Treatment of Asymptomatic Severe Hypertension in the Emergency Department
An Acute Finding of a Chronic Condition

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ABSTRACT
Severely elevated blood pressure (greater than 180/110 mmHg) is a common finding encountered in the emergency department. This blood pressure level has traditionally been categorized as hypertensive urgency when there is no target organ damage or a hypertensive emergency when target organ damage is present. Asymptomatic severely elevated blood pressure is the preferred term instead of hypertensive urgency, as it does not imply a crisis exists. The assessment and management of patients with elevated blood pressure has been extensively studied in the primary care environment. However, most of the guidelines are not for patients with acute illness. Current guidelines are being challenged as not being evidence-based, impractical, and cost-inefficient when applied in the emergency department setting (D. J. Karras et al., 2006). There is general consensus that if treatment is initiated, with an aim to gradually lower the blood pressure level over 24 to 48 hr, then no attempt should be made to rapidly lower it. **Key words:** asymptomatic severely elevated hypertension, blood pressure, emergency department, hypertensive emergency, hypertensive urgency, severely elevated blood pressure, target organ damage

HYPERTENSION (HTN) is the most common primary diagnosis in America. It affects approximately 50 million individuals in the United States and approximately 1 billion worldwide. Despite numerous reports on the benefits of blood pressure (BP) control, 30% are still unaware they have HTN (National Heart, Lung, and Blood Institute [NHLBI], 2003). Approximately 5% of adult emergency department (ED) patients have at least one BP measurement that is severely elevated (Karras et al., 2005).

There is discrepancy in the literature regarding the criteria used to define severely elevated blood pressure (SEBP). The lack of a consistent definition of HTN extremes has resulted in variable data collection in published studies, making comparison difficult, and much of the literature is old (Flanigan & Vitberg, 2006). Most HTN guidelines refer to SEBP as a systolic blood pressure (SBP) greater than 180 mmHg, and a diastolic blood pressure (DBP) greater than 110 mmHg (Karras et al., 2006). This is an adaptation of the Sixth Report of the Joint National Committee (JNC) on prevention, detection, evaluation, and treatment of high BP (NHLBI, 1997).
Traditionally, SEBP is divided into the categories of hypertensive emergency (HE) or hypertensive urgency. An HE refers to the association with target organ damage (TOD) and requires immediate and careful BP reduction. Hypertensive urgency does not involve TOD (Marik & Varon, 2007). However, there is little consensus on what constitutes a hypertensive urgency or even if such a condition exists. The term asymptomatic SEBP is preferred, as it does not imply that a crisis necessarily exists (Shayne & Pitts, 2003).

ED patients with SEBP present with an array of symptoms, varying from life-threatening to minor complaints. This article will specifically address the patient who presents with a minor complaint that is not related to HTN or suggestive of TOD and has an incidental finding of SEBP. This will be referred to as asymptomatic SEBP.

**PATHOPHYSIOLOGY**

BP is controlled by a complex interaction of several physiologic mechanisms. A disturbance in any one of these mechanisms can cause a problem with BP control. There are still many things about HTN that are not fully understood. Between 2% and 5% of HTN patients have an underlying renal or adrenal disease. The remaining patients have essential HTN; that is, there is no known cause. Some of the many interrelated factors involved in BP control include cardiac output (CO), peripheral vascular resistance (PVR), the renin-angiotensin system, autonomic nervous system, endothelial cells, and genetics (Beevers, Lip, & O’Brien, 2001).

The renin-angiotensin system is an important endocrine system involved in BP maintenance. Renin is secreted from the kidneys in response to low perfusion or low salt intake. This leads to a cascade in which angiotensinogen is converted to angiotensin I. Angiotensin I is then converted to angiotensin II, which is a potent vasoconstrictor, by angiotensin-converting enzyme (ACE). It also stimulates the release of aldosterone, which retains sodium and water. The interaction between the renin-angiotensin system and autonomic nervous system, along with other factors such as sodium, circulating volume, vasoactive substances, and genetics all play a role in HTN (Beevers et al., 2001).

