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Hypertensive Emergencies: A Review

How to recognize and manage the various manifestations of uncontrolled hypertension.

ABSTRACT: While acute blood pressure elevations are commonly seen in the ED, not all require emergency treatment. True hypertensive emergencies are characterized by a rapid elevation in blood pressure to a level above 180/120 mmHg and are associated with acute target organ damage, which requires immediate hospitalization for close hemodynamic monitoring and IV pharmacotherapy. Recognizing the clinical signs and symptoms of hypertensive emergency, which may vary widely depending on the target organ involved, is critical. High blood pressure levels that produce no signs or symptoms of target organ damage may be treated without hospitalization through an increase in or reestablishment of previously prescribed oral antihypertensive medication. However, all patients presenting with blood pressure this high should undergo evaluation to confirm or rule out impending target organ damage, which differentiates hypertensive emergency from other hypertensive crises and is vital in facilitating appropriate emergency treatment. Drug therapy for hypertensive emergency is influenced by end-organ involvement, pharmacokinetics, potential adverse drug effects, and patient comorbidities. Frequent nursing intervention and close monitoring are crucial to recuperation. Here, the authors define the spectrum of uncontrolled hypertension; discuss the importance of distinguishing hypertensive emergencies from hypertensive urgencies; and describe the pathophysiology, clinical manifestations, and management of hypertensive emergencies.

Keywords: hypertensive crisis, hypertensive emergency, hypertensive urgency, target organ damage

A *hypertensive emergency* is a sharp rise in blood pressure to a level above 180/120 mmHg that is associated with target organ damage, often involving exigent neurologic, cardiovascular, or renal manifestations.[1] *Hypertensive urgency* is a term used to describe similarly high blood pressure values that neither produce nor worsen target organ damage.[1] The term *hypertensive crisis* is sometimes used to describe the spectrum of severe uncontrolled hypertension, encompassing both hypertensive emergency and hypertensive urgency. The distinction between the latter two is important because the ongoing or imminent target organ damage that characterizes hypertensive emergency warrants immediate hospitalization for close hemodynamic monitoring and IV pharmacotherapy, whereas hypertensive urgency often produces no symptoms of target organ damage and can be managed without hospitalization by simply reinstituting or intensifying previously prescribed oral antihypertensive drug therapy; it does not require immediate blood pressure reduction.[1] Despite these important distinctions, in all hypertensive crises, the goal of treatment is to reduce blood pressure safely without compromising organ perfusion.

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THE PREVALENCE OF HYPERTENSIVE CRISES

An analysis of eight studies conducted in Thailand, France, Italy, and Brazil found that the combined prevalence of hypertensive emergency and hypertensive urgency in EDs was roughly 1.2%, with hypertensive urgency being significantly more common than hypertensive emergency, though prevalence varied across studies.[2] The mean prevalence of hypertensive urgency in EDs was 0.94% and the mean prevalence of hypertensive emergency was 0.3%.

In the United States, although 18% of ED patients have severely elevated blood pressure at or above 180/110 mmHg upon presentation,[3] far fewer have hypertensive emergency, as previously defined, which occurs in conjunction with acute or impending target organ damage. From 2006 through 2013, the estimated number of visits for hypertensive emergency more than doubled, but true hypertensive emergency accounted for only 0.2% of adult ED patients overall and 0.6% of adult ED patients with a diagnosis of hypertension. Mortality rates, however, were relatively high among patients with qualifying hypertensive emergency who presented to U.S. EDs, at 4.8% in 2006 and 4.5% in 2013, underscoring the need for prompt diagnosis and appropriate management of the condition.[4]

