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Hypertension In The Emergency Department: Treat Now, Later, Or Not At All

It's Friday afternoon, and your first patient of the shift is a 58-year-old man with a left temporal headache and a BP reading of 146/96 mm Hg. He has no history of hypertension, and the headache was neither sudden in onset nor the worst of his life. Results of his physical examination are completely normal. Temporal arteritis is a consideration; while developing your management strategy, you question the need to order ancillary tests for end-organ damage from his hypertension.

A couple of patients later, you see a 71-year-old woman sent in by her primary care physician for an evaluation of elevated BP found during a routine preoperative physical. Her triage BP reading is 190/110 mm Hg. She has no symptoms, but her BP is high enough to make you wonder how emergently it needs to be treated.

Your next patient is a 96-year-old woman who takes diltiazem and furosemide and presents with pulmonary edema. Her triage BP reading is 220/130 mm Hg, and her respiratory rate is 28 breaths per minute while sitting in a tripod position. As you are waiting for portable radiography, the nurse asks how you want to manage the patient's BP.

The origin of hypertension can be multifactorial, making its diagnosis and management in the emergency department (ED) topics of considerable discussion. **Table 1 (see page 2)** presents general definitions of hypertension; however, these definitions can be both helpful and misleading in the ED.[1] **Table 2 (see page 2)** presents central questions that should be asked when a patient is evaluated

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CME Objectives

Upon completing this article, you should be able to:

1. Appropriately distinguish hypertensive urgency and emergency.
2. Determine an appropriate diagnostic workup, if any, for a patient with hypertension.
3. Choose appropriate immediate oral or intravenous medications for the patient with hypertension.
4. Identify and appropriately treat hypertension in patients with special circumstances including stroke, aortic dissection, and cocaine use.

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for hypertension in the ED. The sense of impending danger associated with hypertension (ie, use of words such as "urgency" and "crisis") may lead practitioners to aggressively treat patients. However, this approach can be detrimental if it leads to overtreatment, thus converting patients from a stable, asymptomatic, hypertensive state to an unstable, symptomatic, normotensive or hypotensive state.

This issue of *Emergency Medicine Practice* reviews the current evidence related to the diagnosis and management of hypertension with a focus on issues related to ED clinical decisionmaking.

Critical Appraisal Of The Literature

The literature review was launched with an Ovid MEDLINE® (www.ovid.com) search of articles on hypertension published from January 1998 to April 2008. Keywords included *hypertensive urgency, hypertensive emergency, perioperative hypertension,* and *emergency department hypertension.* More than 330 articles were reviewed, and additional references were identified from the bibliographies. The Cochrane Reviews database was also searched and yielded 117 reviews and protocols related to hypertension; however, only 5 were considered relevant for this article.

Table 1. Definitions Of Hypertension[1]

Hypertensive emergency
Blood pressure > 180/120 mm Hg with impending or progressive target organ dysfunction

Hypertensive urgency
Blood pressure > 180/120 mm Hg without impending or progressive target organ dysfunction

Hypertensive crisis
A hypertensive emergency or urgency

Mean arterial pressure
Average blood pressure reading over 1 cardiac cycle; can be calculated as [systolic blood pressure + (2 x diastolic blood pressure)] ÷ 3

Essential hypertension
Hypertension without a specific secondary cause

Secondary hypertension
Hypertension related to an underlying pathologic process, eg, adrenal disease; renal disease; or drug effects, interactions, or withdrawal

Table 2. Central Questions When Treating Hypertension In The ED

- Are ED measurements of blood pressure accurate in determining if the patient is truly hypertensive?
- Is there any evidence that the patient's current blood pressure is contributing to the acute condition?
- Is there any evidence that the patient's current blood pressure is contributing to active end-organ damage?
- How aggressively should the patient's blood pressure be managed?
- What is the appropriate disposition and follow-up?

A supplement released in March 2008 by the *Annals of Emergency Medicine,* "Management of Hypertension and Hypertensive Emergencies in the Emergency Department: The EMCREG-International Consensus Panel Recommendations," was also included.[2] (Note: This consensus document was indirectly funded through the pharmaceutical industry.)

A search of the National Guidelines Clearinghouse (www.guidelines.gov) produced 400 guidelines for the management of hypertension, including the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7)[3] and European guidelines for arterial hypertension.[4] The JNC 7 is a consensus panel convened to study the diagnosis and long-term management of hypertension based on literature published from 1997 through 2003, including data from the Framingham Heart Study, the National Ambulatory Medical Care Survey, and the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). The JNC 7 report gives similar guidelines and recommendations issued by the European Society of Hypertension, the Canadian Hypertension Education Program, and the British Hypertension Society.[4-6]

These guidelines focus on chronic disease in general and do not address immediate evaluation and management in any depth. For example, JNC 7 only briefly mentions hypertensive urgency or emergency:

> "Patients with marked BP elevations and acute target-organ damage (eg, encephalopathy, myocardial infarction, unstable angina, pulmonary edema, eclampsia, stroke, head trauma, life-threatening arterial bleeding, or aortic dissection) require hospitalization and parenteral drug therapy. Patients with markedly elevated BP but without acute target-organ damage usually do not require hospitalization, but they should receive immediate combination oral antihypertensive therapy. They should be carefully evaluated and monitored for hypertension-induced heart and kidney damage and for identifiable causes of hypertension."[3]

This statement is supported by reference to JNC 6; within JNC 6, the same statement is based on expert opinion.[7]

A review of the literature revealed only 1 guideline—from the American College of Emergency Physicians (ACEP) Clinical Policies Subcommittee—that is specifically directed at ED management of hypertension.[8] Most of the literature on ED management is derived from review articles or case series extrapolating from outcome data for long-term hypertension or from small clinical trials involving special situations such as postoperative hypertension, aortic dissection, stroke, or cocaine use. Therefore, by default, the majority of ED-related management recommendations are based on consensus opinion.

Abbreviations Used In This Article

ALLHAT: Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial
ACC: American College of Cardiology
ACE: Angiotensin-converting enzyme
ACEP: American College of Emergency Physicians
AHA: American Heart Association
AHRQ: Agency for Health Research and Quality
ATACH: Antihypertensive Treatment of Acute Cerebral Hemorrhage study
BMP: Basic metabolic panel
BNP: Brain (or B-type) natriuretic peptide
BP: Blood pressure
CARDIA: Coronary Artery Risk Development in Young Adults study
CBC: Complete blood cell count
CHF: Congestive heart failure
CPAP: Continuous positive airway pressure
CXR: Chest x-ray
DBP: Diastolic blood pressure
ECG: Electrocardiogram
ED: Emergency department
EMCREG: Emergency Medicine Cardiac Research and Education Group
ESI: Emergency Severity Index
GFR: Glomerular filtration rate
INTERACT II: Intensive Blood Pressure Reduction in Acute Cerebral Haemorrhage study
IV: Intravenous
JNC: Joint National Committee [on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure]
LAH: Left atrial hypertrophy
LV: Left ventricle
LVH: Left ventricular hypertrophy
MAP: Mean arterial pressure
MI: Myocardial infarction
NSTEMI: Non-ST-segment elevation myocardial infarction
PCP: Phencyclidine
SBP: Systolic blood pressure
SL: Sublingual
SLE: Systemic lupus erythematosus
TIA: Transient ischemic attack
tPA: Tissue plasminogen activator
UA: Urinalysis
US: United States

Epidemiology

Stage I hypertension is currently defined by JNC 7 as a systolic blood pressure (SBP) of 140 to 159 mm Hg or a diastolic blood pressure (DBP) of 90 to 99 mm Hg. *Stage II hypertension* is defined as an SBP greater than or equal to 160 mm Hg or a DBP greater than or equal to 100 mm Hg.[1] A 2003-2004 population-based survey involving 4872 adults revealed that the prevalence of chronic hypertension in the United States (US) was 29%, with hypertension adequately controlled in only 37% of hypertensive patients.[9] An independent risk factor for hypertensive urgency or emergency is poor adherence to a medical regimen.[10] African American race and male sex are also risk factors.[11] An observational study of 449 patients found that hypertensive urgencies are 4 times as common as emergencies. The most frequent end-organ effects were stroke (29%), pulmonary edema (23%), encephalopathy (18%), congestive heart failure (CHF) (15%), and myocardial infarction (MI) or unstable angina (13%).[11]

Essential hypertension is much more common than secondary hypertension. A prospective Japanese study of 1020 patients identified secondary causes of hypertension in 9% of the patients.[12] Similarly, a Scottish retrospective medical record review of 3783 patients identified a secondary cause in only 8% of patients.[13]

A prospective multisite study of triage vital signs for 1396 patients found 20% were hypertensive on presentation to the ED, with 6% having an SBP greater than 180 mm Hg or a DBP greater than 110 mm Hg.[14] A second single-site, prospective screening study involving 765 participants in the United Kingdom found that 28% of ED patients were hypertensive.[15] The rate of hypertension is further elevated in patients presenting to the ED with diseases such as stroke or aortic dissection. A retrospective review of the National Hospital Ambulatory Medical Care Survey involving 563,704 patients presenting with stroke showed that 69% presented with hypertension.[16] A multicenter, multinational trial including 464 patients with aortic dissection found that 49% had an SBP of 150 mm Hg or higher.[17]

Pathophysiology

Hypertension is classified as either essential (primary) or secondary. If a specific cause such as primary aldosteronism, Cushing syndrome, pheochromocytoma, or renovascular hypertension is identified, hypertension is categorized as secondary; about 10% of cases fall into this category.[12] The remaining 90% are classified as essential because the initial pathophysiological mechanism is unclear.[16,17]