A balance between CO and PVR is required to maintain a normal BP level. Most patients with essential HTN have normal CO, but raised PVR. One thought is that the process starts with increased CO as a response to sympathetic overactivity, and the arteriolar vessel walls constrict to compensate and prevent the raised pressure from being transmitted to the capillary bed. The prolonged smooth muscle constriction is thought to cause structural changes and thickening of the vessel wall, which causes an irreversible increase in PVR (Beevers et al., 2001). The result of this persistent mechanical stress ultimately damages the vessel wall, leading to increased vascular permeability, deposition of fibrin, activation of the coagulation cascade and platelets, and cell proliferation. A cycle of vascular reactivity, release of vasoconstrictors, endothelial damage, platelet aggregation, myointimal proliferation, and progressive narrowing of arterioles continues (Tintinelli, Kelen, & Stapczynsky, 2004). This state ultimately leads to fibrinoid necrosis of the arterioles. These collective mechanisms are responsible for end-organ hypoperfusion, ischemia, and dysfunction (Marik & Varon, 2007).

Increased systemic vascular resistance causes left ventricular hypertrophy. As the left ventricular wall thickens, the cavity dilates, function deteriorates, and signs and symptoms of congestive heart failure appear. Elevation in BP increases afterload, resulting in greater myocardial work and oxygen requirements. Coronary artery disease and increased myocardial oxygen requirements may cause angina or myocardial infarction (Kasper et al., 2005).

In addition, central nervous system disorders occur frequently in patients with long-standing HTN. Atherosclerosis is common in those with HTN and may cause cerebral infarction. Elevated arterial pressure and the development of cerebral vascular
microaneurysms can cause cerebral hemorrhage (Kasper et al., 2005). HTN may cause acute renal failure or exacerbate chronic renal failure, whereas renal disease may result in HTN. Decreased glomerular filtration and tubular dysfunction are caused by atherosclerotic lesions of the afferent and efferent arterioles and glomerular capillary tufts. Renal failure is responsible for 10% of deaths caused by HTN (Kasper et al., 2005).

Retinopathy is a directly observable example of atherosclerotic changes. The clinical manifestations vary according to the degree and rapidity of rise in BP and the underlying state of the ocular circulation. The retinal arterioles become more tortuous and narrow and develop abnormal light reflexes. There is increased venous compression at the retinal arteriovenous crossings. Acute elevations of BP result in loss of autoregulation in the retinal circulation, leading to the breakdown of endothelial integrity and occlusion of pre-capillary arterioles and capillaries (McPhee, Papadakis, & Tierney, 2008).

DIFFERENTIAL DIAGNOSES/SIGNS AND SYMPTOMS

One of the first steps in evaluating HTN is to determine whether the BP reading is reliable. JNC VII (NHLBI, 2003) recommends that BP be taken after a person has been seated quietly for at least 5 min in a chair, with feet on the floor and arm supported at heart level. This is rarely the way an initial BP is taken in the ED. BP should be taken in both arms with the appropriate size cuff. Initial BP elevations are often lower when a second reading is obtained. Appropriate pain management and relief of underlying causes such as bladder distention may resolve the elevation. Certain medications and illicit drugs may raise BP, such as oral contraceptives, steroids, non-steroidal anti-inflammatory drugs, nasal decongestants, cold remedies, appetite suppressants, tricyclic antidepressants, monoamine oxidase inhibitors, or cocaine (Marik & Varon, 2007).

Dieterle, Schuurmans, Strobel, Battegay, and Martina (2005) conducted a prospective study to investigate the natural time course of BP to define an optimal time period to screen patients in the ED with an initial BP elevation. They found the time interval between 60 and 80 min after entry into the ED yielded the best diagnostic accuracy to detect or exclude HTN. HTN may be assumed with specificity greater than 90% in those with a BP greater than 165/105 mmHg and excluded with a sensitivity of greater than 90% when the BP is less than 130/80 mmHg when repeated after 60–80 min of entry into the ED.

