Hypertensive Emergency

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KEYWORDS

• Hypertensive emergency • Hypertensive urgency • Hypertensive crisis
• Autoregulation

KEY POINTS

- The distinguishing clinical characteristic of hypertensive emergency is an acute increase in blood pressure that is associated with new or progressive end-organ damage.
- Autoregulation is intended to maintain adequate and stable blood flow to the brain, heart, and kidneys during fluctuations in blood pressure. The lower pressure threshold of autoregulation also corresponds with the threshold of hypoperfusion and is approximately 20% to 25% lower than the existing blood pressure. This physiologic observation is the rationale behind the clinical recommendation to limit the initial blood pressure reduction to 20% to 25% of pretreatment values.
- The management of hypertension should always begin with an accurate assessment of blood pressure.
- The patient-physician interaction following a confirmed measurement of severely increased blood pressure should focus on determining whether end-organ damage is present, paying particular attention to the neurologic, cardiovascular, and renal systems.
- Evidence for end-organ damage dictates urgent transfer to an emergency department or inpatient setting, whereas hypertensive patients without end-organ damage can be safely managed in the outpatient setting, which includes an appropriate follow-up plan.

INTRODUCTION

Hypertension is the most common disorder seen in the primary care setting and management of this disorder has become a cornerstone of outpatient clinical practice. It is estimated that at least 30% of the adult population in the United States has hypertension, defined as a systolic blood pressure (SBP) greater than 140 mm Hg, a diastolic blood pressure (DBP) greater than 90 mm Hg, or anyone taking antihypertensive medication. Patients at times present to the clinic with a severely increased blood pressure (BP), known as a hypertensive crisis. Hypertensive crises have historically been further

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subdivided into hypertensive urgencies and emergencies.[1] This subdivision is clinically significant because it should ultimately determine acute BP management.

TERMINOLOGY

Outpatient management decisions can often be delayed or unnecessarily deferred to an emergency room by not having a clear understanding of how to differentiate hypertensive emergency from urgency.

- Hypertensive crisis: usually defined as an acute and severe increase in SBP greater than or equal to 180 mm Hg or a DBP greater than or equal to 120 mm Hg and can occur in both a hypertensive emergency and urgency situation.
- Hypertensive emergency: the severe increase in BP is associated with new or progressive end-organ damage (**Table 1**) and is a true emergency requiring immediate BP control usually over the course of minutes to hours.
- Hypertensive urgency: the severe increase in BP is not associated with end-organ damage, although non–life threatening symptoms, such as anxiety, headache, epistaxis, palpitations, or mild dyspnea, may be present. It is not an emergency, and, contrary to its name, the BP does not require urgent reduction most of the time but instead can be reduced over the course of hours to days.
- Hypertensive emergency in pregnancy: acute-onset, severe hypertension of greater than or equal to 160/110 mm Hg persisting more than 15 minutes. End-organ damage includes severe preeclampsia, HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome, and eclampsia.[2]

It is important to remember that, during the clinical assessment, regardless of the BP measurement, the emphasis remains on determining whether end-organ damage is present. Hypertensive emergency can present in patients with an acute increase in BP with or without a preexisting history of hypertension. Although the incidence of hypertensive emergency is low at less than 2% of all hypertension presentations annually,[3] knowledge of how to recognize key signs and symptoms as well as an understanding of immediate medical management could help reduce patient morbidity and mortality.

HEMODYNAMIC DETERMINANTS OF BLOOD PRESSURE

An overall understanding of the hemodynamic determinants of BP is important in order to effectively manage hypertension.

Table 1
New or progressive end-organ damage associated with hypertensive emergency

End Organ	Damage Type
Brain	Seizure, transient ischemic attack, cerebral infarction, intracerebral or subarachnoid bleed, hypertensive encephalopathy, posterior reversible leukoencephalopathy
Heart	Acute pulmonary edema, acute congestive heart failure, acute coronary syndrome
Blood vessels	Acute aortic dissection, microangiopathic hemolytic anemia
Kidney	Acute kidney injury
Retina	Papilledema, hemorrhages, retinal edema
Uterus	Eclampsia

Mean Arterial Pressure

A pressure is generated when the heart contracts against the resistance of the blood vessels according to the formula: MAP = CO × SVR, where:

- *MAP* is mean arterial pressure, estimated by DBP + (SBP − DBP)/3 or ([2 × DBP] + SBP)/3
- *CO* is cardiac output, which is the product of stroke volume × heart rate (HR)
- *SVR* is systemic vascular resistance, estimated by [80(MAP − MVP)]/CO, where *MVP* is the mean venous pressure, which equals the mean right atrial pressure or central venous pressure (CVP).