Essential hypertension progresses in varying degrees through interactions among the cardiovascular, renal, and central nervous systems.[11,18] In the presence of hypertension, cardiac remodeling occurs secondary to increased afterload. Antihypertensive medications that can alter this remodeling include angiotensin-converting enzyme (ACE) inhibitors and β-blockers. Cardiac remodeling can be a normal physiologic response, as shown by postnatal development of the left ventricle outpacing development of the right because of increasing systemic vascular

resistance. Left ventricular hypertrophy (LVH) is present in 19% of men and 24% of women, as demonstrated by echocardiographic criteria; interestingly, only 1.3% of both men and women have LVH by electrocardiogram (ECG) criteria.[19] Unfortunately, this ability to remodel can become maladaptive. There is some evidence that LVH may precede the development of hypertension.[20,21] Patients who meet ECG criteria for LVH also face an increased risk of coronary artery disease, heart failure, ventricular arrhythmias, cerebrovascular disease, and sudden death.[22] The increased risk of sudden death is theorized from a combination of increased myocardial oxygen consumption, compression of endocardial capillaries, and decreased ability to dilate veins, which reduce perfusion. In addition, action potential prolongation, altered repolarization, and excessive myocardial fiber stretching may potentiate ventricular arrhythmias.[23]

Atrial fibrillation occurs more often in patients with hypertension and is associated with higher risks of stroke, heart failure, and valvular heart disease.[24] Hypertension has been associated with a 70% increase in risk for atrial fibrillation, and this increased risk remained after adjustments for age, sex, and associated conditions. A prospective observational study of 4731 patients illustrated a 39% increase in risk for atrial fibrillation for every 5-mm increment in atrial enlargement.[24] Atrial fibrillation increased the risk for stroke 3- to 5-fold after adjustments for other risk factors. Finally, the presence of atrial fibrillation doubles the all-cause mortality rate.[24] Management of atrial fibrillation requires a rate control agent such as non-dihydropyridine calcium channel blocker (eg, diltiazem) and ß-blockers.

Hypertension can also increase the frequency of MI. A prospective population-based study involving approximately 4902 adults older than 64 years showed an increased rate of MI from 10 to 22 per 1000-person-years and an in-total mortality from 22 to 29 per 1000-person-years when SBP increased from less than 120 mm Hg to 141-159 mm Hg. Increases in systolic BP above 159 were associated with a further 30% to 40% relative increase in rates of MI and mortality.[25]

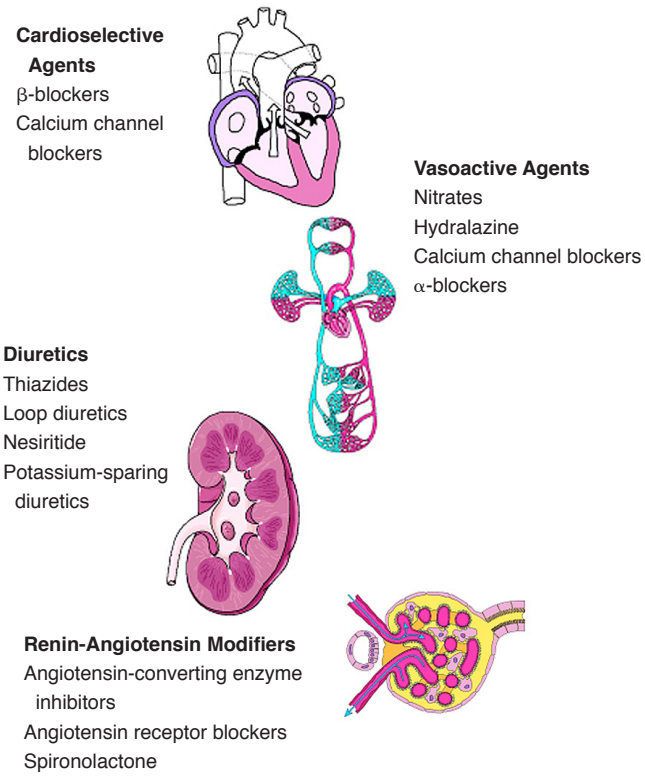
The renal system affects hypertension through control of both total intravascular volume and peripheral vascular resistance via the renin-angiotensin-aldosterone system. Angiotensin II stimulates aldosterone, the antidiuretic hormone, and the sympathetic nervous system. A study of 55 rats showed that plasma renin is substantially elevated when 1 renal artery is clipped, illustrating the pathophysiology of renovascular hypertension. By contrast, there was considerable volume overload and a lesser degree of renin elevation when only 1 kidney was present, which more closely models the decreased filtration ability in chronic kidney disease.[26] Drugs that interact with the renin-angiotensin-aldosterone axis include ACE inhibitors, angiotensin receptor blockers, and spironolactone.

The natriuretic peptides—most notably brain (or B-type) natriuretic peptide (BNP), which is derived from ventricular myocardium—act in multiple ways to decrease BP. Brain (or B-type) natriuretic peptide promotes diuresis by stimulating salt wasting and acts as a vasodilator by activating the guanylate cyclase pathway. In addition, it inhibits the renin-angiotensin system. Nesiritide, or exogenous BNP, was initially proposed as a substitute for nitroglycerin and diuretics in the treatment of decompensated heart failure, but superiority in emergent management has not been demonstrated.[27] Some conflicting evidence implies an increased risk of short-term death in patients treated with nesiritide.[28,29]

Autoregulation in the central nervous system acts to maintain a constant cerebral blood flow regardless of perfusion pressures. This effect is achieved by cerebral vasodilation when systemic BP is low and by vasoconstriction when systemic BP is high.[30] In addition, the sympathetic nervous system can adjust cardiac output with long-term consequences. For example, the Coronary Artery Risk Development in Young Adults (CARDIA) observational study showed a correlation between increased heart rate and hypertension over 10 years, independent of baseline BP readings.[31]

In summary, hypertensive emergencies and

Figure 1. Mechanisms Of Antihypertensive Medications



urgencies can result from either essential hypertension or secondary etiologies, and treatment should be tailored to the underlying pathophysiology. **(See Figure 1.)** Hypertensive emergencies or urgencies generally occur after an abrupt increase in systemic vascular resistance, resulting in endothelial injury, fibrin deposition, and arteriolar necrosis. Hypertensive emergencies are often compounded by the response of the renin-angiotensin system, resulting in further vasoconstriction and end-organ hypoperfusion.[32]

Differential Diagnosis

When encountering a patient with an elevated BP, the emergency clinician must consider whether it is an inaccurate measurement, a benign transitory elevation, a chronic condition, a hypertensive urgency, or a hypertensive emergency. **(See Table 3.)** In a case-control trial of 207 patients, a structured interview on the day after admission was used to compare patients with a hypertensive crisis with patients in other disease states who presented with an elevated BP measurement.[33] The authors reported the following adjusted odds ratios for having a hypertensive crisis: no primary care physician, 4.4; no medical insurance, 2.2; alcohol-related problem, 2.2; noncompliance with antihypertensive regimen, 2.0; and illicit drug use, 1.3. The study did not differentiate between essential hypertension and secondary hypertension.

Causes of secondary hypertension that the emergency clinician should be familiar with include stroke, aortic dissection, preeclampsia, pheochromocytoma, monoamine oxidase inhibitor interactions, thyroid storm, and sympathomimetics (such as cocaine) abuse. These conditions need to be specifically identified because their treatments differ substantially from the treatment of essential hypertension. For example, it is generally recommended that BP be permitted to remain elevated for cerebral protection in stroke patients; in patients with an aortic dissection, however, it is generally recommended that BP be aggressively controlled to avoid rupture or propagation of the dissection. Of note, these recommendations are consensus-based and are not supported by well-designed studies.

Table 3. Differential Diagnosis Of Hypertension In The ED[32]

Acutely Dangerous	Less Acutely Dangerous
Stroke	Obstructive uropathy
Aortic dissection	Hyperthyroidism/hyperparathyroidism
Drug intoxication: cocaine, amphetamine, monoamine oxidase inhibitor	Sleep apnea
Drug withdrawal: antihypertensive, alcohol, sedative-hypnotics	Cushing syndrome
Renal failure	Primary hyperaldosteronism
Pheochromocytoma or other tumor	Renovascular hypertension
Thyroid storm	Essential hypertension

Prehospital Care

Hypertension alone should not necessarily drive a prehospital action, but it should prompt the prehospital provider to assess the patient's stability and gather information concerning possible causes and effects. Pain and anxiety can play a role in elevating BP and must be addressed before focusing on management for an elevated BP. Hypertensive patients should also be evaluated for signs or symptoms of end-organ damage. Respiratory distress or crackles may indicate pulmonary edema; focal or global neurologic deficits may indicate an intracranial event or hypertensive encephalopathy; drug paraphernalia, prescription bottles, or a history of psychiatric illness could indicate a toxic or drug-induced hypertensive state; and chest pain may indicate acute coronary syndrome or aortic dissection.

The patient's personal hypertension history should also be obtained, including the medications he or she is taking and when the last dose was taken. If a patient is found to be hypertensive but is not transported to the hospital, he or she should be told of the finding and given instructions to follow up with a physician. Unfortunately, for some patients, a prehospital evaluation is their only exposure to the medical system.

