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RELIAS MEDIA

Update on the Management of Hypertension in the Emergency Department

It's 7 p.m. on Friday night and you just started your shift in the emergency department (ED). You look at the board and see that there are four patients in the waiting room. As you scan through their vitals, you notice that three of them have elevated blood pressures. The first patient is a 70-year-old male with a headache, dizziness, and confusion. You note that his blood pressure is 189/101 mmHg. The second patient is a 65-year-old female with a history of non-insulin dependent diabetes mellitus, obesity, and hyperlipidemia who is complaining of left ankle pain after she twisted her ankle. The triage note reports that the patient has had bilateral lower extremity edema for "some time." Her blood pressure is 175/80 mmHg. The third patient is a 35-year-old male who was sent in by urgent care for elevated blood pressure and a leg laceration. The patient's blood pressure is 170/80 mmHg.

You have to ask yourself a variety of questions: Is this isolated blood pressure elevation a transient event or is it related to sustained hypertension? Are these patients on antihypertensive treatment? Do they have primary care follow-up? But assuming these patients have sustained elevated blood pressure, the most important question for your evaluation of the patient is, "Should asymptomatic hypertensive patients be treated in the ED?" This article will provide emergency physicians with a roadmap to help navigate the evaluation and management of hypertension in the ED.

Epidemiology

Hypertension affects millions of people worldwide, with substantial costs to the healthcare system. According to 2014 figures from the American Heart Association (AHA), an estimated 77.9 million Americans older than age 20 have hypertension.[1] In addition, it is estimated that hypertension affects approximately 30% of the U.S. population, and that 1-6% of ED patients will present with severe hypertension. Of these, about one-third to one-half will experience end organ damage.[2]

Hypertension remains a major public health challenge. According to the Centers for Disease Control and Prevention (CDC), during 2015-2016, the overall prevalence of hypertension in the United States adult population was 29%, and this prevalence increased with age from 7.5% in the 18- to 35-year-old group, to 33.2% in the 40- to 59-year-old group, and to 63.1% in the those 60 years of age or older.[3] The prevalence was significantly higher in African Americans (48.3%) than in other ethnic groups, such as whites (27.8%), Hispanics (27.8%), or Asians (25.0%).[3,4]

EXECUTIVE SUMMARY

- Elevated blood pressure is common on initial ED assessment at triage; in about 30-40% of patients, it normalizes spontaneously when assessed prior to discharge.
- For patients with sustained asymptomatic hypertension, there is no utility to routine laboratory testing, with the exception of a serum creatinine in patients likely to experience poor follow-up.
- For patients with sustained hypertension, it is reasonable to initiate antihypertensive therapy in the ED to facilitate disposition and follow-up.
- For patients with a hypertensive emergency, treatment includes intravenous agents that target the specific organ system.
- For patients with a hypertensive emergency, the goal is a modest 20-25% reduction in mean arterial pressure during the first 60 minutes of therapy. The exception is for patients with an acute aortic dissection, for whom the goal is to rapidly achieve a systolic pressure of 100-140 mmHg and a heart rate of 60 or less.

In addition, observational studies have shown that people are being diagnosed with hypertension at younger ages and, consequently, there is a much higher population burden with hypertension.[5] According to the CDC data, the prevalence of hypertension from 1999 to 2016 was unchanged, but the prevalence of controlled hypertension increased from 1999 through 2010 and has remained the same through 2016.[3] This may be a result of lowering the cutoff for the diagnosis of hypertension. The 2017 American College of Cardiology (ACC)/AHA guidelines for hypertension, which are used to define hypertension for the purposes for this paper, lowered the blood pressure cutoff, thus increasing the population of individuals assigned the diagnosis of hypertension.[6] These guidelines are listed in Table 1.[7]

When healthcare providers see an elevated blood pressure, the initial thought often is to treat it immediately because untreated hypertension increases the risk for cardiovascular disease, including stroke, heart attack, and heart failure.[8-10] However, providers also realize that treatment in the ED does not come without consequences, including medication side effects, hypotension, and ischemic stroke, to name a few. Therefore, the evaluation and treatment of hypertension requires understanding and using the appropriate standards of care for each scenario. To do so requires an expert understanding of the fundamentals of the medical terminology. Unfortunately, there is no universally accepted definition of hypertension. Therefore, for purposes of this paper, it is important to establish a common ground from which to commence this discussion.

According to the 2017 ACC/AHA guidelines, hypertension is classified into prehypertension, stage 1 hypertension, and stage 2 hypertension. (*See Table 1.*) This seems relatively straightforward; however, these categories are based on the average of at least two blood pressure readings obtained on at least two separate occasions. This is not feasible in the ED; therefore, we need to be careful when labeling someone as hypertensive in the ED, as an elevated blood pressure obtained in triage may not be sustained when later assessed.[11,12]

The cutoffs for prehypertension, stage 1 hypertension, and stage 2 hypertension were based on observational studies of the stages of hypertension and the risk of cardiovascular disease. Although hypertension increases the risk of cardiovascular disease, those with hypertension also were found to have other comorbidities, such as diabetes and chronic kidney disease, that increased their risk of cardiovascular disease. Although genetics plays a role in the risk for hypertension, there are many modifiable risk factors for hypertension and cardiovascular disease that an individual can act on prior to starting treatment for hypertension.[7] (*See Table 2.*)

Furthermore, as the obesity epidemic continues to expand, physicians will see more patients with hypertension at younger ages who likely will have hypertension for most of their lives. In addition, there are various other issues that lead to patients with untreated hypertension presenting to the ED. For example, the lack of primary care and lack of access to insurance leads many individuals to come to the ED as their only source for medical care. Many of these patients are symptomatic by the time they come to the ED, while others simply have hypertensive urgency. The ED may not be the most appropriate place to start blood pressure control for patients, especially after only evaluating their blood pressure once. However, clinicians run the risk of not treating these patients and having them lost to follow-up.[11] These patients may come back to the ED eventually with complications of untreated hypertension, such as stroke or heart attack. Although this article will not fix the global health issue of hypertension, its goal is to present a standard for patients who present to the ED with hypertension.