Systemic hypertension therefore necessitates an increase in CO and/or SVR. Hypertension most typically results from an increase in SVR because even pathologic conditions that initially increase CO eventually have a normalization of CO over time with a resultant increase in SVR to sustain the hypertension.[4]

Blood Volume

Blood volume is an important physical factor that helps determine BP. Approximately two-thirds to three-quarters of the blood volume is contained within the venous capacitance vessels; the remaining one-third to one-quarter is contained within the arterial side. Arterial blood volume is determined by the difference in the blood volume ejected by the heart per unit time (CO) and the outflow through the arterial resistance vessels into the venous capacitance vessels (peripheral runoff). When CO and peripheral runoff are balanced, arterial blood volume and arterial pressure remain constant. If CO increases but peripheral runoff does not increase, then arterial blood volume increases and BP also increases.[4]

Arterial Elasticity and Compliance

Both arterial elasticity and compliance are important determinants of the increase in SBP that occurs for any given increase in blood volume. Arterial elasticity is generally inversely related to age in that younger persons have greater arterial elasticity and arterial elasticity declines with increasing age. Arterial compliance is determined by elastic properties of the large vessels and is reflective of the change in pressure that occurs with a given change in arterial volume. The greater the arterial elasticity, the smaller the increase in systolic pressure during the systolic ejection phase of the cardiac cycle; conversely, a decrease in arterial elasticity, as occurs during atherosclerotic disease, causes a greater increase in SBP during the systolic ejection phase.[4]

Autoregulation

Autoregulation is an important process that works to maintain adequate and stable blood flow during changes in BP (**Fig. 1**). Under normal conditions, tissue perfusion in the brain, heart, and kidneys remains fairly constant. In the presence of severe hypertension, especially chronic hypertension, the baseline MAP is increased and this ability to autoregulate shifts upward to protect the exposed organ from excessive pressure. Under normal circumstances, cerebral blood flow (CBF) is roughly 50 mL/100 g/min. The cerebral circulation has a physiologic protective mechanism, cerebral autoregulation, that maintains fairly constant CBF across a broad range of systemic perfusion pressures. In normal, nonhypertensive persons, cerebral autoregulation keeps CBF constant between MAPs of 60 to 120 mm Hg (see **Fig. 1**). This autoregulation is accomplished by dilatation and constriction of cerebral resistance

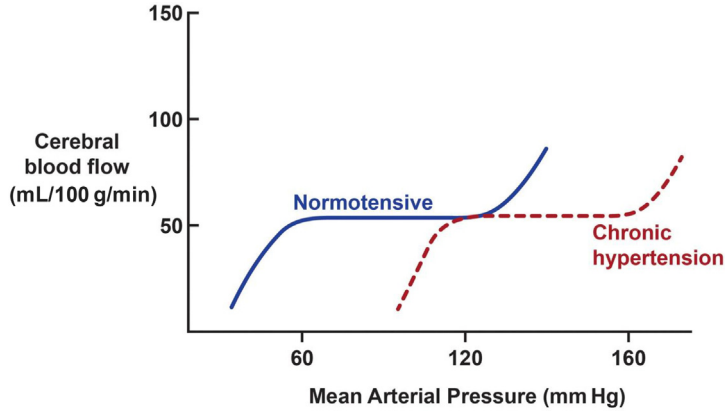

Fig. 1. Cerebral autoregulation.