The goals of prehospital hypertension management are based on its causes and on signs of end-organ effects. For example, many emergency medical system (EMS) protocols allow the use of nitroglycerin and furosemide for acute pulmonary edema.[34,35] Although there is a surprising paucity of studies in the prehospital arena to support these interventions, they are commonly recommended. There has been no standardization of outcome measures, making the few available studies difficult to compare.[36,37]

Trials examining treatment of hypertensive urgencies and emergencies in the prehospital setting have suffered from limited sample size and incomplete follow-up.[38] Data from an Israeli study demonstrated that chlorpromazine is helpful in lowering blood pressure, but there was very little follow-up on clinical outcomes.[39] Although there has been historical interest in using nifedipine in the prehospital setting for hypertensive emergencies,[40] it has fallen out of favor because of safety concerns.[41]

Emergency Department Evaluation

The Emergency Severity Index (ESI) is a 5-level triage system endorsed by the Agency for Healthcare

Research and Quality (AHRQ). This system has shown good inter-rater reliability and a strong relationship with resource use intensity. Unlike pulse, respiratory rate, or oxygen saturation, BP is not specifically mentioned in the ESI triage algorithm, partly because anxiety, stress, and pain can all result in nonpathologic BP elevation. Indeed, the "white coat hypertensives" are well described in the literature, and fortunately, their clinical outcome may be similar to that of nonhypertensive patients.[42,43] Consequently, it is up to institution protocol or the individual triage nurse to determine if an elevated BP represents a high-risk situation (ie, if the patient should be categorized as an ESI level 2 or an ESI level 3-5), depending on how many resources are needed).[44,45]

Measuring Blood Pressure

Blood pressure should be measured as part of every ED visit. Care must be taken when choosing an appropriate cuff for obese patients in order to avoid spuriously high readings.[46] A convenience sample of 53 postsurgical patients with arterial lines compared 2 arm cuffs and found greater random error with the large cuff but consistently elevated systolic and diastolic readings with the smaller cuff.[47] Further, atherosclerosis or orthostatic hypotension in elderly patients can mislead a clinician into defining them as hypertensive. In spite of these concerns, the ED visit is a good opportunity to identify patients with asymptomatic hypertension: 25% to 75% of patients with elevated systolic or diastolic BP in the ED remain hypertensive at follow-up.[48-51]

History

In patients with elevated BP, the history must focus on identifying life-threatening conditions. **(See Table 3, page 5; and Table 4, page 6.)**

Physical Examination

The physical examination should be systematic and complete, since hypertension can be caused by, and result in, multisystem disease. A complete set of vital signs should be checked and rechecked, including pulses and BPs in all extremities in selected cases. A funduscopic examination should be considered to assess for evidence of chronicity of the disease. Evidence of hyperthyroidism should be assessed by checking the thyroid and reflexes. A complete cardiopulmonary examination is critical for establishing the patient's baseline, and an abdominal examination should assess for evidence of an aortic aneurysm. The neurologic examination should evaluate the patient's cognitive status and assess for focal findings. Finally, since drug toxicities may present with elevated BP, a careful search for toxic syndromes should be performed.

Ancillary Testing

The recent Emergency Medicine Cardiac Research and Education Group (EMCREG) consensus panel recommends no ancillary testing in asymptomatic, healthy patients with a BP reading greater than 160/100 mm Hg because many will not have hypertension when rechecked.[2] The ACEP clinical policy on asymptomatic hypertension also does not recommend routine testing in asymptomatic patients.[8] Although a study of 109 asymptomatic patients with BP readings greater than 180/110 mm Hg on 2 measurements found unanticipated abnormal test results in 52% of patients, only 6% of results were clinically meaningful (ie, 2 basic metabolic panels [BMPs], 3 complete blood cell counts [CBCs], 3 urinalyses [UAs], 2 ECGs, and 1 chest radiograph).[52] Thus, testing should be tailored to the individual patient. **(See Table 5.)**

Table 4. Key Questions Regarding History Of The Present Illness

Question	Comments/Concerns
Have you ever been told you have high blood pressure?	Open-ended, inclusive question; many people do not think they have high blood pressure if they are taking—or have in the past taken—medication for it.
Do you have any chest pain?	Myocardial infarction, aortic dissection
Do you have any shortness of breath?	Myocardial infarction, aortic dissection, pulmonary edema, heart failure
Are you on any medications, or are you using any recreational drugs or herbal medicines?	Neuroleptic malignant syndrome; serotonin syndrome; cocaine, phencyclidine, or other sympathomimetics
Have you recently stopped taking any medications or recreational drugs or herbal medicines?	Delirium tremens, clonidine and other drug withdrawal
Have you had any focal weakness, slurring of speech, numbness, or clumsiness?	Stroke, transient ischemic attack, intracranial hemorrhage
Do you snore or wake up during sleep? Do you feel tired throughout the day?	Obstructive sleep apnea
Have you had high blood pressure in the past that has not responded to multiple medications?	Renovascular hypertension, hyperaldosteronism, pheochromocytoma

Complete Blood Count

Little data—and no evidence-based data—support routine collection of a CBC in patients with asymptomatic BP elevation. This test may pick up a rare case of microangiopathic hemolytic anemia; however, the cost-effectiveness of using the CBC to screen for this disorder in patients without a high-risk history has not been assessed.

Basic Metabolic Panel

Though not routinely recommended, the BMP may identify renal failure or other disease processes that result in electrolyte abnormalities such as hyperaldosteronism. For example, a recent retrospective study of 1616 patients found hypokalemia in 45% of patients with primary hyperaldosteronism.[53] The positive predictive value of low potassium levels in the general public was not assessed. Although an anion gap is independently and directly associated with elevated BP, the clinical utility of this information in the ED is questionable.[54]

Electrocardiogram

Findings on an ECG are often abnormal in the setting of prolonged hypertension. Overall, the results are specific, but not very sensitive, for LVH. One cohort study involving 19,434 patients examined both simple, amplitude-based criteria for LVH and more complicated, formulaic computer calculations. Although not the most sensitive, the Cornell criteria for summing the R wave in aVL and the S wave in V3, with a cutoff of 2.8 mV in men and 2.0 mV in women, was the best predictor of future cardiovascular mortality.[55] **(See Figure 2, page 8.)**

In response to hypertension, the heart is remodeled in a cycle, starting with increased wall stress that leads to hypertrophy, which leads to dysfunction, dilation, and additional wall stress. The ECG may show LVH and/or left atrial hypertrophy (LAH). Hypertensive patients with LVH are more likely to have an MI,[22] heart failure,[56] stroke,[57] or sudden death.[58] Patients with LAH are more likely to develop an arrhythmia such as atrial fibrillation.[24] Although the information obtained from the ECG may have limited utility in the ED setting for an individual patient, the additional risk identified by an abnormal ECG result may occasionally change the practitioner's actions. Finally, any acute ST changes, arrhythmias, or conduction abnormalities may be helpful in the proper setting.

Table 5. Emergency Department Laboratory Testing

Test	Utility
Urinalysis	Renovascular hypertension, nephrotic syndrome, nephritic syndrome, preeclampsia
Serum chemistry	Hyperaldosteronism, renal failure
Electrocardiogram	Left ventricular hypertrophy, left atrial hypertrophy, arrhythmias, myocardial infarction
Chest radiograph	Pulmonary edema, cardiomegaly, coarctation of the aorta
Complete blood cell count	Microangiopathic hemolytic anemia
Urine drug screen	Of very limited utility
Pregnancy test	Possible preeclampsia; avoid angiotensin-converting enzyme inhibitors

Chest Radiograph

A chest radiograph (x-ray) (CXR) in conjunction with a good physical examination is helpful in assessing for pulmonary edema and cardiomegaly. Crack cocaine use has been associated with lung changes and pneumomediastinum.[59] Both aortic dissection and coarctation of the aorta have well-described CXR findings, though the CXR is not the diagnostic modality of choice for aortic dissection.[60]

Urinalysis

Urinalysis is often used to assess for renal insufficiency (ie, to assess for the presence of proteinuria and red blood cells). The most rigorous study on the utility of UA in evaluating patients with hypertension correlated the analysis with the BMP.[61] Of note, this study began with the assumption that all hypertensive patients require a BMP. Since this assumption is not universally accepted, a UA may not be necessary either. Consequently, beyond opinion, no good evidence directs when a UA is indicated in the evaluation of patients with elevated BP.

Urine Drug Screen

The urine drug screen is an often-ordered, often-maligned test. Specific drugs tested for in this screen vary from hospital to hospital. There is no way to differentiate exposure from current toxicity on the basis of the screening result, and no good studies demonstrate the impact of a urine drug screen on clinical decision-making.

Pregnancy Test

A positive pregnancy test result may influence the choice of therapeutic intervention. A pregnancy test is indicated in women of childbearing age and may be the key to diagnosing preeclampsia in a molar pregnancy or a very obese patient.

Treatment

Many different drugs are used to treat hypertension.[62] To simplify this broad topic, common ED scenarios are presented in a situation-based format. Of note, many patients with a single high BP read-

ing will show a reduction on the next measurement without any intervention.[63]

Asymptomatic Patients With BP Less Than 180/110 mm Hg

In the ED, BP readings—especially persistently high ones—are good indicators of chronic hypertension.[64] However, patients with BP measurements less than 180/110 mm Hg do not need to be treated in the ED. Instead, these patients should follow up with a primary care provider within 1 week to 1 month[2] for verification, risk stratification, and therapy as needed. Unfortunately, data regarding when and how one needs to follow up are scarce. The patient's ability and willingness to follow up should be evaluated. Common sense dictates that a patient's adherence to the current regimen should also be discussed. For example, patients may have simply missed a few doses of their medications, or they may be having difficulty filling their prescriptions or accessing a primary care provider. Arrangements can then be made to assure that the patient has access to the medications that have already been prescribed.