Pathophysiology of Hypertension

Basic Blood Pressure Autoregulation

Blood pressure is known to be directly proportional to cardiac output (CO) and peripheral vascular resistance (TPR), using the formula: blood pressure = CO × TPR. This formula is deceptively simple, as both cardiac output and peripheral vascular resistance are subject to complex autoregulatory mechanisms involving systemic neurohormonal signaling and localized signaling from specific target organs.[13] For those patients with essential/primary hypertension, many factors are at play that are both intrinsic and exogenous to the patient, including gender, age, genetic factors, drugs/medications, salt intake, sodium regulation, insulin resistance, and obesity, to name a few.

Table 1. Categories of Hypertension According to the 2017 ACC/AHA Guidelines

Hypertension Categories	Description
Hypertension	**Normal** • Systolic: < 120 mmHg • Diastolic: < 80 mmHg **Elevated** • Systolic: 120-129 mmHg • Diastolic: < 80 mmHg **Stage 1 Hypertension** • Systolic: 130-139 mmHg • Diastolic: 80-89 mmHg **Stage 2 Hypertension** • Systolic: ≥ 140 mmHg • Diastolic: ≥ 90 mmHg
Hypertensive emergency	Acute elevation of blood pressure (≥ 180/120 mmHg) associated with end organ damage
Hypertensive urgency	Profound elevation of blood pressure without acute target organ dysfunction

These factors contribute to changes in both cardiac output and peripheral vascular resistance. Renal factors are some of the most important determinants of blood pressure; however, primary/essential hypertension can be attributed directly to renal disease in only approximately 2-5% of patients.[14,15]

Knowledge about the basic renin-angiotensin system is essential to understanding the pathogenesis of hypertension and is the basis for most of the treatments for hypertension. Briefly, the juxtaglomerular apparatus of the kidney monitors both renal perfusion and sodium concentration and modifies renal efferent and afferent blood flow accordingly.[13,15] The juxtaglomerular apparatus is a group of cells in the distal convoluted tubule of the glomerulus and includes the juxtaglomerular cells and the macula densa cells. The juxtaglomerular cells sense the blood flow in the afferent arteriole, acting as baroreceptors. The macula densa cells are chemoreceptors in the distal convoluted tubule that sense sodium concentration.

When renal afferent arteriolar flow decreases, the juxtaglomerular cells secrete renin. Renin converts angiotensinogen into angiotensin I. Angiotensin I is physiologically inactive until it reaches the lungs, where angiotensin-converting enzyme (ACE) converts angiotensin I into angiotensin II. Angiotensin II acts as a vasoconstrictor, which in turn increases blood pressure and subsequently causes increased blood flow to the afferent arterioles of the glomerulus.

Angiotensin II also acts directly and indirectly to increase sodium reabsorption. When angiotensin II is active, it stimulates the increased activity of sodium/ion transport throughout the glomerulus, leading directly to decreased sodium excretion. The other major effect of angiotensin II is to stimulate the adrenal glands to secrete aldosterone. Through the release of aldosterone, angiotensin II indirectly increases sodium reabsorption and water retention, with the ultimate effect of increasing blood flow and maintaining afferent glomerular flow.

The macula densa cells also affect blood pressure via autoregulatory signaling that influences the renin release from the juxtaglomerular cells. Decreased sodium concentration in the distal convoluted tubule is sensed by the macula densa, which in turn signals the juxtaglomerular cells to increase renin release. As discussed above, this will increase systemic blood pressure. Meanwhile, the macula densa signals the afferent arteriole to dilate, bringing more blood flow to the glomerulus and increasing the glomerular filtration rate.

The renin-angiotensin system affects the peripheral vascular resistance portion of the blood pressure = CO × TPR equation. Cardiac output is the product of stroke volume × heart rate, and anything that affects either of these will alter cardiac output. Venous return and the metabolic demands of the body tissues represent major determinants of cardiac output. Although cardiac output is an important modulator of blood pressure, it is not a direct target of medications used in the management of hypertension.

Pathogenesis of Hypertension and End Organ Damage

The pathogenesis of hypertension is complex and related to multiple factors both intrinsic and extrinsic to the patient. One recognized cause of both short-term and long-term hypertension is the sympathetic nervous system.[13-15] Acutely, the release of endogenous endorphins will cause vasoconstriction and an increased heart rate, which in turn causes increased cardiac output and blood pressure. Chronically increased autonomic tone is thought to be a major mediator in the development of hypertension. Patients with autonomic imbalance favoring increased sympathetic tone often are found to have a higher resting heart rate. Additionally, when there is ongoing autonomic stimulation of peripheral vasculature, vascular remodeling has been noted. Increased autonomic tone also has been implicated in cardiac remodeling and the development of left ventricular hypertrophy. Chronic stimulation of the renin-angiotensin-aldosterone system also occurs in patients with increased sympathetic tone. Factors associated with the development of hypertension, such as obesity, insulin resistance, and obstructive sleep apnea, have been found to increase sympathetic tone.[13,14,16]

Vascular remodeling occurs in the setting of chronic blood pressure elevations. This can be macrovascular or microvascular.[14,15,17] Left ventricular hypertrophy and diastolic dysfunction often occur in the setting of chronic hypertension due to increased peripheral vascular resistance and changes in

Table 2. Modifiable Risk Factors for Hypertension and Cardiovascular Disease

- Cigarette smoking/tobacco
- Diabetes
- Dyslipidemia
- Overweight/obesity
- Physical inactivity
- Low fitness level
- Unhealthy diet

Source: Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol* 2018;71:e127-e248.

cardiac perfusion associated with capillary pressure autoregulation. Increased blood pressures can cause barotrauma to large vasculature, leading to endothelial dysfunction, oxidative stress, and stiffening of vascular walls, making them less responsive to autoregulatory mechanisms. Peripheral vasculature is somewhat buffered from chronic elevation of blood pressure because of the effect of vasoconstriction in the capillary beds. However, when blood pressure is chronically elevated, the capillary beds will reach a state of chronic vasoconstriction and luminal narrowing, leading to overall increased peripheral vascular resistance.