vessels in response to reductions and increases in systemic BP. In chronic hypertension, when BP is poorly controlled, the entire autoregulatory curve is shifted to the right (see **Fig. 1**). The curve is shifted rightward, in part, because of the pressure-related hypertrophy of the cerebral resistance vessels, which diminishes their capacity for maximum dilation (necessary to maintain blood flow when systemic pressure decreases). The lower pressure threshold of autoregulation in both the normal and upward-shifted situations also corresponds with the threshold for hypoperfusion and this threshold is approximately 20% to 25% lower than the existing BP.[5] This physiologic observation is the rationale behind the clinical recommendation to limit the initial BP reduction to 20% to 25% of pretreatment values. If the initial BP reduction exceeded this 20% to 25% threshold, the marked reduction in organ blood flow would lead to organ ischemia and infarction.[1]

Pathophysiology Leading to a Hypertensive Crisis

The factors leading to the severe and rapid increase of BP in patients presenting with hypertensive urgency and emergency have been investigated but remain poorly understood. The events that explain the transition from hypertensive urgency to emergency resulting in abrupt end-organ damage are even less well understood. The overall acute increase in BP is thought to result from an abrupt increase in SVR that occurs from an acute increase in humoral vasoconstrictors in conjunction with a failure of the normal autoregulatory function. This abrupt increase in SVR causes an increase in mechanical stress on the vascular wall resulting in endothelial injury and vascular permeability. This vascular injury leads to the activation of platelets and the coagulation cascade, fibrin deposition, and the induction of oxidative stress and inflammatory cytokines, which result in tissue ischemia and the characteristic vascular lesion of fibrinoid necrosis of arterioles and small arteries. This cascade of events results in the propagation of an ongoing cycle of tissue ischemia, further release of vasoactive substances, and continued worsening of hypertension, which accelerates the clinical deterioration of the patient.[6–8]

Note that the rate of BP change influences the degree of end-organ damage as well as the clinical symptoms associated with a given BP increase. Chronic hypertension can, to an extent, protect end organs from abrupt increases in transmitted pressure during acute increases in BP because of arteriolar hypertrophy induced by chronic hypertension. In contrast, far smaller increases of BP can result in true hypertensive

emergencies in the setting of de novo hypertension, such as that seen during preeclampsia or acute drug toxicity.[6–8]

ACCURATE BLOOD PRESSURE ASSESSMENT

The physical examination is of utmost importance for the diagnosis of hypertensive emergency and should start with an accurate assessment of BP. Although hypertensive emergency presents a different diagnostic and treatment dilemma for clinicians compared with chronic hypertension, the BP measurement technique should essentially remain consistent for all patients to ensure accuracy. To ensure an accurate BP measurement, the following steps should be considered[4]:

- Preparing the equipment
  - Use equipment that has been validated as accurate against a mercury sphygmomanometer
  - Use equipment that has been checked for disrepair (eg, cracks or leaks in tubing, breaks in stitching, tears in fabric)
  - Use equipment that has been checked for an intact gauge (the mercury meniscus or aneroid needle is at zero)
  - Obtain appropriate cuff size by measuring circumference of the patient's arm and choosing the cuff size that corresponds with that measurement (the inflatable part of the cuff should cover 80% of the circumference of the upper arm and the cuff length should be greater than two-thirds the distance between the shoulder and elbow)
- Preparing the patient
  - Confirm that the patient has not recently consumed nicotine or caffeine
  - Have the patient sit quietly for 5 minutes before measuring BP
  - Use a sitting or semireclining position with the back supported
  - The arm should be at heart level (middle of the cuff should be at midsternum level)
  - Legs should be uncrossed with feet flat and supported on the floor or on a foot rest and not dangling from the examination table or bed
  - Clear the upper arm of any constrictive clothing (should be able to get at least 1 finger under a rolled-up sleeve)
  - Palpate the brachial artery and position center of cuff bladder over the brachial artery
- Making the BP measurement
  - Support the arm of the patient at heart level
  - For auscultation measurements:
    - Obtain an estimated systolic pressure by palpation before auscultation
    - Inflate the cuff as rapidly as possible to a maximum inflation level (20 mm Hg greater than the estimated SBP)
    - Deflate the cuff slowly at a rate of 2 to 3 mm Hg per second
    - Note the first of 2 regular beats as the SBP (simultaneous palpation helps avoid underestimating systolic pressure caused by an auscultatory gap)
    - Use the last sound heard as the DBP
    - Continue deflation for 10 mm Hg past the last sound to ensure that the sound is not a skipped beat
    - The measurement should be recorded as an even number and to the nearest 2 mm Hg (round upward)
  - Neither the patient nor observer should talk during the measurement
  - If 2 readings are measured, record the average of the readings

- Additional considerations:
  - Additional consideration should be given to assessment of BP in both upper extremities and in the lower limbs. These additional measurements could assist with the diagnosis of additional disorder: (1) thoracic aortic dissection, in which there may be a wide BP variation between arms; (2) abdominal aortic dissection, in which there may be a wide BP variation between the upper and lower extremities; and (3) coarctation of the aorta, in which there may be a difference in the SBP between the upper and lower extremities while the DBP is similar.

