Management of Hypertensive Emergency and Urgency

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Abstract
Severe hypertension is a frequent condition among patients presenting to emergency departments. Historically, this has been referred to as a hypertensive crisis. In addition, these hypertensive crises have been further divided into either hypertensive emergencies or urgencies depending on the presence or absence of target organ damage, respectively. The management differs between these crises in both the rapidity of blood pressure correction and the medications used. Hypertensive emergencies must be treated immediately with intravenous antihypertensive medications. However, hypertensive urgencies may be treated with oral antihypertensive agents to reduce the blood pressure to baseline or normal over a period of 24–48 hr. Appropriate identification, evaluation, and treatment of these conditions are of great importance in the emergency department to prevent progression of organ damage and death. The purpose of this article is to provide an overview of the hypertensive crises and their management. Key words: hypertensive crises, hypertensive emergency, hypertensive urgency, severe hypertension, severely elevated blood pressure, target organ damage

Identification and appropriate treatment of hypertension is an ongoing health care problem. According to a recent National Health and Nutrition Examination Survey, 29% of all U.S. adults have hypertension and an estimated 22% of those are unaware of their condition (Ostchega, Yoon, Hughes, & Louis, 2008). The complications associated with chronic hypertension are well known and are preventable with appropriate primary care. Aneurysm, heart failure, myocardial infarction (MI), stroke, and renal failure are only a few complications of chronic hypertension. Although less emphasized, adverse events associated with acute severe elevations in blood pressure (BP) may also occur. Severe hypertension is a finding in up to 25% of patients presenting to emergency departments (Zampaglione, Pascale, Marchisio, & Cavallo-Perin, 1996). This finding has historically been referred to as a hypertensive crisis. In addition, these hypertensive crises have
been further divided into either hypertensive emergencies or urgencies depending on the presence or absence of target organ damage (TOD), respectively. Appropriate identification, evaluation, and treatment of these conditions are of great importance in the emergency department (ED).

DEFINITIONS

Although frequently discussed in the literature over the past several decades, specific classification and defining criteria for the hypertensive crises have yet to reach consensus. The Joint National Committee on prevention, detection, evaluation, and treatment of high BP provides a definition for hypertensive crisis in their Seventh Report (JNC VII), which consists of a BP greater than 180/120 mmHg (Chobanian et al., 2003). The report further states that a hypertensive crisis in the absence of TOD is categorized as hypertensive urgency and the condition is termed hypertensive emergency in the presence of TOD. Examples of TOD include intracerebral or subarachnoid hemorrhage, cerebral ischemic events, hypertensive encephalopathy, acute MI, acute congestive heart failure (CHF), and eclampsia.

PATHOPHYSIOLOGY

Many conditions may predispose an individual to the development of a hypertensive crisis. The majority of cases arise from essential or primary hypertension (Flanigan & Vitberg, 2006). This could include undiagnosed and, consequently, untreated hypertension, nonadherence to antihypertensive regimens, or abrupt withdrawal from antihypertensive agents (Shea, Misra, Ehrlich, Field, & Francis, 1992). To a lesser degree, it may also arise as a complication of other conditions such as illicit drug use, thyroid storm, trauma, renovascular disease, or adrenal dysfunction as is seen with a pheochromocytoma (Lenders, Eisenhofer, Mannelli, & Pacak, 2005).

The mechanism underlying the abrupt increase in BP is not well understood. It is thought to involve a dysfunction of the autoregulatory system and an increase in systemic vascular resistance (Patel & Mitsnefes, 2005). Autoregulation is primarily achieved via dilation or constriction of vascular beds in response to changes in BP. This is mediated by endothelial release of vasoactive substances as well as activity of the renin-angiotensin-aldosterone system (Ault & Ellrodt, 1985; Vaughan & Delanty, 2000). Although a specific trigger has yet to be elucidated, an abrupt increased release of substances such as angiotensin II and norepinephrine induces vasoconstriction (Ault & Ellrodt, 1985). Endothelial autoregulation through release of nitric oxide and other vasodilators is quickly overcome (Vaughan & Delanty, 2000). This failure of autoregulation leads to endothelial damage from the increase in BP (Vaughan & Delanty, 2000). Endothelial damage then promotes inflammation, activation of the coagulation cascade, and possible thrombosis (Patel & Mitsnefes, 2005; Vaughan & Delanty, 2000). These events ultimately cause ischemic injury to tissues and organs, which may result in death if left untreated. This complex interplay of systems and substances also allows for several targets for pharmacologic intervention.