Renal injury is a known mediator of hypertension.[13,15,17] Most patients with hypertension will have normal kidney function, but over the long term, damage to the microvasculature of the kidney can lead to changes in the functionality of the renin-angiotensin-aldosterone system, in turn leading to worsening or ongoing hypertension. Because of the insidious nature of microangiopathic damage over time, kidney function impairment may be slow in onset or subclinical.

Genetic studies have found that a predisposition to hypertension runs in families.[13,15] Often, patients with hypertension also have a variety of other typical medical problems. Hyperlipidemia, insulin resistance, and obesity frequently are present in people with hypertension, and although these conditions often are associated, and various combinations of these disorders may cluster in families, it is unlikely that there is a common monogenic cause. Clinicians have recognized this cluster of maladies as metabolic syndrome. It is helpful to keep in mind that if patients have one of these diseases, they likely already have or will be prone to one or more other aspects of metabolic syndrome.

Asymptomatic Hypertension and Hypertensive Urgency in the ED

As already noted, hypertension affects a large proportion of adults in the United States, and only about half of those with hypertension receive adequate treatment.[18] The patient may have come to the ED specifically for hypertension, as a result of an elevated blood pressure found using an electronic sphygmomanometer at home, in a store, during a community health fair screening, or in a physician's office, or the elevated blood pressure may be an incidental finding during an ED visit for another reason. Approximately 40% of patients who present to the ED have an elevated blood pressure.[19] The number of patients presenting to EDs with elevated blood pressure is significantly higher than the number presenting to primary care settings who are found to have elevated blood pressure.

Since approximately one-third of adults in the United States have hypertension, it is likely that the etiology of the elevated blood pressure readings for many of these patients in the ED is essential hypertension, but many other causes of acute hypertension must be considered. These include sympathomimetic drug ingestions, alcohol use, substance withdrawal, exogenous steroid use, drug-drug interactions, nonsteroidal anti-inflammatory drugs, anxiety, and pain, although in most of these latter cases, the patient would not be asymptomatic. When a patient presents with symptomatic hypertension, a course of treatment often is clear (or clearer), but when a patient is referred to the ED expressly for hypertension without any symptoms or is found to have hypertension incidentally but has no complaints consistent with a hypertensive emergency, it often is difficult to determine the correct path of treatment.

Asymptomatic hypertension is referred to as "hypertensive urgency." By definition, hypertensive urgency is an acute elevation of blood pressure, often markedly so, without evidence of end organ damage. It may be difficult to distinguish those patients with an acute elevation of blood pressure from those patients who have markedly elevated blood pressure at baseline, so emergency physicians must keep the possibility of hypertensive urgency in mind. There is no validated evidence that treatment of patients with hypertensive urgency/markedly elevated asymptomatic blood pressure has any utility in decreasing short-term risk. Although referral for outpatient treatment is indicated for these patients, certain circumstances, such as discharge to prison, a nursing facility, or a shelter, may mandate treatment in the ED with a prescription for continued treatment upon discharge. Staff at these facilities may be uncomfortable receiving a patient with elevated blood pressure even if the patient is asymptomatic. Until there is more widespread education that asymptomatic hypertension is not acutely dangerous, these facilities may not accept patients until the blood pressure is lowered and/or the patient is started on antihypertensive treatment. Although this acute treatment is not based on any scientific evidence, these patients can be lost to follow-up easily, so the initiation of treatment in the ED with provision of a prescription and directions for continued treatment can provide an additional motivation to the patient to follow up for continued care. The pharmacological treatment options are listed in Table 3.

Management and Treatment

An American College of Emergency Physicians (ACEP) clinical policy from 2013 addresses the conundrum of asymptomatic hypertension in the ED. This clinical policy comments on both the evaluation and treatment of asymptomatic hypertensive patients in the ED. The ACEP clinical policy states that the emergency physician does not

Table 3. Treatment of Asymptomatic Hypertension When There Is a Concern That a Patient May Be Lost to Follow-up

Drug Class	Drug	Starting (Low) Dose	Patient Category	Special Considerations
Thiazide diuretic	Hydrochlorothiazide	• 12.5 to 25 mg qDay	• Non-African American adults (> 18 years old) • African American adults • Patients with diabetes mellitus	• Avoid in patients with chronic kidney disease
Calcium channel blocker	Diltiazem Verapamil Amlodipine Nifedipine	• 120 to 180 mg qDay • 120 mg qDay • 2.5 to 5 mg qDay • 30 mg qDay	• Non-African American adults • African American adults • Patients with diabetes mellitus	• May cause worsening peripheral edema
Angiotensin-converting enzyme inhibitor	Lisinopril Captopril Enalapril	• 5 to 10 mg qDay • 12.5 to 50 mg BID • 5 to 10 mg qDay	• Non-African American adults • Patients with diabetes mellitus • Patients with chronic kidney disease	• Avoid in patients with a personal or family history of angioedema • Avoid in pregnancy
Angiotensin receptor blocker	Losartan Valsartan	• 50 mg qDay • 80 mg qDay	• Non-African American adults • Patients with diabetes mellitus • Patients with chronic kidney disease	• Avoid in pregnancy

Source: Weber MA, Schiffrin EL, White WB, et al. Clinical practice guidelines for the management of hypertension in the community: A statement by the American Society of Hypertension and the International Society of Hypertension. *J Clin Hypertens* 2014;16:14-26

need to perform routine clinical screening to identify end organ dysfunction if the patient presenting with hypertension is asymptomatic.[18] An exception is those patients who have poor follow-up, as routine screening for elevated creatinine may identify patients who require admission for blood pressure management.