Asymptomatic Patients With BP Over 180/110 mm Hg And A History Of Hypertension On Antihypertensive Medications

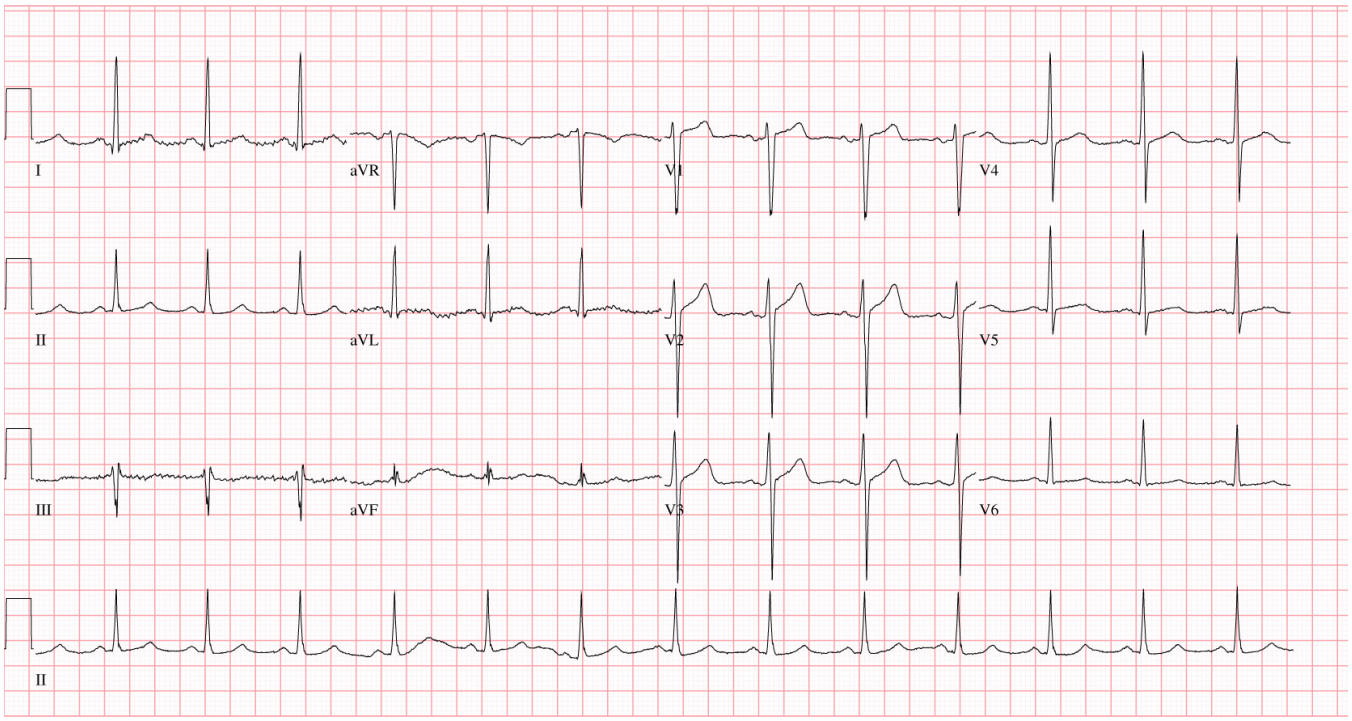
If these patients have missed their medications, they may be restarted on the drugs. Efforts should be made to ensure that the barriers that prevented the patient from taking the medications are addressed. Patients on clonidine may be particularly susceptible to rebound hypertension,[65] so they need to follow up with their healthcare provider within a 24- to 72-hour period. There are no good-quality studies that have assessed the timing of follow-up or the pace of BP reduction for patients off their antihypertensive agent.

For those patients who are compliant with their medications but still have an elevated BP, adjustments must be made. If patients have access to rapid follow-up, it might be best to send them home without treatment and have their primary care provider dictate a new regimen. Alternatively, the primary care provider could be contacted and a new medicine added, with subsequent close follow-up. This option has not been formally studied, however.[8]

Asymptomatic Patients With BP Over 180/110 mm Hg And No History Of Hypertension

In this scenario, patients should be started on antihypertensive medication if they cannot see a primary care provider the next day. The JNC 7 report recommends that primary care providers start 2 agents when a patient comes in with very high BP.[1] It is unclear if this strategy is applicable in the ED setting, however. No good studies show how quickly BP needs to be lowered or which agent or agents are best for reaching that goal. The choice of initial agent is multifactorial. Ethnicity, age, economics, and comorbidities should be assessed when choosing an anti-

Figure 2. Left Ventricular Hypertrophy On Electrocardiogram



hypertensive medication.[66] Some considerations are listed in **Table 6 (page 9), Table 7 (page 10), and Table 8 (page 11).**

Hypertensive Emergencies

No studies have addressed the efficacy of therapies for hypertensive emergencies on clinical outcomes. The broad scope of the term "hypertensive emergency" and variability in authors' criteria for diagnosing this disorder make comparing trials difficult. The treatment of hypertensive emergencies is predicated on the end-organ symptoms the patient is experiencing and whether the patient has essential or secondary disease.[77] Conditions that cause acute end-organ damage require rapid and controlled correction of the BP level. The most common forms of end-organ

Table 6. Outpatient Oral Medications For Hypertension Management

Agent	Starting Dose	Maximum Useful Dosage	Indication	Contraindication	Monitoring	Properties/ Complications
Thiazide diuretics (eg, hydrochlorothiazide)	12.5 mg daily	25 mg daily	Drug of choice for uncomplicated hypertension[1]; works well with other agents	Gout, hypokalemia, hypercalcemia	BMP	Loses effectiveness as GFR decreases; can cause multiple electrolyte disturbances; drug-induced SLE
ACE inhibitor (eg, fosinopril, lisinopril)	5-10 mg daily	40 mg daily	Patients with CHF, diabetes, previous MI with low ejection fraction[67,68]	Bilateral renal artery stenosis; hypovolemia	BMP	Interrupts renin-aldosterone cascade; hyperkalemia; cough; angioedema
Angiotensin receptor blockers (eg, losartan)	25-50 mg daily	100mg daily	Similar efficacy to ACE inhibitors; used for patients who cannot tolerate these inhibitors or in addition to them[69-71]	Bilateral renal artery stenosis; hypovolemia	BMP	Interrupts renin-angiotensin-aldosterone cascade; hyperkalemia; angioedema
β-Blockers (eg, metoprolol)	25-50 mg bid	200 mg bid	Patients with coronary artery disease; long-term management of CHF; rate control; hyperthyroidism[72-74]	Not a good monotherapy for lone hypertension; heart block; bradycardia; sick sinus syndrome; bronchospasm; acute decompensated CHF exacerbation	ECG	Blocks catecholamines; can lead to bronchospasm, bradycardia
Calcium channel blockers (eg, diltiazem)	180-240 mg daily	360-540 mg daily (formulation dependent)	Rate control or coronary artery disease in patients who cannot take β-blockers	Not a good monotherapy for lone hypertension; long-acting agents are safer than short-acting agents; heart block; bradycardia; acute decompensated CHF exacerbation; sick sinus syndrome[75,76]	ECG	Blocks calcium channels in heart and vessels; some cause edema; some affect heart rate
α-2 Agonist (eg, clonidine)	0.1 mg bid	0.3 mg tid	Hypertension resistant to other modalities	Poor adherence to medical regimen	Monitor for hypotension	Orthostatic hypotension; fatigue; withdrawal may lead to severe rebound hypertension
Hydralazine (unknown mechanism of vasodilation)	10 mg 4 qid	100 mg tid	Hypertension associated with pregnancy; hypertension associated with CHF in African Americans resistant to other modalities	Coronary artery disease	NA	Drug-induced SLE

Abbreviations: ACE, angiotensin-converting enzyme; bid, 2 times per day; BMP, basic metabolic panel; CHF, congestive heart failure; ECG, electrocardiogram; GFR, glomerular filtration rate; MI, myocardial infarction; NA, not applicable; qid, 4 times per day; SLE, systemic lupus erythematosus; tid, 3 times per day.

damage in hypertensive emergencies, in order of decreasing frequency, are (1) cerebral infarction or hemorrhage; (2) acute pulmonary edema; (3) hypertensive encephalopathy; (3) acute CHF; and (4) aortic dissection. Preeclampsia is another hypertensive state associated with very high BP, but unique goals and treatment options are associated with this condition. (**See *Emergency Medicine Practice*, May 2009.**)

Acute Ischemic Stroke

Hypertension is found in 77% of patients who have experienced an ischemic stroke.[16] Ischemic stroke may be associated with very high BP of a nonpathologic nature. Acute stroke patients frequently resolve their hypertensive states spontaneously.[78] Compelling data indicate that immediately lowering BP in stroke patients leads to worsening neurologic outcomes.[79] However, antihypertensive therapy is not universally associated with poorer outcomes, and a systematic review of the Cochrane database shows inconclusive results.[80] With the dearth of hard evidence, current treatment guidelines rely on expert opinion. Severely high BP (ie, SBP greater than 220 mm Hg or DBP greater than 120 mm Hg) can be treated with intravenous (IV) labetalol or nicardipine, with the goal of a 10% to 15% reduction.[81] In refractory cases, if the DBP remains over 140 mm Hg, nitroprusside IV may be considered, though there is some concern that it may increase intracranial pressure.

Stroke patients who are hypertensive, have a BP reading greater than 185/110 mm Hg, and are eligible for thrombolytic therapy are uniquely challenging. These patients need close monitoring and tight control. The goal is to get the BP to a stable level just below the cutoff range for thrombolysis (ie, an SBP of 180 mm Hg and a DBP of 105 mm Hg). Because of the time constraints associated with thrombolytic therapy, patients with severely elevated BP who require a nitroprusside drip may not reach a stable level before the therapy window closes.[81]

Despite this recommendation to use caution in giving thrombolytic therapy to patients with difficult-to-stabilize hypertension, a retrospective review found no increase in adverse events in 178 patients requiring aggressive BP-lowering treatment compared with patients not requiring antihypertensive agents.[82]

Table 7. Common Drugs And Pharmacokinetics For Restarting Outpatient Medications After Missed Doses

Drug	Onset	Duration	Excretion
Hydrochlorothiazide	2 h (peaks in 4-6 h)	6-12 h	Urine
Fosinopril	1 h	24 h	Urine and stool
Losartan	6 h	< 24 h	Urine
Metoprolol	1.5 h	10-20 h	Metabolized in liver, excreted in urine
Diltiazem ER	10-14 h	24 h	Metabolized in liver, excreted in urine
Amlodipine	30-50 min	24 h	Metabolized in liver, excreted in urine

Lexi-Comp Online™, Hudson, Ohio: Lexi-Comp, Inc.;1978-2008
http://webstore.lexi.com/s.nl/ctype.KB/it.I/id.342/KB.3234/.f

Abbreviations: h, hour(s); min, minutes.