EVALUATION

An accurate history is an important first step in the evaluation of patients presenting with severe elevations in BP. Important information to obtain in the history includes the previous diagnosis of hypertension and BP control history, preexisting organ disease such as CHF or kidney disease, current medication regimen and adherence, and the use of illicit drugs. Blood pressure readings should be verified through measurement in both arms with an appropriately sized cuff. A significant difference in measurements between arms may suggest an aortic dissection (Macura, Corl, Fishman, & Bluemke, 2003). The aorta provides blood flow to the arms via the subclavian arteries. The aortic origins of these arteries may become occluded by a dissection (Gollege & Eagle, 2008). Depending on the
size and location of the dissection, the occlusion may be unequal between these arteries and affect downstream pressures to varying degrees. The use of a cuff, which is too large or too small, may result in an incorrectly low or high reading, respectively.

Physical examination should focus on the identification of any TOD. This includes an evaluation of the heart and lungs for murmurs, gallops, or pulmonary edema. In addition, all extremities should be assessed for pulses and a focused neurologic and ocular examination should be performed. The neurologic examination should evaluate level of consciousness, alteration in mental status, and signs of hemorrhage such as severe headache. An ocular finding of new exudates, hemorrhages, or papilledema suggests hypertensive encephalopathy (Marik & Varon, 2007; Vaughan & Delanty, 2000). Although a reportedly rare occurrence, aortic dissection should be considered with the presence of severe chest or abdominal pain, back pain, widened pulse pressure, and/or widened mediastinum on chest radiograph (Flanigan & Vitberg, 2006). Pulse pressure is simply the difference between the systolic blood pressure (SBP) and the diastolic blood pressure (DBP). A normal range is approximately 30–50 mmHg. Additional parameters to monitor in the initial presentation include electrolytes, creatinine, blood urea nitrogen, complete blood count, electrocardiogram, chest radiograph, and urine analysis (Vaughan & Delanty, 2000).

**TREATMENT**

Once the history is obtained and the physical examination is complete, the distinction between urgency and emergency delineates the treatment plan.

Hypertensive urgency may be managed with oral antihypertensive agents. The initial assessment of baseline BP is important to determine the goal of therapy. The goal is to reduce the BP to baseline over 24–48 hr (Marik & Varon, 2007). Blood pressure is the product of heart rate and systemic vascular resistance. Pharmacotherapy can be used to lower BP by reducing either heart rate or systemic vascular resistance or both. The vascular beds are capable of maintaining tissue perfusion in the face of changes in systemic BP through vasodilation or vasoconstriction. However, this autoregulatory mechanism only functions with acute changes in BP of less than 20%–25% (Marik & Varon, 2007). Exceeding this limit may lead to ischemia and deleterious effects such as stroke, MI, and death (Flanigan & Vitberg, 2006). This underscores the importance of a slow reduction in BP for patients with hypertensive urgency.

This treatment approach does not seem to correlate with the convention of naming the condition “crisis” or “urgency.” Inappropriately rapid correction of BP has occurred because of this misnomer and has prompted a need for better terminology. Some have proposed a distinction of hypertensive emergency when TOD is present and either severe hypertension or severely elevated BP when TOD is absent (Flanigan & Vitberg, 2006; Hanzik, 2008).

