Approximately one in three US adults, or about 75 million people, have high blood pressure (BP), which has been defined as a BP of 140/90 mm Hg or higher. Unfortunately, only about half (54%) of those affected have their condition under optimal control. From an epidemiologic standpoint, hypertension has the distinction of being the most common chronic condition in the US, affecting about 54% of persons ages 55 to 64 and about 73% of those 75 and older. It is the number one reason patients schedule office visits with physicians; it accounts for the most prescriptions; and it is a major risk factor for heart disease and stroke, as well as a significant contributor to mortality throughout the world.

HYPERTENSIVE URGENCY VS EMERGENCY

Hypertensive urgencies and emergencies account for approximately 27% of all medical emergencies and 2% to 3% of all annual visits to the emergency department (ED). Hypertensive urgency, or severe asymptomatic hypertension, is a common complaint in urgent care clinics and primary care offices as well. It is often defined as a systolic BP (SBP) of ≥ 160 mm Hg and/or a diastolic BP (DBP) ≥ 100 mm Hg with no associated end-organ damage. Patients may experience hypertensive urgency if they have been noncompliant with their antihypertensive drug regimen; present with pain; have white-coat hypertension or anxiety; or use recreational drugs (eg, sympathomimetics).

Alternatively, hypertensive emergency, also known as hypertensive crisis, is generally defined as elevated BP > 180/120 mm Hg. Equally important, it is associated with signs, symptoms, or laboratory values indicative of target end-organ damage, such as cerebrovascular accident, myocardial infarction (MI), aortic dissection, acute left ventricular failure, acute pulmonary edema, acute renal failure, acute mental status changes (hypertensive encephalopathy), and eclampsia.

Determining appropriate management for patients with hypertensive urgency is controversial among clinicians. Practice patterns range from full screening and “rule-outs”—with prompt initiation of antihypertensive agents, regardless of whether the patient is symptomatic—to sending the patient home with minimal screening, laboratory testing, or treatment.

This article offers a guided approach to managing patients with hypertensive urgency in a logical fashion, based on risk stratification, thereby avoiding both extremes (extensive unnecessary workup or discharge...
without workup resulting in adverse outcomes). It is vital to differentiate between patients with hypertensive emergency, in which BP should be lowered in minutes, and patients with hypertensive urgency, in which BP can be lowered more slowly.12

PATHOPHYSIOLOGY
Normally, when BP increases, blood vessel diameter changes in response; this autoregulation serves to limit damage. However, when BP increases abruptly, the body’s ability to hemodynamically calibrate to such a rapid change is impeded, thus allowing for potential end-organ damage.5,12 The increased vascular resistance observed in many patients with hypertension appears to be an autoregulatory process that helps to maintain a normal or viable level of tissue blood flow and organ perfusion despite the increased BP, rather than a primary cause of the hypertension.13

The exact physiology of hypertensive urgencies is not clearly understood, because of the multifactorial nature of the process. One leading theory is that circulating humoral vasoconstrictors cause an abrupt increase in systemic vascular resistance, which in turn causes mechanical shear stress to the endothelial wall. This endothelial damage promotes more vasoconstriction, platelet aggregation, and activation of the renin-angiotensin-aldosterone system, which thereby increases release of angiotensin II and various cytokines.14

HISTORY AND PHYSICAL
A detailed medical history is of utmost importance in distinguishing patients who present with asymptomatic hypertensive urgency from those experiencing a hypertensive emergency. In addition, obtain a full medication list, including any nutritional supplements or illicit drugs the patient may be taking. Question the patient regarding medication adherence; some may not be taking antihypertensive agents as prescribed or may have altered the dosing frequency in an effort to extend the duration of their prescription.5,8 Table 1 (page 42) lists pertinent questions to ask at presentation; the answers will dictate who needs further workup and possible admission as well as who will require screening for end-organ damage.7

The physical exam should focus primarily on a thorough cardiopulmonary and neurologic examination, as well as funduscopic examination, if needed. A complete set of vital signs should be recorded upon the patient’s arrival to the ED or clinic and should be repeated on the opposite arm for verification. Beginning with the eyes, conduct a thorough funduscopic examination to evaluate for papilledema or hemorrhages.5 During the cardiopulmonary exam, attention should be focused on signs of congestive heart failure and/or pulmonary edema, such as increased jugular vein distension, an S3 gallop, peripheral edema, and pulmonary rales. The neurologic exam is essential in evaluating for cerebrovascular accident,
transient ischemic attack, or intracranial hemorrhage. A full cranial nerve exami-
nation is necessary, in addition to motor and sensory testing, at minimum.5,9

RISK STRATIFICATION
According to the 2013 Task Force of the Eu-
ropean Society of Hypertension (ESH) and
the European Society of Cardiology (ESC),
several risk factors contribute to overall car-
diovascular risk in asymptomatic patients
presenting with severe hypertension (see
Table 2).8 This report has been monumental
in linking grades of hypertension directly
to cardiovascular risk factors, but it differs
from that recently published by the Eighth
Joint National Committee (JNC 8), which
offers evidence-based guidelines for the
management of high BP in the general pop-
ulation of adults (with some modifications
for individuals with diabetes or chronic kid-
ney disease or of black ethnicity).15