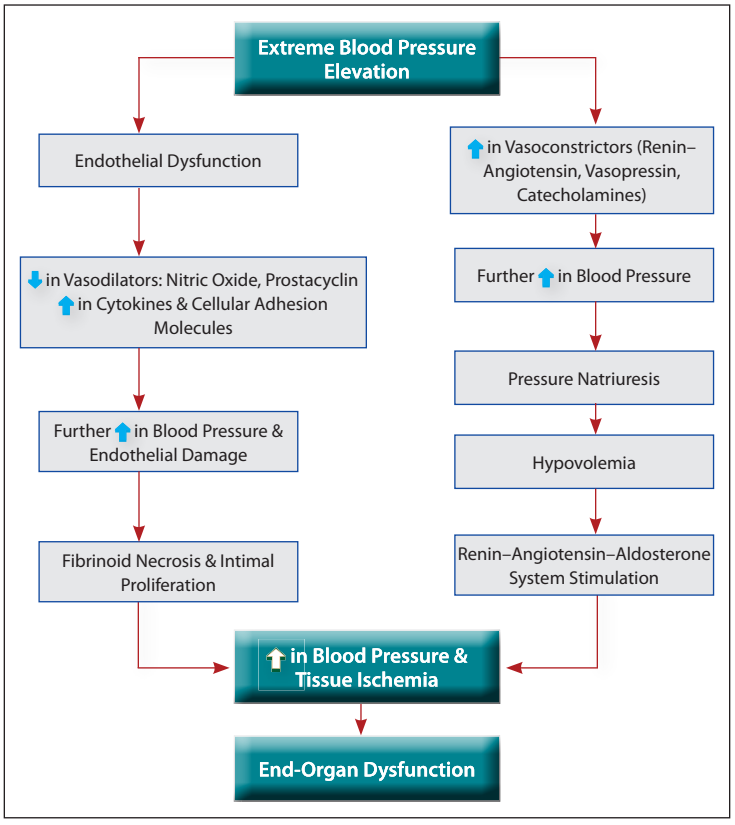
In view of the considerable morbidity associated with hypertensive emergency and the potential for preventing life-threatening deterioration with timely therapy, a thorough understanding of this condition will be of value to nurses in both hospital and ambulatory settings. This article provides an overview of the risk factors for hypertensive emergency; the pathophysiology, clinical manifestations, and management of hypertensive emergency; as well as a discussion of nursing considerations pertinent to the acute and preventive care of patients with this diagnosis.

RISK FACTORS

A 2000 study of 350 patients in a West Birmingham hypertension database found that the majority (55.7%) of patients with hypertensive emergency had no previous diagnosis of hypertension.[5] In patients with a history of chronic hypertension, however, risk factors for developing hypertensive crises include the following[6]:

- female sex
- obesity
- hypertensive heart disease, such as coronary artery disease
- nonadherence to prescribed medication

Figure 1. Pathophysiology of Hypertensive Emergencies[8,12-14]



An increase in renin production resulting from primary hypersecretion or a variety of triggers increases blood pressure. The resulting pressure natriuresis causes hypovolemia, further stimulating renin production. Extremely elevated blood pressure causes endothelial inflammation and dysfunction, which reduces vasodilator production, further exacerbating hypertension. This cycle ultimately terminates in fibrinoid necrosis, intimal hyperplasia, tissue ischemia, and organ damage.

- somatoform disorder
- a greater number of prescribed antihypertensive drugs, which is significantly associated with both nonadherence to prescribed medication and somatoform disorder

In 20% to 40% of patients with blood pressure levels above 200/120 mmHg and advanced retinopathy, secondary causes such as renal parenchymal disease and renal artery stenosis, among others, can be identified.[7] (See *Conditions Associated with Hypertensive Emergency*.[1, 5, 7-10])

Sometimes the initiating event causing a rapid and severe rise in blood pressure can be clearly identified, for instance antihypertensive nonadherence, stress, hyperthyroidism, or the use of drugs such as cocaine,

Conditions Associated with Hypertensive Emergency[1, 5, 7-10]