A more aggressive treatment approach is required for hypertensive emergencies to prevent the progression of TOD. The degree of monitoring required and the use of intravenous antihypertensive medications necessitates admission to an intensive care unit or a specialty area such as a cardiac care unit, which can provide safe monitoring and adequate nurse-to-patient ratios. The goal of treatment, in this case, is a reduction of the BP by 15%–25% in the first hour (Chobanian et al., 2003; Marik & Varon, 2007). Careful attention is necessary to avoid exceeding this goal to prevent further ischemic damage as highlighted earlier. Once this goal is met, the patient is stable, and TOD is mitigated, the BP may be reduced further to a SBP of 160 mmHg and DBP of 100–110 mmHg over the ensuing 2–6 hr period (Chobanian et al., 2003). The BP may then be reduced to baseline over the following 24–48 hr. During this final reduction phase, intravenous antihypertensive therapy should be transitioned to oral agents.
Patients with aortic dissections and ischemic stroke have different goals of treatment. In the case of aortic dissection, the BP should be reduced, as tolerated, within 5–10 min to a SBP of less than 100–120 mmHg (Chobanian et al., 2003; Elliott, 2006; Marik & Varon, 2007). The goal of therapy with ischemic stroke is a reduction in BP to allow for the use of thrombolytic therapy where indicated. The BP must be reduced to less than 185/110 mmHg prior to administration of thrombolytic therapy (Genentech, Inc., 2005; Rhoney & Peacock, 2009b).

The ideal intravenous antihypertensive agent is one that has the following properties: rapid onset, easy titration, short duration of action, well tolerated, and little potential for adverse effects. Table 1 provides a list of the commonly used intravenous antihypertensive agents along with information regarding dosing, titration, pharmacokinetic properties, adverse effects, and indications for use.

Adrenergic Antagonists

The proposed pathophysiology of a hypertensive crisis involves activation of adrenergic receptors and a corresponding increase in systemic vascular resistance (Ault & Ellrod, 1985). Adrenergic antagonists, therefore, are a logical choice for treatment of this disorder. The most ideal and commonly used adrenergic antagonists in the treatment of hypertensive emergency are esmolol, labetalol, and phentolamine.

Esmolol is a cardioselective β1-adrenergic receptor antagonist. The rapid onset, less than 1 min, and short duration of action, 10–20 min, are favorable attributes for controlled reduction of BP (Baxter International, Inc., 2007). The cardioselectivity and negative inotropic and chronotropic properties are particularly advantageous in cases of concomitant increased cardiac output, heart rate, and BP (Marik & Varon, 2007; Rhoney & Peacock, 2009a). Contraindications include bradycardia and decompensated heart failure. Dosage must be carefully calculated as errors have resulted in overdose related fatalities (Rhoney & Peacock, 2009a). Esmolol is dosed using microgram units, and errors may occur if conversion to other measures, such as milligrams, is attempted. In addition, some infusion pumps may not be calibrated to accommodate microgram dosing. No dosage adjustment for hepatic or renal dysfunction is necessary for esmolol because it is metabolized via plasma esterases (Baxter International, Inc., 2007). Common adverse effects observed with the use of esmolol include bradycardia, hypotension, and nausea.

Labetalol functions as an α1- and a nonselective β-adrenergic receptor antagonist. The ratio of α- to β-receptor activity is 1:7 with intravenous use (Marik & Varon, 2007; Rhoney & Peacock, 2009a). The peripheral activity afforded by these receptor targets generates a reduction in peripheral vascular resistance and allows cardiac output to be maintained. In addition, labetalol maintains total peripheral, cerebral, coronary, and renal blood flow (Marik & Varon, 2007; Strandgaard & Paulson, 1996). Therefore, labetalol is attractive for treating hypertensive emergencies with underlying cerebral or coronary ischemia. Labetalol has been used safely and successfully for hypertensive emergency in the setting of pregnancy (Chan et al., 2010; Elatrous et al., 2002; Marik & Varon, 2007). Use is contraindicated in reactive airway disease or chronic obstructive pulmonary disease (Sagent Pharmaceuticals, Inc., 2009). Observed adverse effects include dizziness, bradycardia, and bronchospasm.