Acute Pulmonary Edema/Congestive Heart Failure

Hypertensive acute heart failure is defined as signs and symptoms of heart failure accompanied by high BP and relatively preserved left ventricular function with results from a CXR compatible with acute pulmonary edema.[83]

Acute pulmonary edema can be related to hypertension in patients with both systolic and diastolic heart failure. Pulmonary edema is induced by fluid overload when the heart is unable to maintain adequate forward flow to prevent fluid from backing up into the lungs. Pulmonary edema can occur quickly, especially when associated with acute hypertension. Patients who can tolerate treatment for acute heart failure (including vasodilators and diuretics) without becoming hypotensive tend to have better outcomes. Those who cannot maintain their BP are at high risk for a poor outcome. Mortality in acute heart failure is inversely proportional to BP levels. The goal of treatment in acute heart failure is to improve symptoms, BP control, left atrial pressure, and cardiac output. Pitfalls to avoid while treating patients include hypotension, cardiac ischemia, renal dysfunction, and arryhthmias.[84]

Treatment of acute pulmonary edema often begins prehospital. For example, nitroglycerin and/or furosemide IV are commonly given prior to the patient's arrival in the ED. Nitrates (nitroglycerin and nitroprusside) are vasodilators that act on both venous and arterial systems, although nitroglycerin is much more selective for dilation of veins. Nitrates have also been shown to improve the hemodynamics in patients with heart failure; for example, a prospective randomized study of 104 patients concluded that use of high-dose nitrates and low-dose furosemide was more beneficial than use of high-dose furosemide and low-dose nitrates in treating patients with severe pulmonary edema.[85] Although nitrates have become generally accepted for the management of acute pulmonary edema and hypertension, surprisingly few studies support their use.[86]

Diuretics, specifically loop diuretics, are commonly used to treat acute heart failure. The few studies that have examined their effect on mortality found some evidence that they may be inferior to

other modalities in treating chronic[87] and acute heart failure,[85] raising concerns about their use. Although this evidence is too limited to recommend against the use of diuretics, it does highlight the point that many of the treatments in common practice have surprisingly sparse data behind them. The Japanese Multicenter Evaluation of Long- Versus Short-Acting Diuretics in Congestive Heart Failure (J-MELODIC) study is currently underway to address these issues. The ACEP clinical policy for treatment of CHF includes a class B recommendation for use of continuous positive airway pressure (CPAP) to help treat pulmonary edema and decreasing respiratory effort, reducing the need for intubation. This recommendation is based on results from 6 small studies. The policy also gives a class B recommendation for use of nitroglycerin in patients with dyspnea, while adding furosemide for patients with moderate to severe pulmonary edema. Nevertheless, ACEP cautions against aggressive diuretic use, a caveat based on a retrospective cohort study involving 1681 patients and a prospective cohort study of 412 patients.[88] Other therapies for hypertensive emergencies in the setting of pulmonary edema or CHF include ACE

Table 8. Parenteral Drugs For Treatment Of Hypertensive Emergencies*

Drug	Dose	Onset of Action	Duration of Action	Adverse Effects†	Special Indications
Vasodilators					
Sodium nitroprusside	0.25-10 µg/kg/min as IV infusion‡	Immediate	1-2 min	Nausea, vomiting, muscle twitching, sweating, thiocynate and cyanide intoxication. May increase intracranial pressure	Most hypertensive emergencies; caution with high intracranial pressure or azotemia
Nicardipine hydrochloride	5-15 mg/h IV	5-10 min	15-30 min, may exceed 4 hrs	Tachycardia, headache, flushing, local phlebitis	Most hypertensive emergencies except acute heart failure; caution with coronary ischemia
Fenoldopam mesylate	0.1-0.3 µg/kg/min IV infusion	< 5 min	30 min	Tachycardia, headache, nausea, flushing	Most hypertensive emergencies; caution with glaucoma
Nitroglycerin	5-100 µg/min as IV infusion‡	2-5 min	5-10 min	Headache, vomiting, methemoglobinemia, tolerance with prolonged use	Coronary ischemia
Enalaprilat	1.25–5 mg every 6 hrs IV	15-30 min	6-12 hrs	Precipitous fall in pressure in high-renin states; variable response	Acute left ventricular failure; avoid in acute myocardial infarction
Hydralazine hydrochloride	10-20 mg IV 10-40 mg IM	10-20 min IV 20–30 min IM	1-4 hrs IV 4-6 hrs IM	Tachycardia, flushing, headache, vomiting, aggravation of angina	Eclampsia
Adrenergic Inhibitors					
Labetalol hydrochloride	20-80 mg IV bolus every 10 min 0.5-2.0 mg/min IV infusion	5-10 min	3-6 hrs	Vomiting, scalp tingling, bronchoconstriction, dizziness, nausea, heart block, orthostatic hypotension	Most hypertensive emergencies except acute heart failure
Esmolol hydrochloride	250–500 µg/kg/min IV bolus, then 50–100 µg/kg/min by infusion; may repeat bolus after 5 min or increase infusion to 300 µg/min	1-2 min	10-30 min	Hypotension, nausea, asthma, first-degree heart block, heart failure	Aortic dissection, perioperative
Phentolamine	5–15 mg IV bolus	1-2 min	10-30 min	Tachycardia, flushing, headache	Catecholamine excess

Abbreviations: h, hour; hrs, hours; IM, intramuscular; IV, intravenous; min, minute(s).

* These doses may vary from those in the Physicians' Desk Reference (51st ed.)

† Hypotension may occur with all agents

‡ Requires special delivery system

Reprinted from The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. US Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute. 2004.

inhibitors and vasopressin antagonists. These treatments have not been adequately studied to justify recommendations, but both hold promise.

Hypertensive Encephalopathy

Hypertensive encephalopathy describes reversible cerebral disorders associated with high BP in the absence of cerebral thrombosis or hemorrhage.[89] The theoretical mechanism of hypertensive encephalopathy is a rapid rise in BP that overwhelms the autoregulatory mechanisms of the brain and leads to blood-brain barrier permeability and brain edema. The symptoms of hypertensive encephalopathy can include headache, seizures, visual disturbances, nausea, and vomiting. The diagnosis must be made only after other potential hypertensive emergencies are excluded.

A PubMed search for clinical trials, meta-analyses, and randomized controlled trials of hypertensive encephalopathy yielded 22 articles. None of the trials dealt directly with treatment or prognosis, and no studies suggested which agent is best for short-term reduction of BP in hypertensive encephalopathy. However, nitroprusside should be avoided in patients with this disorder, as the drug has been shown to decrease systemic pressure while preserving intracranial perfusion pressures.[90] In the absence of data, the consensus goal of treatment is often stated as a 20% to 25% reduction in mean arterial pressure or a DBP of 100 to 110 mg Hg.[2]

Acute Intracerebral Hemorrhage

Emergency clinicians walk a tightrope when trying to maintain sufficient perfusion pressure to the brain without worsening the amount of hemorrhage. According to a study by Kazui et al, hematoma expansion is not directly linked to patient deterioration; however, extreme changes in hematoma size are associated with poorer outcomes.[91] No prospective data exist on how to manage elevated BP in these patients. Some studies have assessed the safety of IV antihypertensives, but none have shown efficacy. Although hypertension on presentation is associated with hematoma expansion,[92] no study has shown that treatment of the hypertension prevents this expansion, nor does the association between high SBP and hematoma expansion confirm that one causes the other.

The optimal agent and target pressure for treating intracerebral hemorrhage are still the subjects of much debate. The ongoing ATACH (Antihypertensive Treatment of Acute Cerebral Hemorrhage) and INTERACT II (Intensive Blood Pressure Reduction in Acute Cerebral Haemorrhage) studies are investigating these issues. Intracerebral hemorrhage is not universally associated with intracranial hypertension. As discussed previously, nitroprusside may allow for BP reduction while preventing a drop in intracranial perfusion pressure. Until there is evidence to direct emergency clinicians toward one agent or set of agents, the choice is left to them. Goals of therapy for intracerebral hemorrhage can be found in **Table 9**.

Table 9. Suggested Recommended Guidelines For Treating Elevated Blood Pressure In Spontaneous Intracerebral Hemorrhage[93]

1. If SBP is > 200 mm Hg or MAP is > 150 mm Hg, consider aggressive reduction of BP with continuous IV infusion, with BP monitoring every 5 minutes.
2. If SBP is > 180 mm Hg or MAP is > 130 mm Hg and there is evidence for or suspicion of elevated intracranial pressure, consider monitoring intracranial pressure and reducing BP using intermittent or continuous IV medications to keep cerebral perfusion pressure > 60-80 mm Hg.
3. If SBP is > 180 mm Hg or MAP is > 130 mm Hg and there is no evidence for or suspicion of elevated intracranial pressure, consider a modest reduction of BP (eg, MAP of 110 mm Hg or target BP of 160/90 mm Hg) using intermittent or continuous IV medications; clinically re-examine the patient every 15 minutes.

Abbreviations: BP, blood pressure; IV, intravenous; MAP, mean arterial pressure; SBP, systolic blood pressure.

Aortic Dissection

Aortic dissection occurs when a false lumen is created in the wall of the aorta. The Stanford classification system divides dissections into 2 types, A or B. The type has a direct bearing on management. Type A includes any involvement of the ascending aorta (proximal to the left subclavian artery), whereas type B spares the ascending aorta. Ascending aortic dissections require immediate evaluation by a cardiothoracic surgeon for an emergency surgical procedure. Type B dissections are usually managed medically in collaboration with a surgeon.