MAKING THE DIAGNOSIS OF HYPERTENSIVE EMERGENCY

The clinician should efficiently perform a history and physical examination and also strongly consider additional work-up if available to thoroughly evaluate the patient for evidence of end-organ damage after ensuring an accurate BP measurement has been obtained. The main areas of focus should be the neurologic, cardiovascular, and renal systems (**Fig. 2**). In addition, the patient should be asked about recent or chronic use of medications that can produce a hyperadrenergic state (eg, cocaine, amphetamines, phencyclidine) followed by a urine toxicology assessment if indicated.[9] The patient should also be questioned about recent discontinuation of medications known to cause a rebound hypertension effect (eg, clonidine and minoxidil) and also asked about compliance with any prescribed antihypertensive regimen.[10,11] Ingesting tyramine-rich foods in the setting of monoamine oxidase inhibitor use[12] or the abrupt withdrawal from alcohol[13] could also lead to a hypertensive emergency. A diagnosis of preeclampsia and eclampsia should also be considered in pregnant patients.[2,14] A future work-up for resistant hypertension including secondary causes of hypertension (**Table 2**) should also be considered if clinically indicated.

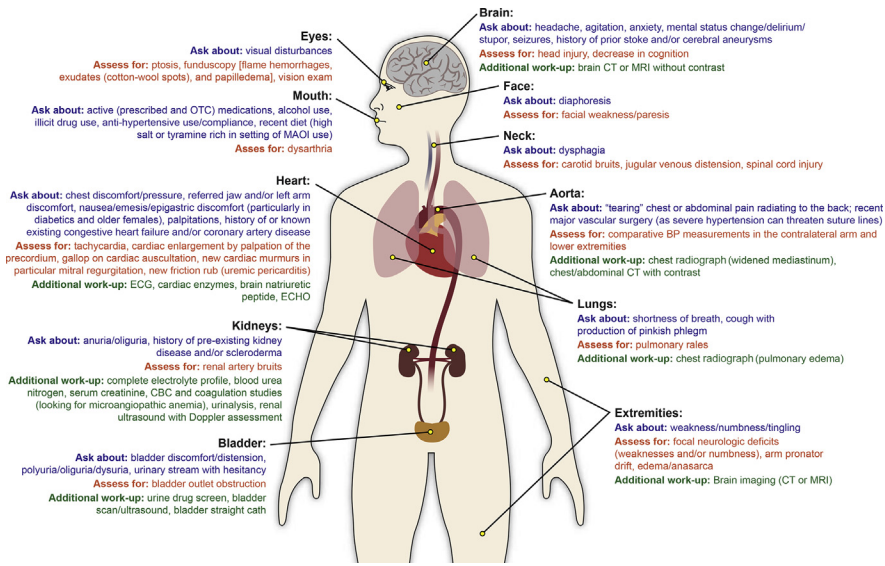

Fig. 2. Clinical assessment for end-organ damage in patients with hypertensive crisis. CBC, complete blood count; CT, computed tomography; ECG, electrocardiogram; ECHO, echocardiogram; MAOI, monoamine oxidase inhibitor; OTC, over the counter.

