral blood vessels, which in turn leads to the development of cerebral edema. If not adequately treated, hypertensive encephalopathy can progress to cerebral hemorrhage, coma, and death.

Hypertensive encephalopathy is most likely to occur in previously normotensive individuals who experience a rapid rise in blood pressure, such as children with acute glomerulonephritis and young women with preeclampsia or eclampsia. Because chronically hypertensive patients usually experience a more gradual rise in blood pressure, cerebrovascular decompensation is less likely. Hypertensive encephalopathy produces characteristic findings on computed tomography (CT). Scans show a posterior leukoencephalopathy that predominantly affects the white matter of the parietooccipital regions bilaterally. CT is useful in excluding other causes of altered mental status such as intracranial bleeding.

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CEREBROVASCULAR HYPTERTENSIVE CRISIS
Hypertension frequently complicates the management of patients with cerebrovascular accidents (CVAs). After a CVA patients generally have focal neurologic deficits that are somewhat predictable based on the territory of the brain affected. A thorough neurologic examination can elucidate clues about the vessel in which flow has been disrupted either by occlusion or by hemorrhage. Ischemic strokes result from three major categories: thrombotic, embolic, and hypoperfusion. Compromised blood flow produces cell death at the center of the ischemic region and reversibly damaged neurons in the periphery, also known as the penumbra. The penumbra’s viability depends on its perfusion. Hemorrhagic stroke is caused by either intracranial or subarachnoid bleeding. Intracranial pressure (ICP) increases and cerebral perfusion pressure (CPP) is reduced at the site of the hematoma. Therefore, maintaining cerebral perfusion is key in both types of CVA, and an understanding of cerebrovascular physiology is helpful in determining the best treatment strategy.

Cerebral blood flow (CBF), a function of CPP, is equal to mean arterial pressure (MAP) minus ICP (CPP = MAP − ICP). The process of vasoconstriction and vasodilation of the cerebral vasculature maintains a steady CBF. However, cerebral autoregulation fails at approximately 25% above or below the patient’s usual MAP. In addition, changes in ICP or brain injury can result in loss of the brain’s ability to autoregulate blood flow. Increased ICP, commonly seen with hemorrhage or edema, decreases CPP and makes the brain more vulnerable to changes in MAP. In normal individuals, CBF remains fairly constant at MAP values of approximately 60 mm Hg up to 150 mm Hg. When MAP decreases to less than the lower limits of autoregulation, the brain becomes hypoperfused and cerebral hypoxia develops, with symptoms such as dizziness, nausea, and syncope. In chronically hypertensive individuals, the lower limit of autoregulation increases, and autoregulation might fail at MAP values that are well tolerated in nonhypertensive individuals. This suggests that chronically hypertensive patients cannot tolerate a rapid return to “normal” blood pressure and that MAP should be acutely decreased by no more than 20% to 25%.

CARDIOVASCULAR HYPTERTENSIVE CRISIS
Hypertensive emergencies involving the heart and great vessels include congestive heart failure, acute coronary syndromes, and aortic aneurysm or dissection. Blood pressure is frequently elevated in patients with acute pulmonary edema, particularly when a high-output state is the cause, such as volume overload with renal failure, thyrotoxicosis, or severe anemia. Transient diastolic dysfunction, which may or may not be a direct result of the elevated blood pressure, also causes acute pulmonary edema with hypertension and congestive heart failure. Symptoms include tachypnea, tachycardia, pulmonary rales, jugular venous distention, and an S\textsubscript{3} gallop.

Acute coronary syndromes are also frequently accompanied by hypertension. Reducing myocardial work by lowering blood pressure and the heart rate has been demonstrated to decrease infarct size in patients not receiving thrombolytic therapy. Classically, patients have symptoms of chest pain, dyspnea, diaphoresis, nausea, and light-headedness.