According to the ESH/ESC study, patients
with one or two risk factors who have grade
1 hypertension (SBP 140-159 mm Hg) are
at moderate risk for cardiovascular disease
(CVD) and patients with grade 2 (SBP 160-
179 mm Hg) or grade 3 (SBP ≥ 180 mm Hg)
hypertension are at moderate-to-high risk
and high risk, respectively.8 Patients with
three or more risk factors, or who already
have end-organ damage, diabetes, or chron-
ic kidney disease, enter the high-risk catego-
ry for CVD even at grade 1 hypertension.8

These cardiovascular risk factors can and
should be used as guidelines for deciding
who needs further screening and who may
have benign causes of severe hypertension
(e.g., white-coat hypertension, anxiety) that
can be managed safely in an outpatient set-
ing. In the author’s opinion, patients with
known cardiovascular risk factors, those
with signs or symptoms of end-organ dam-
age, and those with test results suggestive of
end-organ damage should have a more im-
mediate treatment strategy initiated.

Numerous observational studies have
shown a direct relationship between sys-
temic hypertension and CVD risk in men
and women of various ages, races, and eth-
nicities, regardless of other risk factors for
CVD.12 In patients with diabetes, uncon-
trolled hypertension is a strong predictor
of cardiovascular morbidity and mortality
and of progressive nephropathy leading to
chronic kidney disease.8

SCREENING
Results from the following tests may pro-
vide useful clues in the workup of a patient
with hypertensive urgency.

Basic metabolic panel. Many EDs and
primary care offices offer point-of-care test-
ing that can typically give a rapid (< 10 min)
result of a basic metabolic panel. This use-
ful, quick screening tool can identify renal

### TABLE 1
Vital Questions to Ask When Obtaining History

<table>
<thead>
<tr>
<th>Question</th>
<th>Differential diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have a history of high BP?</td>
<td>Anxiety, medication noncompliance, pain, white-coat HTN</td>
</tr>
<tr>
<td>Do you have any chest pain or syncope?</td>
<td>MI, pulmonary embolism, thoracic aortic dissection</td>
</tr>
<tr>
<td>Are you experiencing any shortness of breath?</td>
<td>HF, MI, pulmonary edema, pulmonary embolism</td>
</tr>
<tr>
<td>Have you had any loss of vision or eye pain?</td>
<td>Acute angle-closure glaucoma, CVA/TIA, papilledema, retinal hemorrhages</td>
</tr>
<tr>
<td>Have you had a severe headache, focal weakness or numbness, speech difficulties, or trouble walking?</td>
<td>CVA, ICH, TIA</td>
</tr>
<tr>
<td>Are you using any recreational drugs?</td>
<td>Cocaine, methamphetamines, or other sympathomimetics</td>
</tr>
<tr>
<td>Have you had multidrug-resistant HTN?</td>
<td>Cushing syndrome, end-stage renal disease, hyperaldosteronism, pheochromocytoma, renal artery stenosis, thyroid storm</td>
</tr>
<tr>
<td>Have you had a positive urine pregnancy test at home or in the ED/clinic?</td>
<td>Preeclampsia/eclampsia</td>
</tr>
</tbody>
</table>

Abbreviations: BP, blood pressure; CVA, cerebrovascular accident; ED, emergency department; HF, heart failure; HTN, hypertension; ICH, intracranial hemorrhage; MI, myocardial infarction; TIA, transient ischemic attack.

failure due to chronic untreated hypertension, acute renal failure, or other disease states that cause electrolyte abnormalities such as hyperaldosteronism (hypertension with hypokalemia) or Cushing syndrome (hypertension with hypernatremia and hyperkalemia).7

**Cardiac enzymes.** Measurement of cardiac troponins (T or I) may provide confirmatory evidence of myocardial necrosis within two to three hours of suspected acute MI.16,17 These tests are now available in most EDs and some clinics with point-of-care testing. A variety of current guidelines advocate repeat cardiac enzyme measurements at various time points, depending on results of initial testing and concomitant risk factors. These protocols vary by facility.

**ECG.** Obtaining an ECG is another quick, easy, and useful way to screen patients presenting with severe hypertensive urgency. Evidence of left ventricular hypertrophy suggests an increased risk for MI, stroke, heart failure, and sudden death.7,18-20 The Cornell criteria of summing the R wave in aVL and the S wave in V5, with a cutoff of 2.8 mV in men and 2.0 mV in women, has been shown to be the best predictor of future cardiovascular mortality.7 While an isolated finding of left ventricular hypertrophy on an ECG—in and of itself—may have limited value for an individual patient, this finding coupled with other risk factors may alter the provider’s assessment.

**Chest radiograph.** A chest radiograph can be helpful when used in conjunction with physical exam findings that suggest pulmonary edema and cardiomegaly.7 Widened mediastinum and tortuous aorta may also be evident on chest x-ray, necessitating further workup and imaging.