Phentolamine is a peripheral α1- and α2-adrenergic receptor antagonist. Historically, phentolamine has been used for treatment of hypertensive emergencies with an underlying sympathetic crisis. Examples include amphetamine overdose, clonidine withdrawal, cocaine toxicity, or pheochromocytoma (Elliott, 2006; Rhoney & Peacock, 2009a). However, current clinical use is limited. This is due to the availability of more familiar agents such as nicardipine or fenoldopam (Marik & Varon, 2007). Phentolamine may cause angina or MI in patients...
<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Titration</th>
<th>Onset</th>
<th>Duration</th>
<th>Adverse Effects</th>
<th>Indicated Crises/Comorbidities</th>
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<tbody>
<tr>
<td>Adrenergic antagonists</td>
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<tr>
<td>Esmolol</td>
<td>500 mcg/kg loading dose over 1 min 25–50 mcg/kg/min initial infusion</td>
<td>May increase by 25 mcg/kg/min every 10–20 min (max 300 mcg/kg/min)</td>
<td>&lt;1 min</td>
<td>10–20 min</td>
<td>Nausea, Flushing, Bradycardia, First-degree heart block</td>
<td>Acute aortic dissection, Acute MI, Perioperative hypertension</td>
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<td>Labetalol</td>
<td>20 mg initial bolus, may repeat with 20–80 mg bolus dosing or 2 mg/min initial infusion</td>
<td>May repeat bolus or adjust infusion every 5–10 min</td>
<td>2–5 min</td>
<td>5–15 min</td>
<td>Nausea, Dizziness, Bradycardia, Bronchospasm</td>
<td>Acute intracerebral hemorrhage, Acute ischemic stroke, Acute MI, Hypertensive encephalopathy, Eclampsia or preeclampsia</td>
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<tr>
<td>Phentolamine</td>
<td>1–5 mg bolus dosing</td>
<td>May repeat bolus dosing as needed (max 15 mg)</td>
<td>1–2 min</td>
<td>10–30 min</td>
<td>Nausea, Flushing, Dizziness, Tachycardia</td>
<td>Sympathetic crisis, Catecholamine toxicity</td>
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<tr>
<td>Calcium channel antagonists</td>
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<tr>
<td>Clevidipine</td>
<td>1–2 mg/hr initial infusion</td>
<td>May double dose every 90 sec until approach desired BP, then increase by less than double every 5–10 min (max 32 mg/hr or 72 hr duration)</td>
<td>2–4 min</td>
<td>5–15 min</td>
<td>Nausea, Headache</td>
<td>Acute MI, Perioperative hypertension, Sympathetic crisis, Catecholamine toxicity, May be used in renal failure</td>
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<tr>
<td>Nicardipine</td>
<td>5 mg/hr initial infusion</td>
<td>May increase by 2.5 mg/hr every 5 min (max 15 mg/hr)</td>
<td>5–15 min</td>
<td>4–6 hrs</td>
<td>Nausea, Headache, Dizziness, Edema, Tachycardia</td>
<td>Acute intracerebral hemorrhage, Acute ischemic stroke, Acute MI, Hypertensive encephalopathy, Eclampsia or preeclampsia, Perioperative hypertension, Sympathetic crisis, Catecholamine toxicity, May be used in renal failure</td>
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(continues)
Table 1. Selected Intravenous Antihypertensive Agents (Continued)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Titration</th>
<th>Onset</th>
<th>Duration</th>
<th>Adverse Effects</th>
<th>Indicated Crises/Comorbidities</th>
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<tr>
<td>Nitric oxide donors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Headache, Dizziness, Tachycardia, Tachyphylaxis</td>
<td>Acute CHF, Acute MI, Acute pulmonary edema, Perioperative hypertension</td>
</tr>
<tr>
<td>Nitroglycerin 5 mcg/min initial infusion</td>
<td>May initially increase by 5 mcg/min every 3–5 min until dose is 20 mcg/min, then may increase by 10 mcg/min every 3–5 min (max 200 mcg/min)</td>
<td>2–5 min</td>
<td>5–10 min</td>
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<tr>
<td>Nitroprusside 0.5 mcg/kg/min initial infusion</td>
<td>May increase by 0.5 mcg/kg/min as needed for desired BP (caution &gt;2 mcg/kg/min, max 10 mcg/kg/min)</td>
<td>Seconds</td>
<td>1–2 min</td>
<td>Nausea, Flushing, Headache, Thiocyanate and cyanide toxicity</td>
<td>Acute CHF, Acute pulmonary edema, Perioperative hypertension</td>
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<tr>
<td>Dopamine agonist</td>
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<tr>
<td>Fenoldopam 0.1 mcg/kg/min initial infusion</td>
<td>May increase by 0.05–0.1 mcg/kg/min every 15 min (max 1.6 mcg/kg/min)</td>
<td>&lt;5 min</td>
<td>30 min</td>
<td>Nausea, Headache, Flushing</td>
<td>Sympathetic crisis, Catecholamine toxicity, May be used in renal failure</td>
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with coronary artery disease (Rhoney & Peacock, 2009a). Commonly reported adverse effects include flushing, headache, and tachycardia.