The common treatments for aortic dissection are narcotics for pain control, a titratable IV β-blocker (eg, esmolol), and nitroprusside for BP control. Calcium channel blockers are considered second-line interventions. The theory behind this management strategy is that reducing the force of left ventricular contractions, thus dilating the vessels, will enhance laminar flow and lessen stress on the aortic wall. Turbulent flow is increased by using a vasodilator alone.

The goal of antihypertensive therapy in aortic dissection is unique in that the target pressure is the lowest pressure tolerated by the patient. Systolic levels of 100 to 120 mm Hg are ideal.[94,95] This goal is in stark contrast to those of previous sections, in which modest decreases in BP are the rule. Once again, there is very sparse evidence behind these recommendations. In addition to lowering the BP, the emergency clinician should attempt to slow the heart rate to reach a target of

Risk Management Pearls For Management Of Hypertension In The ED

1. **Patient factors may lead to an erroneously high BP reading.**
   Recheck the patient's BP. Serial BP measurements are more accurate than a single reading. Be sure the cuff fits the patient properly. If the reading obtained does not fit the clinical scenario, check the patient's BP manually in both arms. Confirm that the true pressure is being measured.

2. **The asymptomatic patient does not need a rapid correction of the BP level.**
   Patients with chronically high BP may have reset the autoregulation parameters that control their cerebral circulation. A rapid decline in pressure might put the patient into intracranial hypotension, resulting in an ischemic stroke. There is no proven benefit from quickly lowering BP in an asymptomatic patient.

3. **Overly aggressive use of antihypertensive agents should be avoided in patients with an acute ischemic stroke.**
   The evidence suggests that having hypertension after a cerebrovascular accident actually protects patients. For extremely high pressures, some control of BP is warranted. **(See section on Acute Ischemic Stroke, page 10 and the Clinical Pathway For Symptomatic Hypertension, page 15.)** Modest reductions in extreme hypertension (DBP > 120 mm Hg) and permissive hypertension for those with more reasonable values is the rule.

4. **Everyone with an elevated BP requires a recheck.**
   People who present to the ED with incidental findings of hypertension are likely to have true hypertension or prehypertension. Although these findings probably would not lead to immediate problems, the ED can be an effective screening tool for patients who use the healthcare system infrequently. This is a ideal opportunity to encourage patients to see their primary care provider or initiate a primary care relationship.

5. **Sublingual nifedipine should be avoided.**
   Nifedipine sublingual (SL) provides unpredictable and relatively long-acting antihypertensive effects. The patient may not respond at all—or more dangerously, may become hypotensive. Long-acting, long-onset medications should be avoided in hypertensive emergencies, as they cannot be well-titrated or controlled.

6. **If emergency clinicians assume the role of primary care providers, the patient requires a primary care workup.**
   Too often, ED clinicians are faced with a situation where they must expand the scope of their practice. Chronically elevated BP needs to be treated. If the ED is becoming the de facto primary care contact, clinicians must do the same workup that a primary care provider would do, including ECG, BMP, and CBC. Furthermore, medications that require monitoring for electrolyte levels or that may cause rebound hypertension should be avoided.

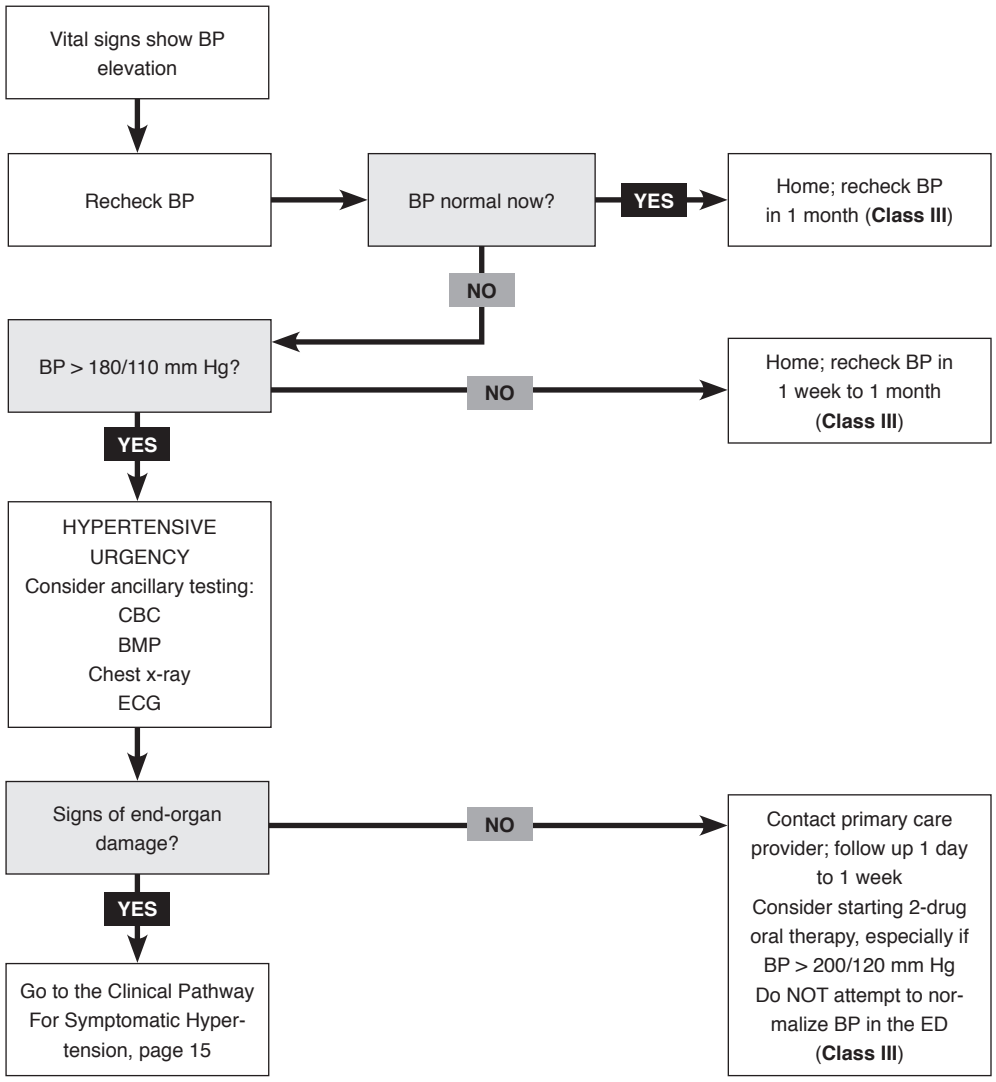
7. **Medications with rapid onset do not necessarily have a short half-life.**
   Drugs such as nicardipine that have rapid onset may have a long half-life after prolonged administration. Any patient receiving an IV medication for a hypertensive emergency should be monitored extremely closely. The rate of BP change should be noted so that extreme high or low values can be avoided. Additionally, medications that are cleared primarily by redistribution or that depend on a damaged end organ for metabolism may have a longer-than-expected effect.

8. **Induction of reflex tachycardia in patients with an aortic dissection should be avoided.**
   Patients with an aortic dissection do benefit from vasodilator therapy, but watch for tachycardia in these patients. Often, a β-blocker is needed to control the patient's heart rate.

9. **Prescribed medications should be affordable.**
   If the patient cannot afford the prescribed medication, he or she cannot take it. A switch to the generic drug will likely improve adherence to therapy.

10. **Complicated dosing regimens should be avoided in patients who have difficulty adhering to them.**
    A once-daily drug might be warranted, despite the increased cost.

Clinical Pathway For Asymptomatic Hypertension



Abbreviations: BMP, basic metabolic panel; BP, blood pressure; CBC, complete blood count; ECG, electrocardiogram; ED, emergency department.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

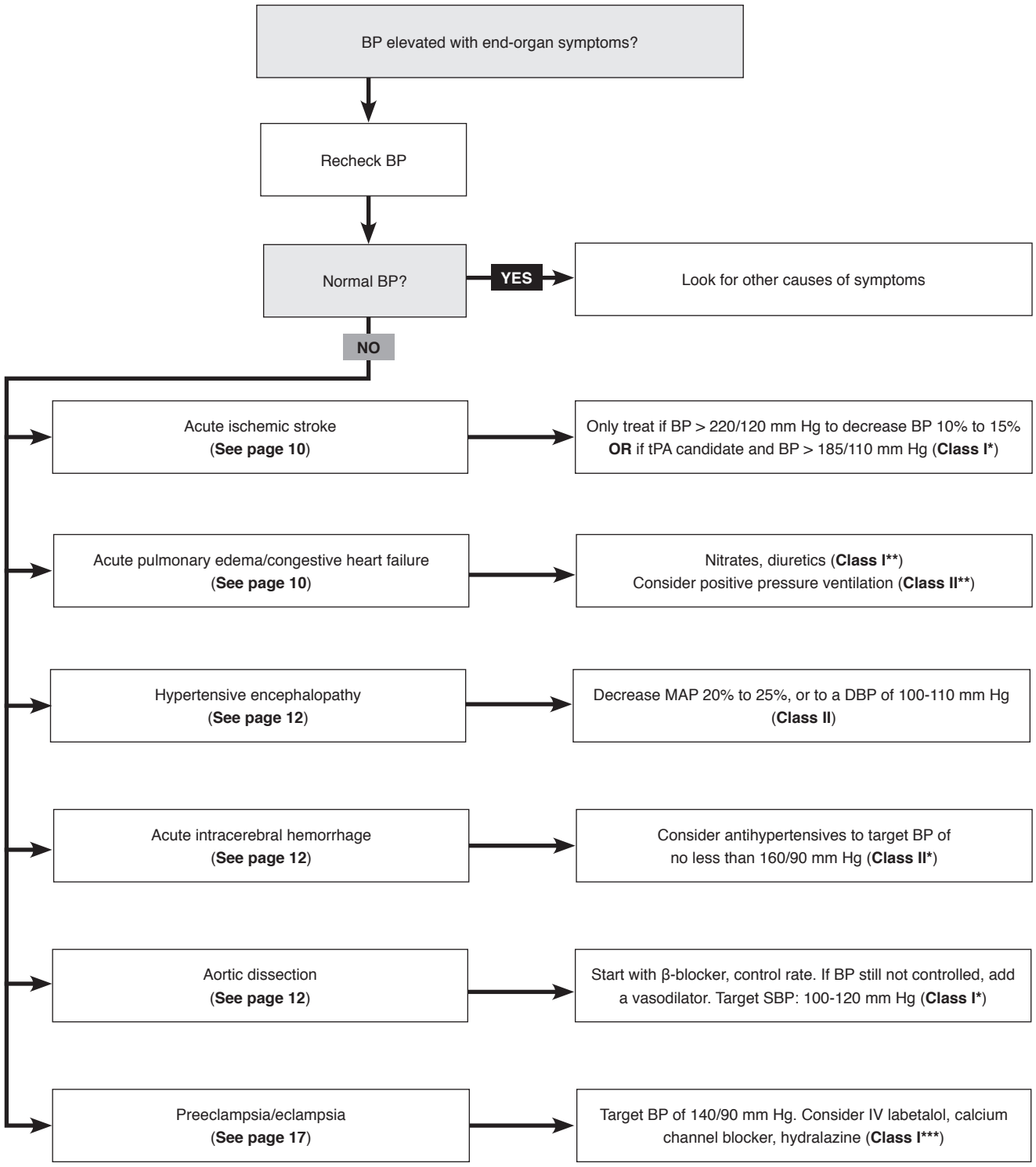
Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA.* 1992;268(16):2289-2295.