Acute aortic dissection is thought to occur as a result of aortic dilation or high blood pressure superimposed on a structural weakness of the arterial wall causing a tear in the intimal layer. Pulsatile pressure extends the dissection by separating the layers of the arterial wall. Historical series report a mortality of 1% to 2% per hour. The stresses that extend the dissection are thought to be related as much to the aortic pulse wave or pulse pressure (the difference between systolic and diastolic pressure over time) as it is to MAP. Heart rate, myocardial contractility, and MAP all contribute to increased pulse pressure. Affected patients include elderly persons with hypertension or atherosclerotic disease and individuals with connective tissue disorders. Hallmark symptoms are acute, severe retrosternal pain radiating to the back or intrascapular pain. Patients may have pulse deficits, neurologic symptoms, or ischemic symptoms in involved organs such as the gut, kidney, or heart.

**RENOSCLEROLARY HYPTERTENSIVE CRISIS**
The kidney is unique in being both a target organ and the cause of many hypertensive emergencies. Hypertension causes 30% of cases of end-stage renal disease, which makes it the second most common cause after diabetes. Nephrosclerosis may develop in chronically hypertensive patients after 10 to 15 years and is manifested as damage to the medial layer of capillaries, reduced kidney size, and nonnephrotic levels of proteinuria without hematuria. By contrast, malignant hypertension damages the intimal layer of the renal capsular bed and may result in enlarged kidneys, cellular urinary sediment, hematuria, and severe proteinuria.

Severe hypertension in a young patient raises the possibility of intrinsic acute renal disease, such as glomerulonephritis. IgA nephropathy has surpassed poststreptococcal glomerulonephritis in frequency, and Henoch-Schönlein purpura is the most likely cause of acute glomerular disease in children.

Renal artery stenosis is present in only 1% of unselected hypertensive patients but is seen in 4% of blacks and 32% of whites who have severe hypertension (diastolic blood pressure > 125 mm Hg with retinopathy). It is also more common in patients with a rapidly progressive course. Occasionally, devastating acute renal failure may occur as a result of intrarenal vasculitis. This is common in the setting of scleroderma and may be responsive to angiotensin-converting enzyme inhibitors (ACEIs).

**CATECHOLAMINE EXCESS**
The most familiar drugs found to cause hypertension in EDs today are sympathomimetic drugs such as phenylephrine, cocaine, and methamphetamine. Tyramine can induce a hypertensive crisis in patients taking a monoamine oxidase inhibitor, and hypertension can complicate withdrawal syndromes from alcohol, benzodiazepines, clonidine, and beta-blockers. Pheochromocytoma can cause intermittent hypertensive crisis.
and may be responsible for many clinical findings besides hypertension, such as headache, sweating, palpitations, pallor, nausea, and rarely, seizures. Some patients with pheochromocytoma may have paroxysms of low blood pressure as well.

**HYPERTENSION IN PREGNANCY**

Third trimester emergencies are addressed separately in Chapter 121. Emergencies include eclampsia and preeclampsia. Pregnant women between 20 weeks’ gestation and 2 weeks postpartum who have any degree of hypertension (≥140/90 mm Hg) or an increase of more than 30/15 mm Hg above their baseline blood pressure, accompanied by peripheral edema and proteinuria, have preeclampsia. Hypertension is important mainly as a symptom of the underlying disorder rather than as a cause. Preeclampsia is essential to recognize because it can progress suddenly to eclampsia, defined by the occurrence of convulsions. Additional symptoms include headache, visual changes, epigastric pain, oliguria, facial and extremity edema, and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count). Eclampsia can rapidly progress to coma or death. Magnesium infusion is more effective than other anticonvulsants in this setting. Because definitive treatment consists of delivery of the fetus, the emergency physician (EP) usually collaborates with an obstetrician early in the patient’s course through the department.

**MEDICAL DECISION MAKING**

EPs evaluate and treat hypertension in a variety of contexts ranging from compliant patients with well-controlled blood pressure, to asymptomatic patients with increased blood pressure, to critically ill patients with increased blood pressure and acute target organ deterioration. Many patients with severely elevated blood pressure have a combination of long-standing, poorly treated hypertension and acute aggravating conditions such as pain, anxiety, or intoxication. Though a major public health risk, elevated blood pressure is rarely a crisis in the ED. Evidence-based national guidelines exist for the evaluation and treatment of hypertension, but there is no good evidence to guide the acute treatment of a patient with severely elevated blood pressure. Instead, the EP relies on an understanding of the disease process, its associated complications, and the health care support available to the patient.