- Essential hypertension
- Renovascular diseases
  - Renal artery stenosis
  - Polyarteritis nodosa
  - Takayasu arteritis
- Renal parenchymal disease
  - Glomerulonephritis
  - Systemic sclerosis
  - Hemolytic uremic syndrome
  - Systemic lupus erythematosus
- Endocrine dysfunction
  - Pheochromocytoma
  - Cushing's syndrome
  - Primary hyperaldosteronism
  - Renin-secreting tumor
- Coarctation of the aorta
- Drugs
  - Cocaine
  - Amphetamines
  - Phencyclidine
  - Sympathomimetics
  - Erythropoietin
  - Cyclosporine
  - Antiangiogenics
- Antihypertensive medication withdrawal (nonadherence)
- Preeclampsia or eclampsia
- Central nervous system disorders
- Head injury, ischemic stroke, hemorrhagic stroke

amphetamines, phencyclidine, or the prescribed use of monoamine oxidase inhibitors.[6, 11] In most cases, however, the precise mechanisms that trigger an acute elevation in blood pressure aren't immediately clear.[7]

PATHOPHYSIOLOGY

Activation of the renin–angiotensin–aldosterone system seems to play an important role in the development of severe hypertension. Renin secretion may increase in response to a variety of conditions, including reduced renal perfusion pressure, reduced sodium delivery, and β-adrenergic receptor stimulation, which together can trigger a series of reactions that convert angiotensin to angiotensin II, a potent vasoconstrictor that also induces proinflammatory cytokines.[8]

With increased systemic blood pressure, the kidneys may produce a diuretic response called pressure natriuresis, a maladaptive increase in renal sodium excretion, which may exacerbate hypovolemia and cause further activation of the renin–angiotensin system.[7]

Under normal circumstances, sudden elevation of blood pressure triggers a vascular myogenic response, which is the inherent property of vascular smooth muscle in small arteries to constrict, thereby limiting sudden increases in blood flow and protecting the capillary endothelium.[8] However, when blood pressure surpasses a critical point, this response may be overwhelmed, resulting in endothelial damage from mechanical injury and proinflammatory molecular changes in the endothelium.[8]

Reduced production of endothelial vasodilators such as nitric oxide and prostacyclin further elevates blood pressure and exacerbates endothelial damage, a vicious cycle that culminates in enhanced vascular permeability, inhibition of fibrinolysis, platelet aggregation, inflammation, thrombosis, and finally end-organ ischemia.[12, 13] (See Figure 1.[8, 12-14])

CLINICAL MANIFESTATIONS

The initial evaluation of a patient with suspected hypertensive emergency consists of assessing the signs and symptoms of damage to such target organs as the brain; heart; kidneys; large blood vessels, including the aorta; and the microvasculature, including the retina.[15]

A survey developed by an Italian Society of Hypertension research working group and administered to members of several Italian emergency medical societies between December 22, 2017, and March 15, 2018, found that symptoms commonly reported by patients with hypertensive emergency included chest pain (89%), visual disturbances (89.8%), dyspnea (82.7%), and headache (82.1%).[16] Less commonly reported symptoms included dizziness (52%), conjunctival hemorrhages (41.5%), tinnitus (38.2%), and epistaxis (34.4%).[16]

In U.S. EDs, a study of incidence trends for hypertensive emergencies found that heart failure, stroke, and myocardial infarction represented the most common hypertensive emergency diagnoses, trailed by intracranial hemorrhage and ruptured aneurysm or dissection.[4] The occurrence of hypertensive emergency with advanced retinopathy was relatively low. Presentations vary with the specific target organ damage involved. (See Table 1[11, 17-21] for clinical findings associated with specific target organ damage, as well as recommended diagnostic tests.)

MANAGEMENT

Patients presenting with hypertensive emergency should be admitted to an ICU for close monitoring and care.[1] The goals of care can be envisioned as follows:

- Identify the cause and specific manifestation of the hypertensive emergency.
- Initiate rapid life-preserving treatment of severely elevated blood pressure.

Table 1. Target-Organ Damage and Associated Clinical Findings in Hypertensive Emergencies[11, 17-21]