In creating this clinical policy, ACEP reviewed several studies that assessed the utility of screening for hypertension using serum chemistry, electrocardiogram (ECG), and chest X-ray.[18,19] The studies reviewed for this policy found that these screening tests resulted in very few changes in patient management based on the results in the setting of an asymptomatic elevated blood pressure. Of all the screening tests that were done in the setting of asymptomatic, markedly elevated blood pressure, serum creatinine was the only test with evidence for utility that might affect ED disposition.[20] The 2013 ACEP clinical policy noted that the creatinine level will detect a small proportion of patients with impaired renal function who present with asymptomatic hypertension, but it is unclear if this differs from those with normal or near-normal blood pressure. Overall, this ACEP clinical policy gave a Level C recommendation to the principle that screening for target organ damage is not required for ED patients with asymptomatic, markedly elevated blood pressure, but can be considered in select patient populations, such as those with poor follow-up.[20]

Many patients who present to the ED do not have easy or any access to outpatient follow-up care and, therefore, are vulnerable to being lost to follow-up despite any recommendations made during their visit to the ED. For these patients, the ED represents a point of contact with the medical system that could serve to better risk stratify and treat those with poor outpatient access.

A 2016 study evaluated patients in an urban ED with elevated blood pressure and screened for evidence of cardiac strain using focused, bedside echocardiogram performed by the emergency physicians.[21] Although this study was small, researchers found that 39% of those screened had some evidence of subclinical heart disease likely related to their hypertension. Although the ACEP clinical policy does not advocate for cardiac screening in the setting of asymptomatic hypertension, this study indicates that a large percentage of patients with hypertension in the ED are likely to have manifestations of the long-term effects of untreated hypertension. This study does not discuss other forms of screening, such as those discussed in the ACEP clinical policy, nor will every emergency physician be able to perform bedside echocardiograms, but it does demonstrate that even if hypertension is subclinical, the manifestations of untreated hypertension are likely to be present in a large percentage of patients with asymptomatic hypertension.

A follow-up study by this same group looked at whether a "brief ED hypertension education intervention" was useful.[22] This educational intervention, which took less than five minutes to perform, included information about the silent nature of hypertension as a disease, the long-term detrimental effects on health due to untreated hypertension, the fact that early subclinical heart disease can be seen on echocardiograms, and, finally, the fact that the changes associated with subclinical hypertensive heart disease potentially are reversible with proper treatment. This small study indicates that not only is it feasible to perform brief educational interventions in the ED regarding hypertension, but also it does seem to have an effect. Emergency practitioners should strongly consider addressing asymptomatic hypertension in vulnerable populations with little or no access to primary care and consider more aggressive treatment and referral.

The second part of the 2013 ACEP clinical policy addresses the treatment of asymptomatic hypertension in the ED.[18] The ACEP policy recommendation states, "in patients with markedly elevated blood pressure, routine ED medical intervention is not required." The consensus recommendations note that patients should be referred for follow-up, and in select patient populations, such as those patients who do not have access to follow-up, the ED physician may treat blood pressure in the ED and/or initiate outpatient antihypertensive treatment.

With respect to management of asymptomatic hypertension in the ED, emergency physician practice varies and is influenced by many factors.[23] A survey of emergency physicians demonstrated that most will advise outpatient follow-up to patients with asymptomatic hypertension in the ED. Conversely, despite evidence that rapid decreases of blood pressure in asymptomatic patients can lead to relative hypotension and patient harm, many physicians continue to administer antihypertensive medications to patients in the ED.[23-26]

In patients with chronic hypertension, clinicians must be aware that decreasing blood pressure to a "normal" level can be dangerous, as the threshold for adverse events, such as ischemia and decreased cerebral and renal blood flow, is seen with higher blood pressure values in those with chronic hypertension compared to the lower blood pressure values necessary to induce cerebral or renal ischemia in those without chronically elevated blood pressure. Factors that influence administration of antihypertensives in the ED include higher initial blood pressure measurements, a history of previously diagnosed hypertension, and primary care physician referral to the ED for blood pressure management.[23,24]

Before deciding to treat asymptomatic hypertension in the ED, the clinician must decide if a patient truly needs management. In a study of outpatients presenting to primary care for hypertension, researchers found that the risk of a major adverse cardiac event (MACE) was low in patients presenting with asymptomatic hypertension.[27] Often, referral to the ED for elevated blood pressure was because of concern for imminent MACE, although there was a < 1% incidence of MACE within six months. Referral to the ED, which often resulted in admission/acute treatment of asymptomatic hypertension, resulted in no better seven-day, 30-day, or six-month outcomes when compared to outpatient treatment alone.

If an emergency practitioner is going to treat asymptomatic hypertension in the ED, caution must be taken to ensure that the patient does not become hypotensive or relatively hypotensive. As the ACEP clinical policy advises, if an emergency physician opts to treat asymptomatic hypertension, the physician should take care to choose the appropriate antihypertensive medication. The provider also should ensure that no harm comes to the patient as a result of a desire to treat elevated blood pressure.