Patients often have elevated blood pressure and nonspecific symptoms. The EP must make a prospective decision about the cause of the symptoms to determine management of the blood pressure. If a hypertensive patient’s chest pain is possibly anginal, immediate parenteral control might be necessary. However, if the EP determines that the pain is not cardiovascular in origin, the same patient might not need immediate treatment of the elevated blood pressure. Findings on assessment of the patient determine the need for treatment.

The concept of hypertensive urgency, which refers to markedly elevated, asymptomatic blood pressure requiring rapid intervention, is no longer widely used. Most patients without acute, progressive target organ disease can have their severely elevated blood pressure managed on an outpatient basis. However, certain patients are at higher risk for near-term complications from their uncontrolled hypertension. This group includes the elderly or frail and especially those with a history of previous end-organ disease (e.g., history of stroke, heart failure, renal insufficiency). These patients do require increased vigilance and possibly aggressive intervention.

**DIAGNOSTIC TESTING**

The nature, severity, and management of hypertensive crises are determined by clinical evaluation. When a patient with markedly elevated blood pressure is seen in the ED, accurate measurement of blood pressure is the first step. Blood pressure that is initially elevated in the ED frequently decreases spontaneously by the time that a second reading is obtained. Any intervention should be based on the composite of several repeated blood pressure measurements. To obtain an accurate measurement, the patient should be seated with the arm at the level of the heart and at least 80% of the arm circumference covered with the cuff bladder. Pressure is evaluated in both arms. Blood pressure measurement with an automated cuff may be inaccurate in patients with atrial fibrillation and other heart rhythm irregularities. Appropriate pain management and relief of the underlying cause (e.g., hypoxia, bladder distention) may resolve the hypertension. Certain medications, over-the-counter preparations, or illicit drugs may transiently exacerbate hypertension (Box 69.3).

**TIPS AND TRICKS**

Elevated triage blood pressure readings often spontaneously improve without treatment. Always recheck abnormal readings.

An initially elevated blood pressure might resolve with proper cuff size or treatment of pain, urinary retention, or hypoxia.

If no emergency is anticipated and immediate parental therapy is not required, patients should be given a dose of the medications that they were supposed to have taken so that some effect has occurred if and when they are ready to be discharged.

Become comfortable with a small number of parental agents; in most instances any one will work.

Elicit a history of use of cocaine or other sympathomimetic drugs.

**BOX 69.3 Medications That Can Elevate Blood Pressure or Interfere with the Effectiveness of Antihypertensive Agents**

- Oral contraceptives
- Steroids
- Nonsteroidal antiinflammatory drugs
- Nasal decongestants
- Cold remedies
- Appetite suppressants
- Tricyclic antidepressants
- Monoamine oxidase inhibitors
If the patient’s blood pressure is persistently elevated, elicitation of the history should start with an assessment of symptoms that might be consistent with target organ compromise. Details include the duration and severity of preexisting hypertension, success with previous blood pressure regimens, and any history of target organ disease (cardiovascular, cerebrovascular, renovascular, and great vessel). Physical examination should be directed toward identifying signs of target organ damage. Funduscopic examination showing retinal hemorrhage or papilledema is sufficient to diagnose accelerated-malignant hypertension. The cardiovascular examination focuses on identifying signs of heart failure (e.g., increased jugular venous pressure, pulmonary rales, and S3 heart sound). The neurologic examination assesses the level of consciousness, visual fields, and the presence of focal motor and sensory deficits.

Fig. 69.1  Management of patients with severely elevated blood pressure (BP). BUN, Blood urea nitrogen; CHF, congestive heart failure; CRI, chronic renal insufficiency; CVA, cerebrovascular accident; DBP, diastolic BP; ECG, electrocardiogram; ED, emergency department; MAP, mean arterial pressure; MI, myocardial infarction; Rx, therapy; SBP, systolic BP; UA, urinalysis.