Table 2
Causes of secondary and resistant hypertension

System	Possible Cause
Neurologic	Head trauma, spinal cord injury, autonomic dysfunction
Cardiac/respiratory	Obstructive sleep apnea
Renal	Chronic kidney disease, renovascular hypertension
Immunologic	Scleroderma, vasculitis
Endocrine	Primary aldosteronism, pheochromocytoma, hyperthyroidism, hypothyroidism, Cushing syndrome, acromegaly, hyperparathyroidism, carcinoid tumor, congenital adrenal hyperplasia, renin-secreting tumor
Drugs inducing or exacerbating hypertension	NSAIDs, oral contraceptives, sympathomimetics, illicit drugs, glucocorticoids, mineralocorticoids, calcineurin inhibitors, erythropoietin, herbal supplements, VEGF inhibitors
Lifestyle	Excessive dietary salt intake, obesity, alcohol consumption
Pregnancy	Preeclampsia, HELLP syndrome, Eclampsia

Abbreviations: NSAIDs; nonsteroidal antiinflammatory drugs, VEGF; vascular endothelial growth factor.

Clues for Previously Uncontrolled Hypertension

Clinicians often encounter patients in the clinic or acute care setting in whom they do not have prior medical records that document important historical trends in BP. Patients are also often unaware of their prior level of BP control. However, there are clinically available clues that can be detected and that could indicate the prior level of BP control (**Box 1**). Documentation of any or all of these findings could suggest that

Box 1
Clinically available clues that indicate poorly controlled hypertension

Retinopathy

- Arteriolar narrowing, AV nicking
- Focal and general arteriolar narrowing, arteriolar silver wiring
- Hemorrhages, exudates, cotton-wool spots, papilledema, and/or microaneurysms

Cardiac examination

- Laterally displaced and/or enlarged point of maximal impulse
- S4 gallop
- Other signs of heart failure (eg, JVP, edema and rales)

Electrocardiogram

- Voltage criteria for LVH
- Inverted or biphasic P wave in precordial lead V1

Volume examination/renal (heart failure or kidney dysfunction)

- Evidence of volume overload (eg, JVP, edema, crackles)
- Increased creatinine level and proteinuria

Abbreviations: AV, arteriovenous; JVP, jugular venous pressure; LVH, left ventricular hypertrophy; PMI, point of maximal impulse; S4, fourth heart sound.

previous BP control has been less than optimal and the current increased reading may not indicate an emergency.[4]

Neurologic End-organ Damage

Severe hypertension with acute neurologic signs and/or symptoms is usually the most complicated and difficult clinical scenario because the differential diagnosis includes varied conditions, such as ischemic stroke, hemorrhagic stroke, and hypertensive encephalopathy, that have different treatments. The patient should be asked about a prior stroke and/or cerebral aneurysm as well as any existing neurologic complaints, to include headache, nausea, emesis, dysphagia, agitation, delirium, stupor, seizures, visual disturbances, focal weaknesses, or numbness/tingling. The physical examination should include looking for obvious signs of recent head injury, an assessment of cognition and speech, carotid artery auscultation, and an assessment for strength and/or sensation deficits. Facial paresis, arm drift/weakness, and abnormal speech are the three most predictive examination findings for the diagnosis of acute stroke.[15] The neurologic examination should also include a direct funduscopic examination to look for flame hemorrhages, exudates, cotton-wool spots, or papilledema. Severe autonomic dysfunction[16–18] is occasionally associated with hypertensive emergency so, if a history of conditions such as Guillain-Barré and Shy-Drager syndrome is present or there is evidence of acute spinal cord injury on examination, severe autonomic dysfunction should be considered. Additional work-up includes checking glucose level to rule out hypoglycemia and obtaining a brain computed tomography (CT) or MRI scan if readily available.

Cardiovascular End-organ Damage

The evolution of several cardiac emergencies, including acute heart failure, acute coronary syndrome, and aortic dissection, could be initiated by severe hypertension.[19–21] The patient should be asked about a prior heart disease history, to include existing congestive heart failure or coronary artery disease as well as any potential cardiac-related complaints such as shortness of breath, weakness/fatigue, irregular heartbeat, coughing with production of pinkish phlegm, chest discomfort/pressure, referred jaw/ear/arm/epigastric discomfort, nausea/emesis, or a tearing chest or abdominal pain radiating to the back. The patient should also be questioned about any recent vascular surgeries because severe hypertension can threaten suture lines. The physical examination should include looking for the presence of jugular venous distension, palpation of the precordium to assess for cardiac enlargement, pulmonary rales, gallop on cardiac auscultation, new cardiac murmurs (in particular mitral regurgitation), ascites, congestive hepatomegaly, or anasarca. Comparative BP measurements in the contralateral arm and lower extremities can also be considered if aortic dissection or coarctation is a diagnostic possibility, as described earlier. If cardiac ischemia is suspected, cardiac enzymes should be checked in addition to an electrocardiogram. Additional work-up to consider includes checking a brain natriuretic peptide level; a chest radiograph to look for cardiomegaly or pulmonary edema in addition to evidence for a widened mediastinum in aortic dissection or rib notching in aortic coarctation; and, if available, a contrasted CT could be obtained if aortic dissection is suspected.