Intravenous (IV) antihypertensives have a much quicker onset of action than oral antihypertensives; therefore, care must be taken when deciding to administer these medications.[26] In one study among inpatients with asymptomatic hypertension who were treated with IV antihypertensive medications, more than 30% had a reduction of blood pressure of > 25%, putting them at risk for decreased end organ perfusion and potentially requiring additional medications or other interventions. For patients with asymptomatic hypertension, oral antihypertensive agents are likely to be safer, resulting in more modest blood pressure reduction over a longer period of time.

The eighth report from the Joint National Committee (JNC 8) (on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure) recommends that for any non-African-American person diagnosed with hypertension, including those with a co-diagnosis of diabetes mellitus, initial treatment for hypertension should be initiated using a thiazide-type diuretic, a calcium channel blocker (CCB), an ACE inhibitor, or an angiotensin receptor blocker (ARB).[28] The report further states that initial antihypertensive therapy for African-American patients should include a thiazide-type diuretic or a CCB. For patients with chronic kidney disease, initial antihypertensive treatment should be either an ACE inhibitor or an ARB. These recommendations do not address the initiation of treatment in the ED; however, they provide a starting point for emergency physicians who have decided to initiate outpatient antihypertensive treatment.

In a 2015 study, researchers looked at the utility of providing prescriptions for antihypertensive medications in the ED.[29] This study was a retrospective data study of two prospective, longitudinal, randomized, controlled trials. Antihypertensives prescribed in the study included thiazide-type diuretics, ACE inhibitors, CCBs, and beta-blockers. The authors found that patients in whom antihypertensive medications were initiated in the ED had statistically significantly lower blood pressures at outpatient follow-up. Additionally, the rate of adverse events was not different between those patients prescribed an antihypertensive and those who were in the control group.

Clonidine, which often is chosen because of its fast onset of action, ease of dosing, and low cost, is not an ideal initial medication for treatment of hypertension, as it has variable effects on blood pressure and cerebral

Table 4. Antihypertensive Medications That Increase Intracranial Pressure

- Nitroprusside
- Nitroglycerin
- Hydralazine

perfusion. In addition, clonidine discontinuation may prompt repeat ED visits for rebound hypertension in noncompliant patients.[24]

In conclusion, emergency physicians face much ambiguity regarding treatment of patients with asymptomatic hypertension. As long as there is no indication of end organ damage that suggests hypertensive emergency rather than asymptomatic hypertension, it is not necessary to perform routine screening tests while a patient is in the ED. Patients who lack resources for outpatient evaluation may benefit from serum chemistry to ensure that they do not have kidney injury associated with the elevated blood pressure, although this still is unlikely to identify many patients who would be managed differently. Patients who have hypertension and are truly asymptomatic will not be harmed by withholding treatment of the blood pressure in the ED, and primary care follow-up is a reasonable course of action. If the emergency physician believes that treatment is indicated, oral antihypertensive medications may be a better option for acute treatment of asymptomatic hypertension, as the risk of causing significant blood pressure reduction requiring intervention for hypotension or end organ damage is greater when using IV antihypertensive medications. Finally, for those patients who do not have easy access to primary care, it is reasonable and safe to initiate oral antihypertensives in the ED.

Hypertensive Emergency

Hypertensive emergency is a broad term involving target end organ damage associated with a blood pressure greater than 180/120 mmHg. The primary symptoms or chief complaints when dealing with a hypertensive emergency include chest pain, back pain, numbness, tingling, shortness of breath, difficulty speaking, weakness, diminished visual acuity, or altered mental status. However, the goal of treatment despite the complaint is the same: to lower the mean arterial pressure and blood pressure and prevent further target organ damage. "Definitive treatment of any hypertensive emergency is the acute reduction of blood pressure to prevent ongoing organ damage."[1]

Table 5. Complications of Hypertensive Emergency

Organ	Specific Damage Associated With Hypertensive Emergency
Heart	Acute aortic dissection, acute pulmonary edema, acute coronary syndrome, acute congestive heart failure
Brain	Transient ischemic attack, cerebral infarction, seizure, hypertensive encephalopathy, intracerebral bleed
Kidney	Acute kidney injury
Eyes	Retinal edema, papilledema, hemorrhages

Sources: Tintinalli JE, Stapczynski JS, Ma OJ, et al. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th edition. New York; McGraw-Hill: 2015: 399-409; Suneja M, Sanders ML. Hypertensive emergency. *Med Clin North Am* 2017;101:465-478.

There are several evidence-based guidelines that clinicians can use to treat hypertension effectively, such as the aforementioned 2017 ACC/AHA and 2014 JNC 8, but these most recent guidelines, including JNC 8, do not provide guidance regarding hypertensive emergency. Furthermore, the 2013 ACEP clinical policy recommendations also do not have a specific recommendation regarding hypertensive emergency.[30]

The 2003 JNC 7 guidelines did comment on hypertensive emergencies. According to the JNC 7 guidelines, hypertensive emergency treatment should commence within the first hour and "[t]he severity of hypertensive crises is not related to the BP level but to the associated organ damage."[31] The most frequent presenting symptoms of hypertensive emergency are cardiogenic pulmonary edema (31% of cases); stroke (22%); myocardial infarction (18%); aortic dissection (8%); acute renal failure (6%); and encephalopathy (5%).[31] However, almost 50% of patients present with nonspecific symptoms, including vomiting, headache, and epistaxis.