**Calcium Channel Antagonists**

Calcium channel antagonists are generally separated into the two categories of nondihydropyridines and dihydropyridines. The primary difference between these is the site of action. Nondihydropyridines are cardioselective, exerting their effects primarily on the myocardium to decrease conduction and contractility. Dihydropyridines, on the contrary, selectively relax vascular smooth muscle to cause vasodilation and a reduction in systemic BP. This selectivity allows for very little effect on contractility or heart rate (Rhoney & Peacock, 2009a). Nicardipine and clevidipine are representatives of the dihydropyridine group, which are available for intravenous use.

Nicardipine has many useful characteristics for hypertensive emergency. It acts in the cerebral vasculature to cause vasodilation and to reduce vasospasm (Rhoney & Peacock, 2009a; Sabbatini, Strocchi, & Amenta, 1995). This is accomplished with very little effect on intracranial pressure (ICP). Nicardipine additionally dilates coronary vasculature to reduce cardiac ischemia (Marik & Varon, 2007; Rhoney & Peacock, 2009a). These properties are particularly useful in patients with cerebral or coronary ischemia such as in subarachnoid hemorrhage or MI. Nicardipine undergoes hepatic metabolism. Patients with liver impairment may be more sensitive to the BP-lowering effects, and a dosage adjustment or alternative agent should be considered (Razak et al., 1990). It is generally well tolerated with adverse effects including hypotension, headache, dizziness, and tachycardia.

Clevidipine reduces systemic BP by selectively dilating arterioles. Clevidipine differs from nicardipine in several ways. It is not associated with reflex tachycardia and the afterload reduction seen with clevidipine has no effect on cardiac filling pressures (Marik & Varon, 2007; Rhoney & Peacock, 2009a). In addition, clevidipine is metabolized via plasma esterases (Pollack et al., 2009). This allows for dosing independent of renal or hepatic function. Clevidipine is supplied only as a lipid emulsion. Allergies to soy or eggs and defects in lipid metabolism are contraindications for use (The Medicines Company, 2008). This preparation is also devoid of preservatives and requires that vials and administration tubing be changed every 4 hr once punctured (The Medicines Company, 2008). Clevidipine is light-sensitive and must be protected from light during storage. However, it is not necessary to protect clevidipine from light during infusion. Similar to nicardipine, clevidipine is generally well tolerated with the most commonly reported adverse effects of nausea and headache.

Nifedipine is an additional dihydropyridine calcium channel antagonist. This agent is available only in oral formulations. Immediate release oral and sublingual preparations have been used in the past for the management of hypertensive emergencies. Adverse effects such as sudden and severe reductions in BP may cause or potentiate existing organ ischemia and have been associated with fatal outcomes (Grossman, Messerli, Grodzicky, & Kowey, 1996; Marik & Varon, 2007). Oral administration offers very little control over BP reduction, which is a key principle in the management of hypertensive emergencies. For these reasons, the use of immediate release nifedipine, via oral or sublingual route, is strongly discouraged for the treatment of hypertensive emergencies. This recommendation is supported by the JNC VII guidelines, the American Heart Association, and the Cardiorenal Advisory Committee of the Food and Drug Administration (Chobanian et al., 2003; Grossman et al., 1996).

Sustained release preparations of nifedipine should also be avoided as an initial treatment option in hypertensive emergencies because of the inability to titrate or reverse the effects on BP. However, these sustained release preparations may be considered for transition to an oral regimen after BP control is obtained with intravenous antihypertensives.
Nitric Oxide Donors

Nitric oxide–induced vasodilation is an autoregulatory mechanism, which is rapidly overcome in cases of hypertensive crisis. Nitric oxide donors such as sodium nitroprusside or nitroglycerin are used in an attempt to exploit or supplement this mechanism.