Clinical Pathway For Symptomatic Hypertension



See Class of Evidence descriptions on page 14.

Abbreviations: ACE, angiotensin-converting enzyme; BP, blood pressure; DBP, diastolic blood pressure; IV, intravenous; MAP, mean arterial pressure; SBP, systolic blood pressure; tPA, tissue plasminogen activator.

*American Heart Association guidelines
**European Society of Intensive Care Medicine guidelines
***Royal College of Obstetricians and Gynaecologists guidelines

60 to 70 beats per minute; these numbers are not evidence-based, however.

The one exception to using β-blockers in combination with a vasodilator is in patients with acute aortic regurgitation caused by the dissection. In these patients, the loss of contractility may reduce forward flow and perfusion, thus causing more harm than good.[96]

Sympathetic Crisis

A number of conditions can result in a sympathetic crisis. For example, recreational drugs such as phencyclidine (PCP), cocaine, and methamphetamine can cause hypertensive urgencies or emergencies. Withdrawal from certain substances such as alcohol or clonidine can also cause a sympathetic crisis. Hormonal abnormalities like those seen in pheochromocytoma or thyroid storm can also lead to this condition.

Cocaine intoxication can present as agitation and hypertension (**see *Emergency Medicine Practice*, January 2008**). Calming the patient and relocating him or her to a quiet place is the first step in the management of this crisis. Benzodiazepines are the first-line pharmacologic treatment followed by vasodilators such as phentolamine and nitroglycerin.[97] Evidence supporting the efficacy of benzodiazepines and nitroglycerin comes from underpowered studies; however, these studies show a trend toward benefit. The use of β-blockers in hypertensive patients with cocaine-induced chest pain is controversial because of concerns about unopposed alpha activation exacerbating the hypertension; however, evidence supports a protective effect in preventing MI.[98]

Methamphetamine, currently a popular drug of abuse, has been reported to cause intracerebral hemorrhage and MI in young adults. The treatment for methamphetamine-overdose-related hypertension has not been assessed. An Ovid MEDLINE® search using the terms *methamphetamine AND hypertension* yielded no randomized controlled trials, meta-analyses, or clinical trials and only 4 articles that directly address the topic. Likewise, the association between methamphetamine-induced hypertension and intracerebral hemorrhage is based on case reports.[99]

Phencyclidine was a popular topic in publications during the 1970s. Due to the lack of hard evidence supporting any specific antidotes for PCP intoxication, the combination of supportive measures and benzodiazepines seems to be a reasonable therapeutic approach.

Pheochromocytomas can cause hypertensive emergencies by releasing large amounts of catecholamine at once. Patients may present with the classic clinical triad of episodic sweating, headache, and tachycardia. However, the same symptoms can be seen with other conditions such as clonidine withdrawal, panic attacks, and the drug intoxications discussed previously. In the ED, treatment for pheochromocytomas is based on perioperative management for removal of these tumors. Oral α-blockers such as doxazosin and

Time- And Cost-Effective Strategies For Hypertension Management

Follow Evidence-Based Guidelines

An interesting retrospective study compared the cost of prescriptions actually given to patients for hypertension with the cost of medications recommended in the current clinical guidelines. The study was limited by many confounding factors, but it did show a 25% reduction in total drug costs when current clinical guidelines were followed. Considering the prevalence of hypertension, use of recommended drugs could have a large effect on total expenditures for prescribed drugs in this country.[110]

Diagnose The Hypertension

Patients should be informed that they have high BP and instructed to arrange for proper follow-up. Depending on their individual risk factors, patients can expect a significant prolongation of quality life once hypertension is controlled.[111] Patients may not bring up their elevated BP with their primary care physician, even if they are told about the elevation during their ED visit. Explicit directions for obtaining a BP check may motivate them to follow up.

Be Mindful Of New Drugs

Drugs such as nesiritide that are very costly and well-advertised are tempting to use, especially when many patients or their families have heard about them. Unless the data suggest a compelling improvement in clinical outcomes, be cautious about switching from an inexpensive, tried-and-true drug to a costly new one. Some new drugs have been shown over time to be better. Consider the venerable adage: Never be the first or last practitioner to prescribe a drug.

Promote Adherence

The medication with the worst cost-to-benefit ratio is the one that sits in the bottle. There are many barriers to patient adherence, including cost, dose scheduling, and understanding of the disease. Minimizing these barriers improves the ability to treat patients.

IV phentolamine may be used.[100] Data also support the use of peripherally acting calcium channel blockers, specifically nicardipine.[101] Other drugs that dilate veins may be used as well. Nitroprusside in combination with labetalol has been shown to be effective.[102] It should be noted that use of a vein dilator in these patients counteracts the α-agonistic effect of using a β-blocker to control heart rate.

Unstable Angina/Non-ST-Segment Elevation Myocardial Infarction

The American Heart Association/American College of Cardiology (AHA/ACC) guidelines provide recommendations on the use of antihypertensive medications in unstable angina/non-ST-segment elevation MI.[103] Nitroglycerin can be used to control both symptoms and BP; however, it should not be used to the exclusion of other therapies that are more likely to have a beneficial effect on outcomes. Patients should be asked if they have recently used sildenafil or one of its analogues, as the combination with nitrates can cause severe hypotension.[104] The use of oral β-blockers in the ED is optional. The current AHA/ACC guidelines suggest that oral β-blockers can be given any time within the first 24 hours of presentation. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers may be used for patients with hypertension and left ventricular dysfunction or pulmonary congestion. Recommendations allow ACE inhibitors to be given within 24 hours of presentation.

Preeclampsia/Eclampsia

Preeclampsia is the combination of hypertension and proteinuria in a pregnant woman after the 20th weeks' gestation. Preeclampsia becomes eclampsia when the patient has a seizure. Hypertension management is unique in pregnancy, in that many common drugs are contraindicated because of the potential for toxic effects on the fetus. Further BP-lowering therapy has not been shown to change outcomes in patients with an SBP of 140 to 170 mm Hg.[105] Patients are usually started on methyldopa or a calcium channel blocker as oral outpatient therapy.

In hypertensive emergencies associated with preeclampsia or eclampsia, the goal of therapy is a reduction in SBP to 140 mm Hg and a DBP of 90 mm Hg. Classic therapy involving hydralazine is not ideal because it can drop the pressure precipitously.[106] Further, the patient's BP may fluctuate widely, although constant monitoring may prevent dangerously high or low levels. Nicardipine and labetalol are reasonable alternatives.[107,108] (**See *Emergency Medicine Practice*, May 2009**)

Renal Failure

The kidney is intimately involved in BP regulation and hypertension. It is the key organ for both volume maintenance and control of the renin-angiotensin system. The prevalence of hypertension in patients with chronic renal failure is linearly related to the glomerular filtration rate (GFR), with the prevalence approaching 100% for those with GFR less than 10 mL/min.[108] The JNC 7 report recommends a BP goal of 130/80 mm Hg in patients with renal failure.[1] The treatment of hypertension in these patients should involve an ACE inhibitor, especially for patients on hemodialysis[109]; however, initiating an ACE inhibitor in the ED should be done with caution, as creatinine and potassium levels must be closely monitored. Data on management of hypertensive emergencies in dialysis patients are sparse, and no specific guidelines are available.

Disposition

According to consensus recommendations, patients with hypertensive urgencies can be discharged from the ED. If the BP is greater than 200/120 mm Hg, oral antihypertensive therapy should be started. For BP greater than 180/110, follow-up should occur within 1 week. If prompt follow-up cannot be ensured, then further consideration for BP treatment or titration of existing BP medications should be given. For BP greater than 160/100, follow-up in less than 1 month is recommended; for BP greater than 140/90 mm Hg, follow-up at 1 month is recommended.[2]

Patients with hypertensive emergencies will generally be admitted to the intensive care unit after receiving titratable IV antihypertensive agents. Patients with type A aortic dissections should be admitted directly to the operating room or transferred to a hospital that offers cardiothoracic surgery. Intracerebral hemorrhages require immediate neurosurgical evaluation. Patients with preeclampsia/eclampsia can have a variety of dispositions; treatment options are limited because of concerns about fetal toxicity associated with most antihypertensive agents.