The goal of treatment for hypertensive emergency is to reduce the blood pressure gradually without causing further end organ damage from malperfusion. Target end organs, such as the brain, kidneys, and heart, are sensitive to sudden changes in systemic blood pressure.[30] Many neurologic sequelae, such as posterior reversible encephalopathy syndrome, stroke, and seizure, can occur from rapid reductions in blood pressure. Although many IV medications are available to treat a hypertensive neurologic emergency, it is best to avoid ones that increase intracranial pressure, such as some commonly used direct vasodilators. (*Table 4 lists medications that increase intracranial pressure.*) The preferred IV agents when treating a neurological hypertensive emergency include the calcium channel blockers nicardipine and clevidipine and the combination alpha- and beta-adrenergic blocker labetalol.[30]

Because the presenting symptoms of hypertensive emergency include ocular, neurologic, and cardiac emergencies such as those listed in Table 5, the initial physical examination is critical for the diagnosis of hypertensive emergency. During the initial exam, several accurate blood pressure assessments should be obtained.[32] Additionally, the physician should obtain a full medical history of the patient, including the duration of patient awareness of and treatment for hypertension; prior episodes of uncontrolled blood pressure; use of other drugs, both prescription and illict, that

Table 6. Type of Hypertensive Emergency and Specific Treatment

Type of Hypertensive Emergency	Treatment
Aortic dissection	Esmolol followed by nitroprusside (or labetalol)
Acute heart failure (pulmonary edema)	Nitroglycerin and loop diuretic, or nitroprusside
Ischemic stroke	Labetalol, clevidipine, nicardipine
Hemorrhagic stroke	Nicardipine or labetalol
Myocardial infarction	Nitroglycerin, labetalol or other beta-blockers
Cocaine-induced myocardial ischemia	Phentolamine and benzodiazepines

Sources: *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th edition. New York; McGraw-Hill: 2015: 399-409; Bauman BM, Cline DM, Pimenta E. Treatment of hypertension in the emergency department. *J Am Soc Hypertens* 2011;5:366-377.

may increase blood pressure; and risk factors for cardiovascular disease.[33] The ED provider also should ask specifically about chest pain, back pain, shortness of breath, headaches, vision changes, and other neurologic symptoms.

After taking a thorough history and placing the patient on a cardiac monitor, the provider should focus on the physical exam of the patient. During the physical exam, the provider should pay close attention to the funduscopic exam and cardiopulmonary exam, and should obtain an ECG, basic blood work (including creatinine and type and screen), and urinalysis to evaluate for end organ damage. The provider also should order a chest X-ray if there is concern about cardiopulmonary sequelae or a computed tomography (CT) scan of the brain if there is concern about neurologic sequelae, including but not limited to aortic dissection and stroke.[34] If available, a bedside echocardiogram can identify regional wall motion abnormalities of a myocardial infarction or global hypokinesis of heart failure.

Furthermore, symptomatic resistant hypertension can be the result of damage or dysfuction of other organ systems. Some of the causes of resistant hypertension include head trauma, autonomic dysfunction, spinal cord injury, renovascular hypertension, hyperthyroidism, Cushing syndrome, primary aldosteronism, and pheochromocytoma. Additionally, other causes include drugs and alcohol, high salt intake, and pregnancy conditions such as preeclampsia and eclampsia.[32] In patients with resistant hypertension, renal ultrasound should be considered as well.

Management and Treatment

The two key principles when treating an acute hypertensive emergency are: choose agents specific to the organ system involved; and rapidly lower the blood pressure enough to stop ongoing target organ damage while avoiding too severe a reduction that may produce cerebral, myocardial, or renal ischemia. For most hypertensive emergencies, the second principle is achieved by having a goal of a 20-25% reduction in mean arterial pressure (MAP) within the first 60 minutes.[36] An exception to this second principle is for patients with an acute aortic dissection. A more rapid reduction in blood pressure is desired to stop propagation of the dissecting intramural hematoma. Table 6 lists types of hypertensive emergencies and common treatments. Table 7 lists common drugs used for hypertensive emergency.

Aortic Dissection

Aortic dissection is one of the most critical types of hypertensive emergency, as mortality may occur within hours. In patients with aortic dissection, the goal primarily is a reduction in heart rate to reduce the shearing forces and stress on the aorta. Secondarily, the blood pressure needs to be lowered to prevent worsening of the dissection. The goal of treatment is a systolic blood pressure between 100-140 mmHg and a heart rate less than or equal to 60 beats per minute.[2] Therefore, a short-acting beta-blocker, such as esmolol, followed by a vasodilator, such as nitroprusside, is the preferred treatment.

Myocardial Infarction

Blood pressure control in myocardial infarction helps decrease myocardial work, wall stress, and, therefore, the need for additional oxygen. According to the ACC/AHA, IV nitrates are the preferred treatment for myocardial infarction. Intravenous nitrates improve perfusion to the heart and decrease left ventricular preload. Concomitant beta-blockers also help lower heart rate and blood pressure.[2] However, IV beta-blockers have several contraindications and are not to be used in patients with heart failure, cardiogenic shock, heart block, and those dependent on beta-agonists for their respiratory status.