Target Organ Dysfunction	Associated Clinical Findings	Recommended Diagnostic Tests
Acute heart failure with pulmonary edema	Dyspnea, orthopnea, cough, fatigue, basilar lung crackles, third heart sound, jugular venous distension	ECG, chest X-ray, BNP, echocardiogram (if aortic dissection is suspected)
ACS	Crushing chest pain or pressure radiating to the jaw, shoulders, or epigastrium; new-onset murmur (for ACS caused by aortic dissection)	12-lead ECG, cardiac enzymes and cardiac troponin I (for ACS caused by ischemia or MI)
Acute aortic syndrome, including acute aortic dissection	Syncope, neurologic deficit, limb ischemia, chest pain with or without radiating to the back, new onset murmur, aortic insufficiency, asymmetry of pulse and blood pressure between the arms (in aortic dissection)	Chest X-ray, contrast-enhanced CT angiography of chest and abdomen, transesophageal echocardiography
Hypertensive encephalopathy	Headache, visual disturbances, nausea, vomiting, altered mental status, seizures, visual field deficits, cortical blindness	Contrast-enhanced CT or MRI of the brain
Ischemic or hemorrhagic stroke	Altered sensorium, focal neurologic deficits	CT or MRI of the brain
Preeclampsia/eclampsia	Pregnant > 20 weeks, dyspnea, visual disturbances, headache, seizures	Electrolytes, serum creatinine, urinalysis, liver function test

ACS = acute coronary syndrome; BNP = brain natriuretic peptide; CT = computed tomography; ECG = electrocardiogram; MI = myocardial infarction; MRI = magnetic resonance imaging.

- Monitor the patient for medication-related adverse effects as well as for symptoms of renal, coronary, or cerebral ischemia from excessive blood pressure lowering.
- Provide preventive health education related to nutrition, medication adherence, and disease monitoring.

Blood pressure targets may vary based on the specific clinical findings. The goal is not to achieve a particular blood pressure value but to preserve organ perfusion and prevent hypertensive target organ damage.[7] Close interdisciplinary collaboration between nurses and physicians is essential in stabilizing these critically ill patients. Target organ damage and medical comorbidities influence medical decisions concerning target blood pressure, the time frame for achieving blood pressure control, and the choice of pharmacologic agents to be administered. Initially, IV medications are preferred because of their rapid onset, ability to titrate, and relatively short half-life.[7] (See Table 2[22] for IV medications commonly used in hypertensive emergency.)

Intraarterial blood pressure monitoring is used because it is the most accurate means of assessing blood pressure in real time, and accuracy is essential in preventing overly aggressive treatment that could result in complications. However, placement of an arterial line for monitoring should not delay the initiation of therapy. For most adults presenting with hypertensive emergency, systolic blood pressure should be reduced by no more than 25% within the first hour, followed by a more gradual reduction to 160/100 mmHg within the next two to six hours before being cautiously reduced to normal over the subsequent 24 to 48 hours.[1] Some clinical conditions, such as aortic dissection, preeclampsia, or pheochromocytoma may require more rapid blood pressure reduction, while others, such as some cases of ischemic stroke, might warrant less aggressive approaches.

Once controlled, medications can be switched to oral formulations. Some clinical situations necessitate alternative management or special considerations, as discussed below.

ACUTE CONGESTIVE HEART FAILURE

Seen in up to 23% of ED visits for acute hypertension,[4] acute congestive heart failure often occurs in patients with such preexisting cardiac pathologies as coronary artery disease or valve defects, which may predispose to the development of acute systolic or diastolic dysfunction.[11] Even in the absence of previous heart disease or fluid excess, accelerated hypertension increases afterload and left ventricular strain, often culminating in cardiogenic pulmonary edema.[11]

Table 2. Common IV Antihypertensive Medications Used in Hypertensive Emergencies[22]