The patient’s symptoms direct the EP’s diagnostic evaluation. For example, dyspnea or signs of heart failure are an indication for a chest radiograph, and neurologic findings are an indication for CT of the head. Box 69.1 summarizes some common symptoms and their associated end-organs. Few studies have assessed the prognostic value of laboratory testing in asymptomatic patients with severely elevated blood pressure. However, asymptomatic patients with blood pressure persistently higher than 180/110 mm Hg warrant a brief assessment of target organ function (Fig. 69.1). Because renal failure is silent, measurement of serum creatinine or urinalysis (or both) for evidence of renal failure or nephritis is reasonable. An electrocardiogram (ECG) is useful in assessing the baseline level of left ventricular hypertrophy, ischemia, or infarction. The presence of left ventricular hypertrophy on an ECG carries a poor prognosis and necessitates a more vigilant...
follow-up. When renovascular disease or hypercortisolism is suspected to be a cause of the hypertension, serum should be drawn to determine plasma renin activity and aldosterone levels before administering medications. A urine screen for cocaine and amphetamines may help confirm extrinsic causes of the elevated blood pressure. The value of obtaining a chest radiograph or a complete blood count in patients in the ED without relevant symptoms is likely to be low.

TREATMENT

PREHOSPITAL MANAGEMENT

To date, the role of prehospital treatment of hypertension or hypertensive emergencies is minimal. Hypertension complicating angina or pulmonary edema should be treated with nitrates (other medications are not time critical if transport time is brief). Emergency medical service providers should not lower blood pressure in patients with a possible stroke because of the possibility of extending the ischemic region.

HOSPITAL MANAGEMENT

Ideally, ED management of hypertensive crises would entail the administration of fast-acting, rapidly reversible medications that can easily be titrated to the desired effect of a 20% decrease in MAP. No single agent is optimal in all cases of hypertensive crisis. The agent of choice and the manner of administration depend on the severity of the blood pressure abnormality, the severity and type of end-organ dysfunction present, and the clinical findings.

HYPERTENSIVE EMERGENCY

The goal of therapy in patients in hypertensive crisis is a 20% to 25% reduction in MAP over a period of 1 to 2 hours. The ideal drug for treating hypertensive emergencies would easily titrate blood pressure through rapid onset, rapid maximal effect, and rapid offset. These characteristics are found only in parenteral agents. Table 69.1 summarizes the most commonly used medications and doses.

Nitroprusside is the classic agent for the treatment of patients with hypertensive emergencies. Nitroprusside decreases both preload and afterload without significant reflex tachycardia through arteriovenous vasodilation. It has a quick onset of action, and its effect lasts for only 2 to 5 minutes after use of the drug is discontinued. Hemodynamics must be monitored closely to prevent inadvertent hypotension. Thiocyanate toxicity may occur if the drug is administered for more than 48 to 72 hours, particularly in patients with renal failure. It is contraindicated in pregnancy. Because no oral form is available, the patient must be switched to a different antihypertensive once control is achieved. Despite the many benefits of nitroprusside, other agents are often better suited for individual hypertensive crises.

Nicardipine is a rapidly acting parenteral calcium channel blocker. It has a predictable and smooth onset of action, but it is relatively long acting. Esmolol is a beta-blocking agent that is both rapid in onset and of short duration, thus making it easy to titrate. Labetalol, an easily titratable medication, combines alpha- and beta-blockade, which makes it more potent than esmolol. Comparatively, labetalol maintains a more consistent CPP and has a longer half-life. This enables the administration of miniboluses instead of a constant infusion but makes it more difficult to titrate downward. Caution is advised when administering beta-blockers to patients with asthma, chronic obstructive pulmonary disease, acute congestive heart failure, cocaine abuse, or other contraindications to beta-blockade. Fenoldopam is a parenteral dopaminergic receptor blocking agent with an excellent efficacy and safety profile. Fenoldopam holds some promise as being equivalent to nitroprusside in efficacy without the rare side effects associated with nitroprusside’s cyanide moiety and perhaps with less overshoot hypotension, but it is costly. Other options include enalaprilat, a parenteral ACEI, and phenolamine, a pure alpha-blocking agent.