**Urinalysis.** In a patient presenting with asymptomatic hypertensive urgency, a urine dipstick result that shows new-onset proteinuria, while not definitive for diagnosis of nephrotic syndrome, may certainly prove helpful in the patient’s workup.5,13

**Urine drug screen.** In patients without a history of hypertension who present with asymptomatic hypertensive urgency, the urine drug screen may ascertain exposure to cocaine, amphetamine, or phencyclidine.

### TABLE 2
**Cardiovascular Risk Factors for Asymptomatic Hypertension**

| Male sex |
| Age: men ≥ 55 y, women ≥ 65 y |
| Tobacco use, particularly cigarettes |
| Dyslipidemia |
| Diabetes |
| Obesity or metabolic syndrome |
| Established CVD or CAD (eg, CABG, CVA, MI, PCI) |
| Family history of premature CVD (men < 55 y, women < 65 y) |
| Estimated GFR < 60 mL/min |

Abbreviations: CABG, coronary artery bypass graft; CAD, coronary artery disease; CVA, cerebrovascular accident; CVD, cardiovascular disease; GFR, glomerular filtration rate; MI, myocardial infarction; PCI, percutaneous coronary intervention.

Source: Mancia et al. J Hypertens. 2013.8

**Pregnancy test.** A pregnancy test is essential for any female patient of childbearing age presenting to the ED, and a positive result may be concerning for preeclampsia in a hypertensive patient with no prior history of the condition.7

### TREATMENT

Knowing who to treat and when is a vast area of debate among emergency and primary care providers. Patients with hypertension who have established risk factors are known to have worse outcomes than those who may be otherwise healthy. Some clinicians believe that patients presenting with hypertensive urgency should be discharged home without screening and/or treatment. However, because uncontrolled severe hypertension can lead to acute complications (eg, MI, cerebrovascular accident), in practice, many providers are unwilling to send the patient home without workup.12 The patient’s condition must be viewed in the context of the entire disease spectrum, including risk factors.

The Figure (page 44) offers a disposition pathway of recommendations based on risk factors.
stratification as well as screening tools for some of the less common causes of hypertensive urgency. Regardless of the results of screening tests or the decision to treat, affected patients require close primary care follow-up. Many of these patients may need further testing and careful management of their BP medication regimen.
How to treat
For patients with severe asymptomatic hypertension, if the history, physical, and screening tests do not show evidence of end-organ damage, BP can be controlled within 24 to 48 hours.5,10,11,21 In adults with hypertensive urgency, the most reasonable goal is to reduce the BP to ≤ 160/100 mm Hg; however, the mean arterial pressure should not be lowered by more than 25% within the first two to three hours.13

Patients at high risk for imminent neurovascular, cardiovascular, renovascular, or pulmonary events should have their BP lowered over a period of hours, not minutes. In fact, there is evidence that rapid lowering of BP in asymptomatic patients may cause adverse outcomes.6 For example, in patients with acute ischemic stroke, increases in cerebral perfusion pressure promote an increase in vascular resistance—but decreasing the cerebral perfusion pressure abruptly will thereby decrease the cerebral blood flow, potentially causing cerebral ischemia or a worsening of the stroke.9,14

Treatment options
A broad spectrum of therapeutic options has proven helpful in lowering BP over a short period of time, including oral captopril, clonidine, hydralazine, labetalol, and hydrochlorothiazide (see Table 3).7,9,12,15 Nifedipine is contraindicated because of the abrupt and often unpredictable reduction in BP and associated myocardial ischemia, especially in patients with MI or left ventricular hypertrophy.14,22,23 In cases of hypertensive urgency secondary to cocaine abuse, benzodiazepines would be the drug of choice and β-blockers should be avoided due to the risk for coronary vasoconstriction.7
For patients with previously treated hypertension, the following options are reasonable: Increase the dose of the current antihypertensive medication; add another agent; reinstate prior antihypertensive medications in nonadherent patients; or add a diuretic.

In patients with previously untreated hypertension, no clear evidence supports using one particular agent over another. However, initial treatment options that are generally considered safe include an ACE inhibitor, an angiotensin receptor blocker, a calcium channel blocker, or a thiazide diuretic. A few examples of medications within these categories include lisinopril (10 mg PO qd), losartan (50 mg PO qd), amloclidine (2.5 mg PO qd), or hydrochlorothiazide (25 mg PO qd).

Close follow-up is essential when an antihypertensive medication is started or reinstated. Encourage the patient to reestablish care with their primary care provider (if you do not fill that role). You may need to refer the patient to a new provider or, in some cases, have the patient return to the ED for a repeat BP check.

CONCLUSION
The challenges of managing patients with hypertensive urgency are complicated by low follow-up rates with primary physicians, difficulty in obtaining referrals and follow-up for the patient, and hesitancy of providers to start patients on new BP medications. This article clarifies a well-defined algorithm for how to screen and risk-stratify patients who present to the ED or primary care office with hypertensive urgency.

REFERENCES