Cocaine-induced/ Sympathomimetic-induced Myocardial Ischemia

In patients with sympathomimetic-induced myocardial ischemia, such as from cocaine or amphetamine use, the first-line medication is IV benzodiazepines. If these are ineffective, the next line medication is phentolamine or nitroglycerin. Beta-blockers have been avoided when treating cocaine-induced chest pain because of the concept that beta-blockade would leave unopposed alpha-adrenergic activity to produce complications. Case series and a recent meta-analysis have failed to substantiate an adverse effect of beta-blocker therapy in cocaine-induced chest pain.[37]

Acute Heart Failure and Pulmonary Edema

The goal of therapy in acute heart failure and pulmonary edema is to

Table 7. Common Drugs Used for Hypertensive Emergency

Drug	Dose	Onset	Adverse Reaction
Esmolol	250 to 500 mcg/kg/min bolus followed by drip	1-2 minutes	Hypotension, heart failure
Hydralazine	10 to 20 mg IV	10-20 minutes	Tachycardia, headache, flushed face
Labetalol	20 to 80 mg bolus then infusion	5-10 minutes	Orthostatic hypotension, vomiting, bronchoconstriction, dizziness, heart block
Nicardipine	5 to 15 mg/h IV	5-10 minutes	Headache, flushed face
Nitroglycerin	5 to 100 mcg/min	2-5 minutes	Methemoglobinemia, headache, tolerance
Nitroprusside	0.25 to 10 mcg/kg/min	seconds	Thiocyanate and cyanide intoxication Muscle twitching
Phentolamine	5 to 15 mg IV bolus	1-2 minutes	Tachycardia, flushed face

Sources: Adebayo O, Rogers RL. Hypertensive emergencies in the emergency department. *Emerg Med Clin North Am* 2015;33:539-551; Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. *Hypertension* 2003;42:1206-1252.

reduce preload and afterload, as well as diuresis. This effect can be achieved by using nitrates and loop diuretics.

Ischemic Stroke and Intracerebral Hemorrhage

For acute ischemic stroke patients, antihypertensive treatment is recommended only if the patient is eligible for IV alteplase or mechanical thrombectomy, with the goal of a systolic less than 185 mmHg and a diastolic of less than 110 mmHg.[38] For other ischemic stroke patients not eligible for fibrinolytic or thrombectomy therapy, there is no consistent evidence that lowering even markedly elevated blood pressure is beneficial.[39] The best guidance is to follow the locally developed consensus protocol for the the management of such patients.

Elevated blood pressure is common in spontaneous acute intracerebral hemorrhage, and poor outcomes are associated with elevated systolic blood pressures. Using IV agents to decrease the systolic blood pressure in these patients to between 160 and 140 mmHg generally is safe and is recommended to improve functional outcome.[40]

Disposition

It should be noted that most of the recommended treatments for hypertensive emergency are based primarily on the opinions of experts and studies involving small population samples; therefore, it is vital that an ED physician evaluate each patient based on the specific symptoms presented combined with the patient's medical and pharmacological history, rather than solely on the medical journals and texts.

All patients with hypertensive emergency should be admitted to a cardiac care or intensive care unit for close blood pressure control, preferably through an arterial line. Subspecialty consultation, including cardiology, neurology, ophthalmology, or nephrology as needed, also should be obtained for these patients as well. The patient's mean arterial blood pressure should be monitored closely and brought down to target levels without precipitously decreasing blood pressure.

In conclusion, there are no uniformly agreed upon guidelines when dealing with hypertension in the ED. Hypertensive emergency warrants immediate treatment, while asymptomatic hypertension usually does not warrant immediate treatment unless there is concern that the patient will be lost to follow-up.

Case Conclusions

The first patient is hypertensive, dizzy, and confused and, therefore, is classified as hypertensive emergency. This patient should be treated immediately with IV antihypertensives, such as nicardipine, and requires further workup such as a CT scan, ECG, blood work, and cardiac monitoring.

The second patient presents with asymptomatic hypertension, since she presented with ankle pain and incidentally was found to have an elevated blood pressure. This patient's pain should be treated, and her blood pressure should be rechecked. If the patient's blood pressure remains elevated, the clinician can call the patient's primary doctor and arrange for close follow-up for a blood pressure recheck. However, if the patient is homeless, does not have a primary care physician, or if there is concern that the patient will be lost to follow-up, treatment may be initiated in the ED.

The third patient should have his laceration repaired and pain treated. If the patient still is hypertensive, he should be referred for close outpatient follow-up.

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CME/CE Questions

1. A 45-year-old otherwise healthy African American male presents to the ED for evaluation of knee pain. Incidentally, you note that his blood pressure is 185/91 mmHg. After you treat his pain, his blood pressure is still 173/89 mmHg. The patient states that he has no primary care provider and the soonest appointment he can get is in three months. You decide to treat his hypertension. What medications should be considered?
   a. Thiazide diuretic
   b. Clonidine
   c. Angiotensin-converting enzyme inhibitor
   d. Angiotensin receptor blocker
2. Angiotensin II has both direct and indirect effects on blood pressure. What is the mechanism of action?
   a. Cleaving angiotensinogen to form active angiotensin I, which in turn causes vasoconstriction and increased blood pressure, and stimulates aldosterone release causing indirect sodium reabsorption
   b. Direct vasoconstriction leading to increased blood pressure and increased blood flow to the kidneys, direct effect on the renal collecting system causing increased sodium reabsorption, and stimulation of aldosterone release
   c. Inhibits angiotensin-converting enzyme, which directly inhibits the release of active aldosterone, causing increased sodium and water excretion and subsequent blood pressure reduction
   d. Direct constriction of the glomerular afferent arteriole, which stimulates surrounding capillary beds to constrict, thereby increasing blood pressure systemically
3. A 65-year-old male presents with sudden onset of headache, dizziness, blurred vision, and hypertension. His blood pressure is 230/140 mmHg. Which of the following medications is the treatment of choice for this condition and will not increase intracranial pressure in this patient?
   a. Nitroprusside
   b. Nitroglycerin
   c. Hydralazine
   d. Nicardipine
4. Hypertensive emergency is defined as:
   a. blood pressure of ≥ 160/120 mmHg.
   b. blood pressure of ≥ 150/80 mmHg with evidence of end organ damage.
   c. blood pressure of ≥ 200/100 mmHg.
   d. blood pressure of ≥ 180/120 mmHg with evidence of end organ damage.
5. An 80-year-old female presents to the emergency department with chest pain. She says that her pain began an hour ago and "feels like something is tearing." Her blood pressure is 220/120 mmHg, heart rate is 120, respiratory rate 20, temperature 99°F, and O_2 saturation is 98% on room air. Her ECG shows a normal sinus rhythm with T-wave inversions in the inferior leads. You obtain a chest X-ray, which is concerning for a widened mediastinum. What is the appropriate pharmacological treatment prior to definitive therapy?
   a. Esmolol followed by nitroprusside drip
   b. Nitroprusside followed by esmolol drip
   c. Nitroprusside only
   d. Esmolol only