CEREBROVASCULAR CRISIS

Blood pressure control should be undertaken with caution in patients with cerebrovascular hypertensive emergencies. Parenteral drugs that have a short half-life, are easily titrated, and have minimal effect on the cerebral vasculature are ideal. Because labetalol does not dilate cerebral capacitance vessels, it is theoretically attractive in patients with intracerebral disorders. Caution should be used with direct vasodilators such as nitroprusside in patients with focal brain injury because they can extend an area of ischemia. Although nicardipine is safe and widely used, other calcium channel blockers have been linked to a rise in ICP and are therefore not favored in patients with brain injury.

Treatment of elevated blood pressure in the setting of ischemic CVAs is controversial. When systemic blood pressure is reduced, cerebral autoregulation may fail, thereby extending the ischemic penumbra surrounding the infarct and leading to extension of the stroke. Alternatively, infarction may lead to edema, elevated ICP, and a further reduction in CBF. The current American Stroke Association guidelines recommend lowering blood pressure in patients with stroke only when MAP is greater than 130 mm Hg or systolic blood pressure is greater than 220 mm Hg.

Theoretically, treatment of elevated blood pressure in patients with hemorrhagic CVAs and subarachnoid hemorrhage should be more aggressive than in patients with ischemic strokes. The rationale is to decrease the risk for ongoing bleeding from ruptured small arteries and arterioles; however, the relationship between rebleeding and systemic blood pressure is unproven. As with ischemic CVAs, overly aggressive treatment of hypertension may worsen brain injury by decreasing CPP when ICP is increased. The American Stroke Association guidelines for blood pressure control in patients with hemorrhagic stroke are similar to those for ischemic stroke: blood pressure should be lowered only when MAP is greater than 130 mm Hg or systolic blood pressure is greater than 220 mm Hg. Nimodipine, an oral calcium channel blocker, may be administered to decrease the incidence of vasospasm and rebleeding after subarachnoid hemorrhage, but the drug is not recommended for blood pressure control.

CARDIOVASCULAR CRISIS

Nitroglycerin (NTG) is favored for the treatment of severe hypertensive complicating cardiac ischemia. NTG is a direct vasodilator that affects the venous more than the arterial vasculature. NTG dilates the coronary arteries and, in contrast to nitroprusside, promotes a favorable redistribution of blood flow to ischemic areas. Beta-blockers are also effective and
recommended therapy for acute coronary syndromes. The goal of treatment in patients with acute coronary syndromes is reduction of blood pressure to normal if evidence of ischemia persists. However, careful blood pressure reduction requires intensive patient monitoring; overly vigorous lowering of blood pressure may worsen the ischemia because coronary perfusion depends on diastolic blood pressure.

Most critical cases of congestive heart failure are treated with a combination of NTG, furosemide, and an ACEI. For patients with pulmonary edema and hypertension, sublingual NTG should be initiated while preparing intravenous NTG. Captopril should be administered orally or sublingually or enalaprilat administered intravenously. If systemic fluid overload is present, intravenous furosemide should be administered. However, up to 25% of patients with heart failure and severely elevated blood pressure may have “dry failure” in which pressure natriuresis makes them fluid depleted. Further diuresis may exacerbate the process and continue to stimulate

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### Table 69.1 Parenteral Drugs for Treatment of Hypertensive Emergencies