HTN has a reputation as being a “silent killer” because of its insidious onset and prolonged disease state before manifestations of clinically significant damage (Bauman, et al., 2007). If the patient’s BP level is persistently increased, the most important diagnosis to rule out is an HE. An HE is distinguished by the presence of TOD. There is no particular BP threshold associated with organ damage; however, it is uncommon with a DBP less than 130 mmHg. The absolute level is not as important as the rate of increase (Marik & Varon, 2007). The major target organs involved in an HE are the brain, heart, great vessels, kidney, and the gravid uterus (Flanigan & Vitberg, 2006). Each of these systems should be reviewed.

A focused history and physical examination can detect signs and symptoms of TOD not initially obvious (Decker, Godwin, Hess, Lenamond, & Jagoda, 2006). History should include use of prescriptions, over-the-counter medications, and recreational drug use. If the patient is known to have HTN, history of present illness should include previous control, medications and dosage, adherence to regimen, and time of last dose of medication. Inquire about a medical history of cardiovascular, cerebrovascular, or renal disease; diabetes, hyperlipidemia, chronic obstructive pulmonary disease, asthma, or gout; or a family history of HTN or premature heart disease (Tintinelli et al., 2004).

HTN is a risk factor for intracerebral hemorrhage, subarachnoid hemorrhage, and acute
ischemic stroke. One of the most prominent symptoms is a headache. Other symptoms include dizziness, light-headedness, vertigo, tinnitus, visual changes, confusion, weakness, or syncope. Perform a complete neurological examination, searching for hemiparesis, alterations in speech or mental status, ataxia, or cranial nerve deficits (Kasper et al., 2005).

Acute aortic dissection is a rare but potentially catastrophic illness. Aortic dissection is associated with HTN in about 90% of cases (Tintinelli et al., 2004). Thoracic and abdominal aortic aneurysms commonly produce no symptoms. If symptoms are present, they may include chest pain, shortness of breath, or back pain. Assess for pulse discrepancy among limbs, a new aortic regurgitation murmur, a pulsatile abdominal mass, or bruit (Kasper et al., 2005).

Ischemic heart disease is the most common form of TOD associated with HTN (NHLBI, 2003). Search for symptoms such as chest pain, shortness of breath, diaphoresis, weakness, dizziness, or nausea. Auscultation may reveal a faint murmur of aortic regurgitation, a fourth (atrial) heart sound, third (ventricular) heart sound, or a summation gallop. Assess the lungs for any signs of pulmonary edema such as rales and palpate the lower extremities for edema (Gilmore, Miller, & Stead, 2005).

BP and renal function are intrinsically related. Worsening renal function in the setting of elevated BP, with elevation of blood urea nitrogen and creatinine levels, proteinuria, or the presence of red blood cells and red blood cell casts in the urine is considered an HE that requires immediate reduction of BP. Inquire specifically about hematuria, anuria, or any changes in voiding pattern.

Fundoscopic examination of the retina is essential. In Grade I retinopathy, there is minimal diffuse or focal narrowing of arterioles. In Grade II, an increased light reflex is seen as “copper and silver wiring.” In Grades III and IV, extensive microvascular changes are seen. Focal ischemia appears as “cotton-wool spots,” and vessel leakage is evident by hard exudates and hemorrhages. Grades I–II are considered relatively mild target-organ effects and are evidence of chronic HTN. Grades III and IV are evidence of accelerated retinopathy, with Grade IV being distinguished by papillitis (inflammatory disk edema) because of infarction and hypoxia of the optic disk, and defines an HE (Tintinelli et al., 2004).