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- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

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Update on the Management of Hypertension in the Emergency Department

Categories of Hypertension According to the 2017 ACC/AHA Guidelines

Hypertension Categories	Description
Hypertension	**Normal** • Systolic: < 120 mmHg • Diastolic: < 80 mmHg **Elevated** • Systolic: 120-129 mmHg • Diastolic: < 80 mmHg **Stage 1 Hypertension** • Systolic: 130-139 mmHg • Diastolic: 80-89 mmHg **Stage 2 Hypertension** • Systolic: ≥ 140 mmHg • Diastolic: ≥ 90 mmHg
Hypertensive emergency	Acute elevation of blood pressure (≥ 180/120 mmHg) associated with end organ damage
Hypertensive urgency	Profound elevation of blood pressure without acute target organ dysfunction

Modifiable Risk Factors for Hypertension and Cardiovascular Disease

- Cigarette smoking/tobacco
- Diabetes
- Dyslipidemia
- Overweight/obesity
- Physical inactivity
- Low fitness level
- Unhealthy diet

Source: Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol* 2018;71:e127-e248.

Treatment of Asymptomatic Hypertension When There Is a Concern That a Patient May Be Lost to Follow-up

Drug Class	Drug	Starting (Low) Dose	Patient Category	Special Considerations
Thiazide diuretic	Hydrochlorothiazide	• 12.5 to 25 mg qDay	• Non-African American adults (> 18 years old) • African American adults • Patients with diabetes mellitus	• Avoid in patients with chronic kidney disease
Calcium channel blocker	Diltiazem Verapamil Amlodipine Nifedipine	• 120 to 180 mg qDay • 120 mg qDay • 2.5 to 5 mg qDay • 30 mg qDay	• Non-African American adults • African American adults • Patients with diabetes mellitus	• May cause worsening peripheral edema
Angiotensin-converting enzyme inhibitor	Lisinopril Captopril Enalapril	• 5 to 10 mg qDay • 12.5 to 50 mg BID • 5 to 10 mg qDay	• Non-African American adults • Patients with diabetes mellitus • Patients with chronic kidney disease	• Avoid in patients with a personal or family history of angioedema • Avoid in pregnancy
Angiotensin receptor blocker	Losartan Valsartan	• 50 mg qDay • 80 mg qDay	• Non-African American adults • Patients with diabetes mellitus • Patients with chronic kidney disease	• Avoid in pregnancy

Source: Weber MA, Schiffrin EL, White WB, et al. Clinical practice guidelines for the management of hypertension in the community: A statement by the American Society of Hypertension and the International Society of Hypertension. *J Clin Hypertens* 2014;16:14-26

Antihypertensive Medications That Increase Intracranial Pressure

- Nitroprusside
- Nitroglycerin
- Hydralazine

Complications of Hypertensive Emergency

Organ	Specific Damage Associated With Hypertensive Emergency
Heart	Acute aortic dissection, acute pulmonary edema, acute coronary syndrome, acute congestive heart failure
Brain	Transient ischemic attack, cerebral infarction, seizure, hypertensive encephalopathy, intracerebral bleed
Kidney	Acute kidney injury
Eyes	Retinal edema, papilledema, hemorrhages

Sources: Tintinalli JE, Stapczynski JS, Ma OJ, et al. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th edition. New York; McGraw-Hill: 2015: 399-409; Suneja M, Sanders ML. Hypertensive emergency. *Med Clin North Am* 2017;101:465-478.

Type of Hypertensive Emergency and Specific Treatment

Type of Hypertensive Emergency	Treatment
Aortic dissection	Esmolol followed by nitroprusside (or labetalol)
Acute heart failure (pulmonary edema)	Nitroglycerin and loop diuretic, or nitroprusside
Ischemic stroke	Labetalol, clevidipine, nicardipine
Hemorrhagic stroke	Nicardipine or labetalol
Myocardial infarction	Nitroglycerin, labetalol or other beta-blockers
Cocaine-induced myocardial ischemia	Phentolamine and benzodiazepines

Sources: *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th edition. New York; McGraw-Hill: 2015: 399-409; Bauman BM, Cline DM, Pimenta E. Treatment of hypertension in the emergency department. *J Am Soc Hypertens* 2011;5:366-377.

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Labetalol	20 to 80 mg bolus then infusion	5-10 minutes	Orthostatic hypotension, vomiting, bronchoconstriction, dizziness, heart block
Nicardipine	5 to 15 mg/h IV	5-10 minutes	Headache, flushed face
Nitroglycerin	5 to 100 mcg/min	2-5 minutes	Methemoglobinemia, headache, tolerance
Nitroprusside	0.25 to 10 mcg/kg/min	seconds	Thiocyanate and cyanide intoxication Muscle twitching
Phentolamine	5 to 15 mg IV bolus	1-2 minutes	Tachycardia, flushed face

Sources: Adebayo O, Rogers RL. Hypertensive emergencies in the emergency department. *Emerg Med Clin North Am* 2015;33:539-551; Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. *Hypertension* 2003;42:1206-1252.

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