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSE</th>
<th>ONSET OF ACTION</th>
<th>DURATION OF ACTION</th>
<th>ADVERSE EFFECTS</th>
<th>SPECIAL INDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprusside</td>
<td>0.25-10 mcg/kg/min as IV infusion (maximal dose for 10 min only)</td>
<td>Immediate</td>
<td>1-2 min</td>
<td>Nausea, vomiting, muscle twitching, sweating, thiocyanate and cyanide intoxication</td>
<td>Most hypertensive emergencies; caution in patients with high intracranial pressure or azotemia</td>
</tr>
<tr>
<td>Nicardipine hydrochloride</td>
<td>5-15 mg/hr IV</td>
<td>5-10 min</td>
<td>1-4 hr</td>
<td>Tachycardia, headache, flushing, local phlebitis</td>
<td>Most hypertensive emergencies except acute heart failure; caution in patients with coronary ischemia</td>
</tr>
<tr>
<td>Fenoldopam mesylate</td>
<td>0.1-0.3 mcg/kg/min as IV infusion</td>
<td>&lt;5 min</td>
<td>30 min</td>
<td>Tachycardia, headache, nausea, flushing</td>
<td>Most hypertensive emergencies; caution in patients with glaucoma</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>5-100 mcg/min as IV infusion</td>
<td>2-5 min</td>
<td>3-5 min</td>
<td>Headache, vomiting methemoglobinemia, tolerance with prolonged use</td>
<td>Coronary ischemia</td>
</tr>
<tr>
<td>Enalaprilat</td>
<td>1.25-5 mg every 6 hr IV</td>
<td>15-30 min</td>
<td>6 hr</td>
<td>Precipitous fall in pressure in high-renin states; response variable</td>
<td>Acute left ventricular failure; avoid in patients with acute myocardial infarction</td>
</tr>
<tr>
<td>Hydralazine hydrochloride</td>
<td>5-20 mg IV 10-50 mg IM</td>
<td>10-20 min 20-30 min</td>
<td>3-8 hr</td>
<td>Tachycardia, flushing, headache, vomiting, aggravation of angina</td>
<td>Eclampsia</td>
</tr>
<tr>
<td>Diazoxide</td>
<td>50-100 mg as IV bolus repeated or as 15- to 30-mg/min infusion</td>
<td>2-4 min</td>
<td>6-12 hr</td>
<td>Nausea, flushing, tachycardia, chest pain</td>
<td>Now obsolete; used when intensive monitoring not available</td>
</tr>
<tr>
<td>Labetalol hydrochloride</td>
<td>20-80 mg as IV bolus every 10 min or as 0.5- to 2.0-mg/min IV infusion</td>
<td>5-10 min</td>
<td>3-6 hr</td>
<td>Vomiting, scalp tingling, burning in throat, dizziness, nausea, heart block, orthostatic hypotension</td>
<td>Most hypertensive emergencies except acute heart failure</td>
</tr>
<tr>
<td>Esmolol hydrochloride</td>
<td>250-500 mcg/kg/min for 1 min, then 50-100 mcg/kg/min for 4 min; may repeat sequence</td>
<td>1-2 min</td>
<td>10-20 min</td>
<td>Hypotension, nausea</td>
<td>Aortic dissection, perioperative</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>5-15 mg IV</td>
<td>1-2 min</td>
<td>3-10 min</td>
<td>Tachycardia, flushing, headache</td>
<td>Catecholamine excess</td>
</tr>
</tbody>
</table>


*These doses may vary from those in the Physicians’ Desk Reference (51st edition).
†Hypotension may occur with all agents.
‡Requires a special delivery system.
the renin-angiotensin axis. The decision should be based on clinical judgment of whole-body fluid status. Although beta-blockers have been found to improve survival in patients with chronic congestive heart failure, use in patients with acute pulmonary edema may precipitate immediate worsening because of their negative inotropic effects and bradycardia. Intravenous nesiritide improves hemodynamic function and symptoms in patients with decompensated heart failure and has a modest antihypertensive effect, but it has not been well studied in the setting of hypertensive crisis.