Medication	Onset of Action, min	Duration of Action	Contraindications	Potential Adverse Effects
Clevidipine (Cleviprex)	2–4	5–15 min	• Severe aortic stenosis • Defective lipid metabolism • Allergy to soy or eggs	• Headache • Nausea and vomiting • Hypotension • Reflex tachycardia
Enalprilat (Vasotec)	15–30	6–≥ 12 hours	• Renal insufficiency • Pregnancy (category D)	• Headache • Hypotension
Esmolol (Brevibloc)	1–2	10–30 min	• Severe sinus bradycardia • 1st or 2nd degree AV heart block • Decompensated heart failure • Cardiogenic shock • Current use of IV calcium channel antagonists • Pulmonary hypertension	• Risk of hypotension • Bradycardia • Cardiac failure • Asthma exacerbation • Increases the effect of hypoglycemic agents in diabetes mellitus and masks hypoglycemic tachycardia
Fenoldopam (Corlopam)	5–10	30–60 min	• No contraindications, but may increase intraocular pressure in patients with glaucoma or intraocular hypertension	• Headache • Flushing • Nausea • Hypotension
Hydralazine	10–20, IV 20–30, IM	1–≥ 4 hours, IV 4–6 hours, IM	• Coronary artery disease • Mitral valvular rheumatic heart disease	• Tachycardia • Headache • Nausea and vomiting • Diarrhea • Palpitations • Angina
Labetalol	5–10	2–4 hours	• Asthma • > 1st degree heart block • Cardiogenic shock • Severe bradycardia	• Nausea • Dizziness
Nicardipine (Cardene)	5–15	1.5–≥ 4 hours	• Aortic stenosis	• Headache • Hypotension • Reflex tachycardia
Nitroglycerin	2–5	5–10 min	• Intracranial hypertension • PDE-5 inhibitor use • Severe anemia • Circulatory failure and shock	• Headache • Dizziness • Paresthesia • Methemoglobinemia
Nitroprusside (Nitropress, Nipride RTU)	0.5–1	1–10 min	• Inadequate cerebral perfusion • Acute coronary syndrome • Recent PDE-5 inhibitor use	• Cyanide toxicity • Hypotension
Phentolamine	1–2	10–30 min	• Hypersensitivity to any ingredients	• Arrhythmias • Cerebrovascular spasm • Myocardial infarction • Injection site pain

AV = atrioventricular; PDE-5 = phosphodiesterase-5.

Managing cardiogenic pulmonary edema involves gradually reducing blood pressure levels as low as tolerated without producing signs of hypotension or hypoperfusion.[11] Nitroglycerin and nitroprusside are the preferred IV agents owing to their favorable effects on both preload and afterload reduction.[7] Avoid administering medications that increase cardiac work, such as hydralazine, or reduce cardiac contractility, such as β-blockers.[11] Although diuretics are not typically used to treat hypertensive emergencies, in the case of acute pulmonary edema, concomitant administration of loop diuretics can further lower blood pressure by reducing volume overload.[11] Noninvasive positive-pressure ventilation can also help manage pulmonary edema by reducing venous return.[7]

ACUTE CORONARY SYNDROME

During hypertensive emergency, endothelial injury activates the coagulation cascade in the coronary arteries, triggering platelet aggregation, which, in conjunction with the release of vasoactive mediators, can compromise myocardial blood flow.[11] Immediate recognition and proper diagnosis of myocardial infarction depend on a careful history, an electrocardiogram (ECG), and laboratory studies including measuring cardiac enzyme levels. A retrospective data analysis of 236 patients who had presented with hypertensive crisis found that patients with elevated cardiac troponin I levels had nearly three times the risk of major adverse cardiovascular or cerebrovascular events at two years' follow-up than patients whose cardiac troponin I levels were normal.[23]

Since blood pressure fluctuation is common in the early phase of acute coronary syndrome (ACS) often due to pain or anxiety, these factors should be addressed before targeting blood pressure with antihypertensive therapy.[24] While blood pressure targets for ACS patients have not been established, the American Heart Association recommends a slow blood pressure reduction that maintains diastolic pressure at or above 60 mmHg so as not to compromise coronary perfusion.[24]

To prevent reflex tachycardia and a subsequent increase in myocardial oxygen demand, preferred agents include[11,24]

- nitroglycerin, a vasodilator that reduces cardiac preload.
- labetalol, which reduces systemic vascular resistance while maintaining cardiac output and without producing reflex tachycardia.
- esmolol, for patients who do not have contraindications to β-blockers.

ACUTE AORTIC DISSECTION

The annual incidence of all acute aortic syndromes, including aortic dissection, is relatively low in the general population, ranging from four to six per 100,000 person-years, though it rises to 30 or more per 100,000 person-years in people over age 65.[25] The mortality rate is high for both type A dissections, which involve the ascending aorta, and type B dissections, which involve only the descending aorta.