Renal End-organ Damage

Renal damage from severe hypertension may present as acute oliguria or as any of the typical features of renal failure, including nausea/emesis, anorexia, mental status changes, uremic pericarditis, or edema. It may be difficult to determine initially whether the renal failure is an acute or a chronic process if the patient is new to

the practice; therefore, establishing the timeline for the onset of these symptoms is important to help determine whether the patient had preexisting kidney disease. Renal artery stenosis secondary to fibromuscular dysplasia in younger patients or atherosclerosis in older patients can lead to severe hypertension and renal dysfunction.[22,23] Systemic scleroderma can involve the kidneys and present with severe hypertension, hyperreninemia, azotemia, and microangiopathic anemia.[24] The physical examination should determine whether a cardiac friction rub or renal bruits are present on auscultation. If unexplained hardening or scarring of the skin is present in the setting of a history of gastroesophageal reflux, dysphasia, and attacks of discoloration of the hands and feet in response to cold, then scleroderma renal crisis can be included in the differential. Work-up to assess renal manifestations of severe hypertension should include a complete electrolyte profile, blood urea nitrogen level, serum creatinine level, coagulation studies, and a urinalysis. Doppler ultrasonography assessment to look for renal artery stenosis should also be considered if readily available.

Hormonal Causes of Hypertensive Emergency

Excess hormonal causes, such as pheochromocytoma, renin-secreting tumors, and aldosterone-secreting tumors, can result in a hypertensive emergency. In these cases, the hypertension is treated and a more thorough evaluation follows later, but these conditions are briefly discussed here for completeness. Pheochromocytomas can be familial or present as part of a multiple endocrine neoplasia syndrome so a family history of refractory hypertension or the presence of other endocrine organ cancers could provide a diagnostic clue. Recurring episodes of headache, diaphoresis, palpitations, tachycardia, and anxiety are suggestive symptoms of pheochromocytoma. The work-up should include measurement of plasma and/or urine catecholamine and metanephrine levels in addition to imaging.[25] Severe hypertension from an excess of aldosterone, whether from primary hyperaldosteronism or from a secondary cause like excess renin secretion, occasionally results in muscle weakness, spasms, and cramps from the resultant hypokalemia but most of the time is fairly asymptomatic. Secondary hyperaldosteronism can contribute to a decreased CO so there may be heart failure symptoms. An aldosterone/renin ratio is used for differentiation of primary and secondary causes and additional testing and imaging needs to be performed.[26]

TREATMENT APPROACH AND CONSIDERATIONS

The differentiation between hypertensive urgency and emergency using the encounter history, physical examination, and additional work-up options as described earlier is vital because it determines the goals of treatment, including whether or not the patient should be expediently transferred to the emergency department (ED). BP reduction for a nonemergency (increased BP with no evidence for end-organ damage) can be done over hours to days without need for a visit to the ED. Patel and colleagues[27] recently showed that the rate of major adverse cardiovascular events was low in patients who presented to an outpatient setting with hypertensive urgency and, although referral of these patients to the ED did result in more hospitalizations, ED referral was not associated with improved outcomes. There are limited definitive data with regard to hypertensive urgency and emergency management. The recent Eighth Joint National Committee (JNC 8) guidelines do not address hypertensive crisis management[28]; however, by using JNC 7 guidelines and other consensus statements, some general principles can safely be applied.