With aortic dissection, progression of the vascular injury is dependent not only on the elevated blood pressure but also on the aortic ejection velocity or tachycardia-induced shear forces. Therefore, a rate-controlling agent such as esmolol should be initiated before starting nitroprusside to avoid the effects of reflex tachycardia. Alternatively, labetalol, nicardipine, or fenoldopam has been suggested as possible substitutes for nitroprusside. Labetalol achieves its maximal effect within minutes and then remains effective for several hours, thus allowing titration with small boluses and avoiding the constant monitoring and increased cost required with nitroprusside. Nicardipine and fenoldopam are vasodilators that would require the intensive monitoring needed with nitroprusside but are less toxic alternatives.

RENOVASCULAR CRISIS

When compared with nitroprusside, fenoldopam may improve outcomes in patients with hypertension and acute renal failure. Even though malignant hypertension may precipitate acute renal failure by injuring the kidney’s microvasculature, this causal chain is often reversed with chronic renal failure when long-term renal damage is manifested as severe hypertension. Because this distinction cannot be made in the ED, all these patients should have their blood pressure lowered. Both nitroprusside and labetalol are excellent choices in this setting. Although ACEI drugs definitely improve the prognosis of patients with chronic hypertension and mild proteinuria, they should be used cautiously in hyperkalemic patients with acute uremia. Nitroprusside may cause thiocyanate poisoning over a period of several days in patients with renal failure; it should be used only if briefly or if the patient will undergo dialysis soon.

Treatment with an ACEI may reverse high blood pressure dramatically in patients with unilateral renal artery stenosis, but it may provoke acute renal failure and severe hyperkalemia in patients with bilateral stenosis, particularly if they are taking supplemental potassium or a potassium-sparing diuretic. This complication can be completely reversed by discontinuing the ACEI.

CATECHOLAMINE EXCESS

Patients with severe hypertension secondary to pheochromocytoma are treated with the pure alpha-blocker phentolamine administered intravenously. It may be accompanied by a beta-blocker if needed for tachycardia. Administration of beta-blockers alone in the setting of any sympathomimetic (e.g., cocaine) may leave the alpha receptors “open,” with subsequent worsening of the hypertension. Thus, an attractive alternative to beta-blockers is labetalol, a beta-blocker with some alpha-antagonist properties. However, the alpha- and beta-blockade with labetalol may not be equally effective. Additionally, benzodiazepines are useful adjuncts in patients with cocaine-induced catecholamine excess. They decrease both the central and peripheral sympathomimetic outflow stimulated by cocaine, thereby lowering the heart rate, psychomotor hyperactivity, and blood pressure.

HYPERTENSION IN PREGNANCY

The mainstay of antihypertensive treatment of pregnant patients in hypertensive crisis in many institutions is hydralazine administered intravenously in boluses of 5 to 10 mg every 20 to 30 minutes. This is in addition to magnesium therapy for control of seizures. If the hypertension is refractory to hydralazine, second-line agents are diazoxide and beta-blockers. Calcium channel blockers have been studied in pregnant patients with chronic hypertension, but they may not be effective in treating proteinuric hypertension.

CONSULTATION

Hypertensive emergencies require admission to a monitored setting. These patients generally require emergency involvement of an appropriate specialist for management of a neurologic, cardiovascular, or renovascular crisis. Close blood pressure monitoring, preferably with an arterial line, is indicated. Patients with preeclampsia or eclampsia require emergency obstetric consultation.
has shown that the absolute level of a patient’s blood pressure warrants immediate or aggressive treatment. Rather, in patients with asymptomatic, elevated blood pressure and no evidence of target organ disease, the most important intervention is to ensure proper follow-up. The goal should be lifelong control of the blood pressure.

When the elevated blood pressure may be the artifact of a systemic process such as pain or infection, the best strategy is to refer the patient for reevaluation of the blood pressure once the primary problem has resolved. If the patient has discontinued the blood pressure medications, the regimen should be restarted, barriers to compliance evaluated, and a primary care physician contacted to ensure reevaluation in a week. The hypertension guidelines recommend a thiazide-type diuretic as an initial agent, usually in combination with a drug from another class. The second agent may be from a number of categories and is best chosen in accordance with any compelling indications in the patient’s history (Box 69.4).