Sodium nitroprusside reduces both preload and afterload via arteriolar and venous vasodilation. The potency of this agent with respect to BP reduction necessitates intra-arterial monitoring for optimal dose titration. The ultra-short duration of action, 1–2 min, allows for rapid titration as well as quick offset in the case of overcorrection of BP. Sodium nitroprusside may worsen underlying ischemia by shunting blood flow from areas of decreased vasodilation and ischemia to areas of low resistance (Rhoney & Peacock, 2009a). This has been referred to as “coronary steal” in the myocardium and a “cerebral steal-like effect” in the cerebral vasculature (Immink et al., 2008; Rhoney & Peacock, 2009a). This precludes use in acute MI or in cases of cerebral ischemia. Sodium nitroprusside has been shown to increase ICP and use should be avoided where this is a concern (Rhoney & Peacock, 2009a; Griswold, Reznik, & Mendoza, 1981; Cottrell, Patel, Turndorf, & Ransohoff, 1978). Cerebral vascular resistance is reduced, which allows for an increase in cerebral blood flow and, consequently, ICP (Cottrell et al., 1978). Patients may develop tachyphylaxis to sodium nitroprusside evidenced by increasing dose requirements. Potential cyanide toxicity is frequently cited as a reason for using an alternative agent for BP reduction. Sodium nitroprusside contains cyanide moieties, which are released during metabolism of the parent drug. This cyanide load is effectively neutralized by a combination of liver metabolism to thiocyanate and renal excretion at lower dosages and shorter durations of treatment (Gracia & Shepherd, 2004). Larger doses, greater than 4 mcg/kg/min, or longer duration of treatment at lower doses may overcome these mechanisms and lead to cyanide toxicity (Marik & Varon, 2007). Hallmark signs of toxicity include hypoxia and acidosis. These may manifest as agitation or confusion to coma, an initial tachypnea followed by bradypnea, hemodynamic compromise, and eventual cardiac arrest (Gracia & Shepherd, 2004). Sodium nitroprusside is light-sensitive and must be protected from light in storage and while infusing. In summary, sodium nitroprusside is a safe and effective agent for use in non–ischemic hypertensive emergency in patients with normal hepatic and renal function when used at lower doses for a short duration with appropriate monitoring of BP reduction.

Nitroglycerin differs from sodium nitroprusside in that it acts primarily to venodilate and reduce preload (Rocco & James, 2006). The efficacy and safety of nitroglycerin is largely dependent on volume status (Marik & Varon, 2007; Varon, 2008). Volume-depleted patients may experience reflex tachycardia, hypotension, and worsening ischemia (Marik & Varon, 2007; Varon, 2008). Therefore, an adequate assessment and correction of volume status is paramount. Tachyphylaxis may also develop thereby limiting the use of nitroglycerin (Elkayam et al., 1987). Nitroglycerin is particularly useful in combination with other antihypertensive agents in cases of hypertensive emergency with an underlying acute coronary syndrome or acute pulmonary edema (Rhoney & Peacock, 2009a). Dizziness, headache, tachycardia, and hypotension are common adverse effects of nitroglycerin.

Dopamine Agonist

Fenoldopam is a peripheral dopamine-1 receptor agonist. Stimulation of this receptor generates peripheral artery vasodilation and consequently reduces systemic vascular resistance and BP (Marik & Varon, 2007; Tumlin et al., 2000). Fenoldopam has demonstrated improvement in both BP and renal function for patients with severe hypertension (Elliott et al., 1990; Shusterman, Elliot, & White, 1993). Therefore, it should be considered for hypertensive emergency in patients with underlying renal disease or acute...
renal failure as a manifestation of TOD. As fenoldopam has been shown to increase intracranial pressure and the effects on ICP have not been adequately studied, it should not be used when increases in these are of concern (Rhoney & Peacock, 2009a). Adverse effects observed are generally mild and include flushing, headache, and nausea (Abbott Laboratories, 2004; Tumlin et al., 2000).

CONCLUSION

Severe elevations in BP may range from a benign presentation only requiring oral antihypertensive therapy and a gradual reduction in BP over 24–48 hr to a critical presentation of associated TOD and the need for emergent reduction of BP over 24–48 hr to a critical presentation of TOD. As renal failure is a manifestation of TOD. As fenoldopam has been shown to increase intracranial pressure and the effects on ICP have not been adequately studied, it should not be used when increases in these are of concern (Rhoney & Peacock, 2009a). Adverse effects observed are generally mild and include flushing, headache, and nausea (Abbott Laboratories, 2004; Tumlin et al., 2000).

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