Hypertensive Urgency

Patients with nonemergent hypertension are not likely to benefit from aggressive normalization of BP and there could even be increased morbidity if rapid correction is attempted. Rapid correction potentially results in hypotension, further contributing to ischemic complications such as stroke or myocardial infarction. The goal after the exclusion of end-organ damage is to gradually reduce the BP over the next 24 to 48 hours to a more pathophysiologic safe level, generally defined as less than or equal to 160/95 mm Hg. There is rarely a compelling reason to treat hypertensive urgency with intravenous medications because safe and effective treatment can usually be accomplished using common oral medications. The JNC 7 guidelines specifically comment that, "Unfortunately, the term 'urgency' has led to overly aggressive management of many patients with severe, uncomplicated hypertension."[1]

Therapy can be initiated with fast and short-acting oral medications such as clonidine, captopril, labetalol, or nicardipine with a definitive plan to transition to longer-acting antihypertensives that are more suitable for chronic therapy.[28] Multiple dosage adjustments are likely to be needed over the following weeks to months. Hospital observation should still be considered in those patients with hypertensive urgency who remain symptomatic, those who fail to show any improvement in BP despite initial therapy, those with extreme increases of BP, and those very unlikely to obtain follow-up care. The risks of starting an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin receptor blocker (ARB) acutely in patients with chronic kidney disease need to be carefully considered if the likelihood of the patient obtaining appropriate follow-up care seems low. A notable exception is in scleredema renal crisis, which is a hypertensive emergency and the initiation of an ACE inhibitor is the recommended therapy regardless of the serum creatinine measurement.[24]

Hypertensive Emergency

Patients with evidence or high suspicion for end-organ damage should be expediently referred from the outpatient setting to the ED because a rapid BP reduction over minutes to hours is often indicated to prevent additional end-organ damage. This need for immediate but controlled BP management usually indicates the requirement for monitoring in a critical care setting; however, the initiation of goal-directed therapy in the clinic should be strongly considered because delay could result in end-organ damage progression. Several rapid-acting intravenous antihypertensive agents are available for the treatment of hypertensive emergencies and the choice of which agent to use is mainly related to the clinical manifestations of end-organ damage. Clinicians should be knowledgeable of frequently used medications, potential clinical scenarios in which these medications can be used most effectively, and the side effect profiles/risks for each of these medications in order to successfully initiate therapy (**Table 3**). No large clinical trials have been conducted to define specific treatment goals or to compare the efficacy of the various medications available to treat hypertensive emergency. In general, the BP should be reduced no more than 25% within the first hour and then to 160/100 to 110 mm Hg within 2 to 6 hours.[1] An alternative and more conservative approach is to reduce the BP approximately 10% in the first few hours and then by no more than 25% during the first 24 hours.

Two notable exceptions to this general approach are patients with an aortic dissection and those with an ischemic stroke. The estimated acute mortality for an aortic dissection is approximately 40%. The SBP goal for an aortic dissection is less than 120 mm Hg in addition to a heart rate goal of less than 60 beats per minute. These goals should be obtained quickly and then BP should ideally be titrated as low as

Table 3
Common presenting scenarios of hypertensive emergency with treatment considerations

Clinical Scenario	BP Reduction[a]	IV Drug Options	Additional Considerations
Acute ischemic stroke	tPA candidate: ≤185/110 mm Hg tPA not planned: lower if BP ≥220/120 mm Hg	Labetalol, esmolol, nicardipine	Avoid nitroprusside; can lead to intracranial edema
Acute hemorrhagic stroke	Reduce to <180/105 mm Hg to avoid hematoma expansion and edema	Labetalol, esmolol, nicardipine	Avoid nitroprusside; can lead to intracranial edema
Hypertensive encephalopathy	Reduce BP 20%–25% to reduce intracranial pressure	Labetalol, esmolol, nicardipine	Avoid nitroprusside; can lead to intracranial edema
Acute heart failure	Reduce BP until resolution of acute pulmonary edema	Nitroglycerine, nitroprusside, furosemide	β-Blocker or calcium-channel blocker use could cause exacerbation of symptoms
Acute coronary syndrome	Reduce BP to reduce cardiac workload and improve coronary perfusion	Nitroglycerine, nitroprusside, labetalol, metoprolol, esmolol, nicardipine	Consider type A dissection as cause of acute coronary syndrome; avoid selective β-blockers if cocaine abuse suspected
Acute aortic dissection	Reduce BP to <120/80 mm Hg (lower if tolerable) and HR to <60 bpm in order to reduce wall shear stress	Labetalol, nitroprusside, nicardipine	Avoid β-blockers if severe aortic regurgitation is noted
Acute renal failure	Reduce pressure in the kidney	Nitroprusside, nicardipine	Caution with ACE inhibitors and ARBs unless scleredema renal crisis suspected; monitor for cyanide toxicity if nitroprusside is used
Eclampsia	Reduce intracranial pressure and maintain placental perfusion	Labetalol, hydralazine, magnesium	Definitive treatment is delivery of fetus
Sympathetic crisis	Reduce BP until symptom resolution	Phentolamine, nitroglycerine, nicardipine, labetalol	Avoid β-blocker monotherapy (except for labetalol)
Pheochromocytoma	Reduce BP until symptom resolution	Labetalol, phentolamine	Avoid β-blocker monotherapy (except for labetalol)