Summary

Hypertension, hypertensive urgency, and hypertensive emergency are common ED occurrences, both incidentally and as primary diagnoses. Specific patients, including those with aortic dissection or prior thrombolytic treatment, require identification and immediate treatment. Otherwise, the temptation to over-treat should be avoided, and outpatient follow-up specifically for hypertension should be recommended for optimal long-term BP management.

Patients with asymptomatic hypertension should not have their BP rapidly corrected in the ED. These patients should be referred to a primary care physician for further evaluation and care. No clear evidence supports the screening of this patient population with any laboratory tests; clinical judgment is crucial.

Patients with symptomatic hypertension should receive a workup and management that is tailored to the end organ involved. The mechanism of action, side effects, and pharmacodynamics should all be considered when choosing a specific antihypertensive agent.

Case Conclusions

The 58-year-old man's headache responded to metoclopramide, and his erythrocyte sedimentation rate was only 7 mm/h. He had a history of hypertension that was being followed by his primary care provider, so no further evaluation for BP was indicated in the ED.

The 71-year-old woman sent in by her primary care provider was evaluated, and an ECG showed normal sinus rhythm without signs of ischemia or arrhythmia. A creatinine value of 0.8 mg/dL and a potassium level of 4.1 mEq/L were noted. After discussion with her primary care provider, you administered lisinopril and hydrochlorothiazide and recommended follow-up for a BP recheck in 5 days.

The 96-year-old woman was found to have crackles at bilateral lung bases and an elevated BNP level. A CXR shows pulmonary edema. A nitroglycerin drip was started, and the patient was placed on CPAP. She also received a dose of furosemide IV after her metabolic profile was assessed. She slowly improved, and her BP dropped to 175/110 mm Hg. She did not require intubation and was transferred to the coronary care unit.

Discussion Of Off-Label Drug Use

The only medication listed in this article that has a specific indication for hypertensive crisis is nitroprusside. Labetalol IV has an indication for severe hypertension. Nitroglycerin IV has an indication for treatment of perioperative hypertension and control of CHF in acute MI. Nesiritide has an indication for acutely decompensated CHF not responding to diuretics. It can be argued that these 4 medications have a label indication for use in acute BP management.

Phentolamine is indicated only for the prevention or treatment of hypertensive episodes in patients with pheochromocytoma, so its use in other sympathetic crises such as cocaine intoxication is off-label. Nicardipine and enalaprilat are indicated for short-term treatment of hypertension when oral therapy is not feasible.

The other agents have indications for more long-term use; administration of these agents in a hypertensive urgency or emergency should be based on the type of chronic condition they are designed to treat. Amlodipine has an indication for chronic stable angina and vasospastic angina. Both diltiazem IV and esmolol IV have indications for management of atrial fibrillation or atrial flutter. Hydrochlorothiazide, lisinopril, losartan, and metoprolol have indications for chronic hypertension.

The benzodiazepine lorazepam is indicated as a preanesthetic agent or for status epilepticus. Midazolam is indicated for use as a sedative agent. Neither of these benzodiazepines has a label indication for sympathetic crises.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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1. Which of the following clinical scenarios is a hypertensive urgency?
   a. A 63-year-old man with repeated BP readings of 167/88 mm Hg who presents with acute alcohol intoxication
   b. A 65-year-old woman with repeated BP measurements of 185/120 mm Hg who presents with a minor arm laceration
   c. A 78-year-old man with a BP reading of 250/140 mm Hg with altered mental status
   d. A 79-year-old man with repeated BP measurements of 154/104 mm Hg who presents with conjunctivitis

2. Which of the following clinical scenarios qualifies as a hypertensive emergency?
   a. A 23-year-old woman who is 30 weeks pregnant with a BP reading of 140/95 mm Hg and no proteinuria
   b. A 56-year-old man with a history of poorly controlled hypertension who presents with a finger fracture and is found to have a BP reading of 195/120 mm Hg in triage
   c. A 58-year-old woman with an aortic dissection who presents with tearing chest pain radiating to the back and a BP reading of 180/120 mm Hg
   d. An asymptomatic 65-year-old man with no medical history who is sent in from a health fair with a BP reading of 200/110 mm Hg

3. According to a 2003-2004 study, the prevalence of hypertension in the US is closest to:
   a. 10%
   b. 30%
   c. 50%
   d. 70%

4. Which of the following tests must be performed on every patient presenting to the ED with a BP > 180/120 mm Hg?
   a. BMP
   b. CBC
   c. Urinalysis
   d. Urine drug screen
   e. None of the above

5. Which of the following events associated with hypertension most commonly leads to a hypertensive emergency?
   a. Acute pulmonary edema
   b. Aortic dissection
   c. Cerebral infarction or hemorrhage
   d. Hypertensive encephalopathy

6. ACE inhibitors are associated with which of the following side effects?
   a. Angioedema
   b. Cough
   c. Hyperkalemia
   d. All of the above

7. What is the BP goal for patients receiving tissue plasminogen activator for an acute ischemic stroke?
   a. 120/80 mm Hg
   b. 140/90 mm Hg
   c. 180/105 mm Hg
   d. 210/110 mm Hg
   e. 240/120 mm Hg

8. **Nitroprusside is a medication that can maintain intracranial pressure. In which of the following situations should it NOT be used?**
   a. Aortic dissection
   b. Extreme hypertension with DBP greater than 140 mm Hg
   c. Hypertensive encephalopathy
   d. Ischemic stroke with severe hypertension

9. **Which of the following medications is most appropriate for treating a hypertensive emergency associated with a cocaine overdose?**
   a. Diazepam IV
   b. Furosemide IV
   c. Metoprolol IV
   d. Oral hydrochlorothiazide
   e. Nifedipine SL

10. **Which of the following medications should NOT be given if the patient has been taking sildenafil?**
    a. ACE inhibitors
    b. β-Blockers
    c. Hydralazine
    d. Nitrates

11. **Hypertensive urgency requires normalization of BP prior to leaving the ED.**
    a. True
    b. False

Physician CME Information

Date of Original Release: June 1, 2010. Date of most recent review: March 15, 2010. Termination date: June 1, 2013.

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals & Objectives: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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In October 2009, we launched a new free benefit for *Emergency Medicine Practice* subscribers: *EM Practice Guidelines Update*. This monthly online publication was created to help emergency clinicians stay current with clinical policies and practice guidelines that can assist clinical decision-making in the emergency department. The practice guidelines are drawn from a variety of specialty sources, are analytically reviewed using a systematic approach, and include an editorial commentary, if indicated. We hope this free, online, interactive publication will become a valued resource to our *Emergency Medicine Practice* subscribers and will help facilitate best practice in emergency care. To begin staying up-to-date in less than an hour per month, download the June issue, "Current Guidelines For The Diagnosis And Management Of Thoracic Aortic Disease," at www.ebmedicine.net/EMPGU.

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Hypertension In The Emergency Department: Treat Now, Later, Or Not At All

McKinnon M, O'Neill J. June 2010, Volume 12; Number 6

This issue of *Emergency Medicine Practice* reviews the current evidence related to the diagnosis and management of hypertension with a focus on issues related to emergency department clinical decisionmaking. *For a more detailed discussion of this topic, including figures and tables, clinical pathways, and other considerations not noted here, please see the complete issue on the EB Medicine website at www.ebmedicine.net/topics.*

Key Points	Comments
Distinguish between hypertension, hypertensive urgency, and hypertensive emergency, and treat appropriately.	See Table 1, page 5.[1]
The patient with asymptomatic hypertension should usually have an outpatient visit to confirm the diagnosis before initiating antihypertensive therapy.	The recent EMCREG consensus panel recommends no ancillary testing in asymptomatic, healthy patients with a BP > 160/100 mm Hg, because many will not have hypertension when re-checked.[2] The ACEP Clinical Policy on Asymptomatic Hypertension also does not recommend routine testing in asymptomatic patients.[8]
Hypertension has been associated with a 70% increase in risk for atrial fibrillation, and this increased risk remained after adjustments for age, sex, and associated conditions.	A prospective observational study of 4731 patients illustrated a 39% increase in risk for atrial fibrillation for every 5-mm increment in atrial enlargement.[24] Atrial fibrillation increased the risk for stroke 3- to 5-fold after adjustments for other risk factors. Finally, the presence of atrial fibrillation doubles the all-cause mortality rate.[24]
Identify the undiagnosed hypertensive patient.	The rate of stroke in 4907 adults over 65 years increased from 6 to 17 per thousand person years when the systolic blood pressure increased from < 120 to ≥ 140 mm Hg.[31]
In hypertensive emergencies, tailor the workup and management to the organ system involved.[77]	Conditions that cause acute end-organ damage require rapid and controlled correction of the BP level. The most common forms of end-organ damage in hypertensive emergencies, in order of decreasing frequency, are (1) cerebral infarction or hemorrhage; (2) acute pulmonary edema; (3) hypertensive encephalopathy; (3) acute CHF; and (4) aortic dissection. **(See Clinical Pathway For Symptomatic Hypertension, page 15.)**
Compelling data indicate that immediately lowering BP in stroke patients leads to worsening neurologic outcomes.[79]	With the dearth of hard evidence, current treatment guidelines rely on expert opinion. Severely high BP (ie, SBP greater than 220 mm Hg or DBP greater than 120 mm Hg) can be treated with IV labetalol or nicardipine, with the goal of a 10% to 15% reduction.[81]
Tempting as it is to treat to a "normal" blood pressure, it should not be decreased more than 25% except in the case of aortic dissection.	The goal of antihypertensive therapy is unique in aortic dissection in that the target pressure is the lowest pressure tolerated by the patient. Systolic blood pressure levels of 100 to 120 are ideal.[94,95]

See reverse side for reference citations.

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