In principle, in individuals without a previous measurement of elevated blood pressure, the blood pressure needs to be rechecked at another visit before the diagnosis of hypertension can be made. However, in individuals with readings persistently higher than 180/110 mm Hg in the ED, the latest national guidelines recommend that combination therapy be started immediately (same day). In the best scenario, the EP contacts a primary physician for the patient, who then selects an initial antihypertensive agents or agents and provides follow-up within about a week.

An intermediate group of patients has severely elevated blood pressure and known target organ disease but no active decompensation. An example is a severely hypertensive patient with a previous history of myocardial infarction or stroke. Immediacy arises because a patient with known target organ disease may be considered at higher risk for a hypertension-related adverse event in the short term. However, there is no good evidence base for the best management of these patients. A treatment strategy should be initiated in the ED, although blood pressure does not necessarily need to be lowered during the visit. These patients do require an increased level of vigilance. It may be reasonable to treat them as outpatients, although some may need to be held for short-term observation if their medication compliance or blood pressure monitoring is uncertain; the decision depends on clinical judgment (see Box 69.5).

In practical terms, hypertensive emergencies require an immediate (within 1 to 2 hours) decrease in blood pressure, hypertensive urgency requires initiation of a strategy to decrease and monitor blood pressure over a 24- to 48-hour period, and uncontrolled severe hypertension requires therapy to decrease blood pressure within 1 week. Stratification within these categories involves careful clinical evaluation and understanding of target organ disease and treatment strategies.

**BOX 69.4 Initial Drug Choices for Hypertension**

Use unless contraindicated. Start with a low dose of a long-acting once-daily drug and titrate the dose; low-dose combinations might be appropriate.

**Uncomplicated Hypertension**

- Diuretics
- Beta-blockers

**Diabetes Mellitus (Type 1) with Proteinuria**

- Angiotensin-converting enzyme (ACE) inhibitors

**Heart Failure**

- ACE inhibitors
- Diuretics

**Isolated Systolic Hypertension (Older Person)**

- Diuretics preferred
- Long-acting dihydropyridine calcium antagonist

**Myocardial Infarction**

- Beta-blockers (without intrinsic sympathomimetic activity)
- ACE inhibitors (with systolic dysfunction)

**BOX 69.5 Discharge Criteria for “Hypertensive Urgency”**

1. Likely to be compliant with established primary care
2. Known to have hypertension
3. Reversible precipitating cause (e.g., medication noncompliance, adverse drug effect)
4. Able to resume a previously effective medication regimen
5. Can be seen for follow-up within 7 days.

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**PRIORITY ACTIONS**

**Severely Elevated Blood Pressure**

- Recheck the patient’s blood pressure with the correct method in both arms.
- Evaluate and treat aggravating conditions (e.g., pain, anxiety, intoxication).
- Evaluate for evidence of target organ damage.
- Elicit a history of target organ disease.
- Administer therapy based on the underlying pathology.
- Reevaluate continuously for signs of response to therapy or deterioration.

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**RED FLAGS**

- Diagnosing a hypertensive emergency when one does not exist. Patients with hypertensive emergencies have evidence of acute end-organ dysfunction.
- Reducing blood pressure too quickly or to too low a level in patients with chronic hypertension whose autoregulation curve has been reset can lead to cerebral or cardiac ischemia.
- Lowering a patient’s blood pressure acutely without an urgent indication.
- Failing to diagnose hypertension or preeclampsia in pregnant patients with blood pressures higher than 140/90 mm Hg or with an increase in blood pressure of more than 30/15 mm Hg.
- Neglecting to match the antihypertensive agent to the clinical scenario.
Be sure that any elevated blood pressure is noted and addressed.
Be sure to document any change in blood pressure with treatment.
Document possible causes of the elevated blood pressure.
List any past medical history of target organ disease.
List current antihypertensives and any recent changes in medications or noncompliance.
Document the presence or absence of end-organ dysfunction found during assessment of the patient’s elevated blood pressure.
Document patient counseling for medications, reasons to return, and primary care follow-up.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES