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; IV, intravenous; tPA, tissue plasminogen activator.
[a] General rule: BP should be reduced no more than 25% within the first hour and then to 160/100 to 110 mm Hg within 2 to 6 hours.

end organs allow, with attention to measuring BP in both arms.[20] Ischemic strokes represent approximately 85% of total stroke events. The initial assessment for patients suspected of having an ischemic stroke is to determine whether or not the patient is a candidate for tissue plasminogen activator (tPA), because this decision determines the most appropriate BP management. If the patient is a tPA candidate, the BP should be promptly reduced to less than or equal to 185/110 mm Hg before administration of tPA and maintained at less than or equal to 180/105 mm Hg for at least 24 hours after administration. If the patient is not a tPA candidate, a BP greater than or equal to 220/120 mm Hg should be safely decreased; however, for an SBP between 140 and 220 mm Hg, there is no definitive proof that reducing the BP is beneficial unless the patient has another clear indication to decrease the BP.[29]

Hypertensive emergencies can also arise from states of catecholamine excess such as a pheochromocytoma,[25] interactions between monoamine oxidase inhibitors and sympathomimetic drugs,[12] or cocaine use.[9] The sole use of β-blockers in these situations could result in unopposed alpha-adrenergic stimulation and additional peripheral vasoconstriction worsening the hypertension. The use of a ganglion blocking agent such as intravenous phentolamine (or in less urgent situations oral phenoxybenzamine) must therefore precede the use of a pure β-blocker in these situations. Alternatively, labetalol, which has combined alpha-adrenergic and beta-adrenergic blocking properties, is safe and effective in these situations.[26] Rebound hypertension following sudden discontinuation of high-dose clonidine (>1.2 mg/d) is also a state of catecholamine excess, and although this situation also responds quickly to combined alpha-adrenergic and beta-adrenergic blockade, resumption of clonidine is another simple alternative.[10]

Other supplemental treatment options, in addition to reducing the BP, should also be considered while awaiting patient transfer to the ED. Intravenous loop diuretics (furosemide, bumetanide, or torsemide) should be considered if volume overload is suspected. Oxygen should be administered to treat hypoxemia. Morphine has vasodilator properties and may reduce preload and the sensation of air hunger if needed. Noninvasive ventilation may be used to relieve symptoms in patients with pulmonary edema and severe respiratory distress or in those patients who fail to improve with initial pharmacologic therapy.

SUMMARY

Hypertensive emergency results from an increased BP (generally greater than 180 mm Hg systolic and/or 120 mm Hg diastolic) that is associated with acute and/or evolving end-organ damage primarily to the neurologic, cardiovascular, and renal systems. Properly diagnosing hypertensive emergency and differentiating it from hypertensive urgency and/or uncontrolled chronic hypertension using accurate BP measurement techniques, a pertinent patient interview, physical examination, and additional work-up if available is essential to appropriate patient triage and initial treatment. A diagnosis of hypertensive emergency requires urgent transfer to an ED or inpatient setting but initiation of goal-directed therapy in the clinic should not be delayed because delay could result in end-organ damage progression. Several antihypertensive agents are available and the choice of which agent to use is mainly dictated by the type of end-organ damage present, although no large clinical trials have been conducted to compare efficacy or define specific treatment goals. The most efficient way to prevent further episodes of hypertensive emergency in each patient is to ensure that the patient has close follow-up on discharge with the primary care provider.

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