On the basis of this evaluation, the clinician should be able to distinguish between an HE and asymptomatic SEBP and formulate a plan. Positive findings indicate this is not a case of asymptomatic SEBP and would require further diagnostic tests and treatment. A complete blood cell count, electrolytes, blood urea nitrogen, creatinine level, and urinalysis are valuable first-line investigations. For those with chest pain, back pain, or shortness of breath, evaluate a chest x-ray for findings of mediastinal widening or pulmonary edema. A 12-lead electrocardiogram (ECG) should be evaluated for evidence of myocardial ischemia or hypertrophy. If neurological signs are present, a CT head is appropriate (Gilmour et al., 2005).

**DIAGNOSTIC TESTS**

The assessment and management of patients with elevated BP has been extensively studied in the primary care environment. Evidence-based standards for classifying and treating HTN have been established by JNC VII (NHLBI, 2003). They recommend the following routine laboratory test before initiating therapy: ECG, urinalysis, blood glucose, hematocrit, serum potassium, creatinine, estimated glomerular filtration rate, and calcium. However, JNC VII (NHLBI, 2003) clarifies that the HTN management guidelines are intended for use in primary care, and not for patients with acute illness. Imposing these guidelines and rigorous standards in the ED may not be appropriate, as they were not derived for or validated for use in the ED setting (Karras et al., 2004). In a review of the management of ED patients with asymptomatic SEBP, Shayne and Pitts (2003) noted the absence of clinical trials documenting the utility of routinely testing ED patients for silent TOD and questioned the rationale of routinely performing extensive testing of ED patients with SEBP.
Recommendations for laboratory screening in the ED setting are stated vaguely that they “may” be useful (Decker et al., 2006; Marx et al., 2006).

Karras et al. (2007) conducted a study to determine the prevalence of unanticipated, clinically meaningful test abnormalities in ED patients with asymptomatic SEBP. In their study of 109 patients, 57 had at least one abnormal test result. Of those, five had an abnormality possibly related to the elevated BP. No patient had laboratory findings indicative of an HE or that changed the ED management. They concluded that screening is highly unlikely to disclose results leading to changes in ED management.

Diagnostic tests are not absolutely necessary to diagnose asymptomatic SEBP. JNC VII (NHLBI, 2003) does recommend initial routine laboratory tests before initiating therapy. However, this is to screen for TOD and identify compelling indications for the use of an individual drug class. In a rural setting, the patient may be sent with orders to the nearest facility capable of conducting the laboratory tests, chest x-ray, and ECG. Slovis and Reddi (2008) do recommend obtaining a basic metabolic panel before initiating or restarting antihypertensive medications in the ED.

TREATMENT PLAN

A review of literature reveals that there is no evidence-based guideline for the appropriate treatment of the ED patient with an incidental finding of asymptomatic SEBP and recognizes the need for further studies to address this (Decker et al., 2006; Karras et al., 2005, 2007; Shayne & Pitts, 2003; Tilman et al., 2007). Current guidelines are challenged as not being evidence-based, impractical, and cost-inefficient when applied in the ED setting with a high prevalence of patients who have poorly controlled BP (Karras et al., 2006).

There is general consensus that if treatment is initiated, with an aim to gradually lower the BP level over 24 to 48 hr, then no attempt should be made to rapidly lower it. Rapid correction of SEBP below the autoregulatory range of the vascular beds can result in marked reduction in perfusion causing ischemia and infarction (Marik & Varon, 2007). There are reports that demonstrate the occurrence of stroke, hypotension, myocardial infarction, and even death from use of oral agents to acutely lower BP level (Flanigan & Vitberg, 2006). Treatment is probably best initiated by the patient’s primary provider, who can monitor this chronic condition over-time (Decker et al., 2006; Flanigan & Vitberg, 2006; Gilmore et al., 2005). According to JNC VII (NHLBI, 2003), marked elevations of BP without TOD usually do not require hospitalization but should receive immediate combination oral antihypertensive therapy. Decker et al. (2006) point out that the term immediate is used in context of an outpatient setting, and they could not find evidence that acute management of elevated BP improved patient outcomes.

Both the patient and provider often feel that it is beyond the scope of the ED visit to address this chronic condition (Decker, 2006). It has been in this author’s experience that patients are often adamant that they do not want their HTN addressed, they just want their chief complaint treated. However, the ED functions as a healthcare safety net in many communities and is frequently the only opportunity for many individuals to come in contact with a healthcare provider (Tilman et al., 2007).

The American College of Emergency Physicians released a clinical policy, which makes explicit evidence-based recommendations for the treatment of asymptomatic patients with HTN (Decker et al., 2006). The following recommendations were made: if the BP level is persistently elevated above 140/90 mmHg, the patient should be referred for follow-up of possible HTN and BP management; initiating treatment of asymptomatic HTN in the ED is not necessary when patients have follow-up; rapidly lowering BP level in asymptomatic patients is unnecessary and may be harmful in some patients; when ED treatment of asymptomatic HTN is initiated, BP management should attempt to gradually lower BP; and there should be no expectation that BP...
level will be normalized during the initial ED visit. In a recent article, Slovis and Reddi (2008) recommend that oral antihypertensive treatment should be considered for patients with SBP greater than 180 mmHg or DBP greater than 110 mmHg and is indicated if SBP is greater than 200 mmHg or DBP is greater than 120 mmHg.

The following treatment plan is proposed on the basis of a review of current literature (Figure 1). If the initial BP is greater than 140/90 mmHg, repeat it after about 60 min. In addition, consider possible causes of transient elevations such as pain, anxiety, over-the-counter medications, or recreational drugs. If the repeat BP level remains between 140/90 and 179/109 mmHg, instruct the patient to follow up with their primary provider in 24–48 hr. If the BP level is 180/110 mmHg or greater, perform a history and physical focused on identifying an HE. Any positive findings warrant evaluation and treatment of HE. If this is a case of an incidental finding of asymptomatic SEBP, consider whether the patient has a history of HTN. If the patient has run out of medication, then refill it, consult the primary care provider, and recommend follow-up in 24–48 hr. If there is a history of HTN and the patient ran out of medication, then refill it, consult the primary care provider, and recommend follow-up in 24–48 hr. If there is a history of HTN and the patient is not on medication and does not have a primary care provider, then offer treatment on the basis of JNC VII (NHLBI, 2003) guidelines. If the BP level is greater than 200/120 mmHg, treatment is indicated. Although this may not be an appropriate guideline for use in the ED, it is the best evidence-based guideline available until further studies validate and ED guideline. Give the patient the opportunity to decide whether they want treatment initiated in the ED, or they want to follow up with a primary care provider in 24–48 hr. A consent form may be initiated to give the patient the opportunity to accept or decline treatment, and agree to follow up with a provider (Figure 2). This conveys the importance of follow-up to the patient, gives them the opportunity to participate in decision making about their care, and documents that treatment has been offered to them, and that follow-up has been recommended. If the patient chooses to accept treatment, order a complete blood cell count, electrolytes, ECG, chest x-ray film, estimated glomerular filtration rate, and start treatment on the basis of the JNC VII (NHLBI, 2003) guidelines. Further cost/benefit analysis would be necessary before initiating use of a consent form. However, the message is that it is an option to start treatment in the ED, it is important that the patient is made aware of their BP. The risks, and that follow-up is recommended.

**PHARMACOTHERAPY**

If the patient chooses to accept treatment, or his or her BP level is greater than 200/120 mmHg, a medication should be chosen to gradually lower the BP level. Diagnostic results, comorbid conditions, compelling evidence recommendations by JNC VII (see Table 1), and advantages and disadvantages of drug class guide medication choice.

Thiazide diuretics have been virtually unsurpassed in preventing cardiovascular complications of HTN. They enhance the effect of other medications in a multidrug regimen, are useful in achieving BP control, and are less expensive than other antihypertensive medications. These should be used as an initial therapy for most patients, either alone or in combination with another drug class. They are also useful in slowing demineralization in osteoporosis. Use thiazide diuretics cautiously in patients with gout or a history of hyponatremia. The drugs act by inhibiting sodium chloride transport in the distal convoluted tubule. This moderately increases sodium and chloride excretion because approximately 90% of the filtered sodium load is reabsorbed before reaching this site (Brunton, Lazo, & Parker, 2006).

β-Blockers are recommended in patients with ischemic heart disease, acute coronary syndrome, heart failure, and diabetes. They should generally be avoided in patients with
Figure 1. Recommended treatment of hypertensive patient. BP = blood pressure; H & P = history and physical; HE = hypertensive emergency; h/o = history of; HTN = hypertension; PCP = primary care provider; CBC = complete blood count; BMP = basic metabolic panel; ECG = electrocardiogram; CXR = chest x-ray; GFR = glomerular filtration rate; f/u = follow up.
Your blood pressure was _____ today.

It is advised that you start medication to gradually lower your blood pressure. Basic blood tests, a chest x-ray, and an electrocardiogram (an electric tracing of your heart) are needed. These tests will help determine the best medication for you to start. They will also help to find out if the high blood pressure has caused any damage that is less obvious.

I agree to receive evaluation and treatment of hypertension today. I understand that this is a chronic condition that will need long-term control by my primary care provider. I understand some of the risks of uncontrolled hypertension include kidney damage, blindness, heart disease, stroke, and aneurism. I agree to follow up with my primary care provider in 24–48 hr for reevaluation.

X

I do not wish to receive evaluation and treatment of hypertension today. I understand that this is a chronic condition that will need long-term control by my primary care provider. I understand some of the risks of uncontrolled hypertension include kidney damage, blindness, heart disease, stroke, and aneurism.

I agree to follow up with my primary care provider in 24–48 hr for reevaluation.

X

Figure 2. Consent or refusal for hypertension treatment.

asthma, reactive airway disease, or second or third heart block. They compete with adrenergic neurotransmitters for binding at sympathetic receptor sites. Their usefulness in treating HTN include a negative chronotropic effect that decreases heart rate at rest and after exercise, a negative inotropic effect that decreases CO, reduction of sympathetic outflow from the central nervous system, and suppression of renin release from the kidneys (Brunton et al., 2006).

Angiotensin-converting enzyme inhibitors are recommended in patients with acute coronary syndrome, post–myocardial infarction, heart failure, diabetes, chronic kidney disease, and cerebrovascular disease. These should not be used in women who are pregnant or likely to become pregnant or patients with a history of angioedema. They have a high affinity for ACE and compete with angiotensin I to block its conversion to angiotensin II. Peripheral vascular resistance is lowered as formation of this potent vasoconstrictor is blocked (Brunton et al., 2006).

Angiotensin receptor blockers (ARBs) are useful for the treatment of heart failure, diabetes, and chronic kidney disease. They have been shown to reduce the progression to macroalbuminuria in diabetes. They should not be given to women who are pregnant or likely to become pregnant. They block the effects of angiotensin II, thereby promoting vasodilation, increasing renal salt and water excretion, reducing plasma volume, and decreasing cellular hypertrophy (Brinton et al., 2006).

Calcium channel blockers are useful for the treatment of ischemic heart disease and diabetes. Within the cardiac myocyte, calcium

Table 1. Compelling indications for individual drug class (NHLBI, 2003)

<table>
<thead>
<tr>
<th>Compelling indication</th>
<th>Initial therapy options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart failure</td>
<td>Thiazide diuretic</td>
</tr>
<tr>
<td></td>
<td>ACE inhibitor</td>
</tr>
<tr>
<td></td>
<td>ARB</td>
</tr>
<tr>
<td></td>
<td>β-Blocker</td>
</tr>
<tr>
<td></td>
<td>Aldosterone antagonist</td>
</tr>
<tr>
<td>Post–myocardial infarction</td>
<td>ACE inhibitor</td>
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<tr>
<td></td>
<td>β-Blocker</td>
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<tr>
<td></td>
<td>Aldosterone antagonist</td>
</tr>
<tr>
<td>High CVD risk</td>
<td>Thiazide diuretic</td>
</tr>
<tr>
<td></td>
<td>ACE inhibitor</td>
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<td></td>
<td>β-blocker</td>
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<tr>
<td></td>
<td>Calcium channel blocker</td>
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<tr>
<td>Diabetes</td>
<td>Thiazide diuretic</td>
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<td></td>
<td>ACE inhibitor</td>
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<td></td>
<td>β-Blocker</td>
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<tr>
<td></td>
<td>Calcium channel blocker</td>
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<tr>
<td>Chronic kidney disease</td>
<td>ACE inhibitor</td>
</tr>
<tr>
<td>Recurrent stroke prevention</td>
<td>ACE inhibitor</td>
</tr>
</tbody>
</table>

Note. ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; CVD = cardiovascular disease.
Table 2. Lifestyle modification recommendations (NHLBI, 2003)

<table>
<thead>
<tr>
<th>Modification</th>
<th>Recommendation</th>
<th>Average SBP reduction rangea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain normal body weight (body mass index 18.5–24.9 kg/m²)</td>
<td>5–20 mmHg/10 kg</td>
</tr>
<tr>
<td>DASH eating plan</td>
<td>Adopt a diet rich in fruits, vegetables, and lowfat dairy products with reduced content of saturated and total fat</td>
<td>8–14 mmHg</td>
</tr>
<tr>
<td>Dietary sodium reduction</td>
<td>Reduce dietary sodium intake to less than 100 mmol/day (2.4-g sodium or 6-g sodium chloride)</td>
<td>2–8 mmHg</td>
</tr>
<tr>
<td>Aerobic physical activity</td>
<td>Regular aerobic physical activity (e.g., brisk walking) at least 30 min/day, most days of the week</td>
<td>4–9 mmHg</td>
</tr>
<tr>
<td>Moderation of alcohol consumption</td>
<td>Men: limit to 2 drinksb or less per day</td>
<td>2–4 mmHg</td>
</tr>
<tr>
<td></td>
<td>Women and lighter weight persons: limit to 1 drinkb or less per day</td>
<td></td>
</tr>
</tbody>
</table>

Note. SBP = systolic blood pressure; DASH = Dietary Approaches to Stop Hypertension.
*aEffects are dose and time dependent.

b1 drink = 1/2 oz or 15-ml ethanol (e.g., 12 oz beer, 5 oz wine, 1.5 oz 80 proof whiskey).

binds to troponin, removing the inhibitory effect of troponin on the contractile tissue and allowing interaction of actin and myosin leading to contraction. Thus these drugs have a negative inotropic effect. Contraction of vascular smooth muscle is dependent on the free intracellular concentration of calcium. Inhibition of the movement of calcium through calcium channels can decrease the total amount of calcium that reaches intracellular sites. BP level is lowered by relaxing arteriolar smooth muscle and decreasing PVR (Brunton et al., 2006).

CONCLUSION

All patients with a BP level greater than 140/90 mmHg should be referred for follow-up. Patient education should center on lifestyle modifications (Table 2), understanding that this is a chronic and asymptomatic process, setting BP goals, and understanding medication regimens and adverse effects. Administering oral antihypertensive medications to an asymptomatic ED patient to acutely lower BP is not recommended. If the patient has asymptomatic SEBP 180/110–199/119 mmHg, consider starting antihypertensive treatment. If the patient has asymptomatic SEBP greater than 200/120 mmHg, antihypertensive treatment is indicated. Medication should be chosen to gradually lower BP level and based on JNC VII (2003) recommendations.

REFERENCES