Surgery is the recommended treatment for type A aortic dissection, whereas type B is generally treated medically in the absence of other life-threatening complications. According to data from the International Registry of Acute Aortic Dissection study, which were reported in 2000, even with surgery, 26% of patients with type A dissection do not survive, and if treated nonsurgically because of age or comorbidities, this figure rises to 58%.[25,26] Since hypertension is identified as a risk factor in up to 80% of aortic dissections,[25] it should be on the clinician's radar for patients presenting to the ED with acute chest pain and elevated blood pressure.

Medical management involves effective pain control and rapid lowering of systolic blood pressure to 100 to 120 mmHg with a simultaneous reduction of heart rate to 60 beats per minute, referred to as "anti-impulse therapy."[20,27] The purpose of anti-impulse therapy is to reduce left ventricular force and aortic wall stress, thereby limiting tearing and preventing rupture. This should not, however, compromise cerebral perfusion.

Labetalol is typically the drug of choice for rapid blood pressure reduction because of its α- and β-adrenergic blocking effects.[7,22]

Alternative treatments include esmolol, which has a shorter half-life and may be used in patients with relative contraindications to β-blockers.[11] Nitroprusside can be added if targets are not reached with a β-blocker alone.[7]

For patients with significant contraindications to β-blockade, calcium channel blockers may be used.[28]

HYPERTENSIVE ENCEPHALOPATHY

Normally, with a rise in systemic blood pressure, cerebral arterioles constrict in order to maintain a constant rate of blood flow to the brain, a phenomenon called cerebral autoregulation.[19] But when blood pressure rises very rapidly, the autoregulatory response may be insufficient to prevent cerebral hyperperfusion; a weakening of the blood–brain barrier; and fluid extravasation into brain tissue, particularly tissue in the posterior regions.[19] In the absence of ischemia or hemorrhage, clinical and radiologic findings gradually resolve with the control of blood pressure. The syndrome is often referred to as posterior reversible encephalopathy syndrome.[19]

Controlled blood pressure reduction is the mainstay of treatment for hypertensive encephalopathy.

A 20% to 25% reduction in initial mean arterial blood pressure is recommended in recent European Society of Cardiology guidelines.[7] Reducing mean arterial blood pressure further could increase risk of cerebral hypoperfusion. After an initial gradual blood pressure reduction over at least 24 hours, further measures may be pursued; however, there is no clear guidance as to the optimal time over which to reduce blood pressure or to escalate treatment to normalize blood pressure. Medications that can be infused continuously, such as nicardipine or labetalol, are preferred to avoid blood pressure fluctuations, which can disrupt cerebral blood flow in the setting of impaired autoregulation.[9] Nitroprusside should be avoided in hypertensive encephalopathy because it has vasodilatory effects and the potential to worsen cerebral edema and increase intracranial pressure.[29]

dipine are recommended as initial agents in recent stroke guidelines.[21]

Sodium nitroprusside may be considered for patients with AIS if other agents fail to control blood pressure or if diastolic blood pressure is greater than 140 mmHg.[21]

Blood pressure management following recanalization in patients with AIS remains an area of study. To promote cerebral perfusion, common practice is to allow blood pressure as high as 180/105 mmHg for the first 24 to 48 hours following IV thrombolysis with tissue plasminogen activator.[34] However, higher blood pressure following thrombectomy is associated with worse outcomes, hemorrhage, and reperfusion injury.[34]

Current literature suggests better outcomes with systolic blood pressure goals of less than 160 mmHg, or even less than 140 mmHg, following successful mechanical thrombectomy.[35] Precise goals may be contingent on the degree of successful recanalization, so all members of the health care team should know the precise goals of the intervention.

In all hypertensive crises, the goal of treatment is to reduce blood pressure safely without compromising organ perfusion.

ACUTE ISCHEMIC STROKE

Sudden onset of a focal neurologic deficit, such as facial or limb weakness, ataxia, aphasia, dysarthria, or visual field loss, often indicate acute ischemic stroke (AIS) or transient ischemic attack.[30] According to the 2003 International Society of Hypertension statement on management of blood pressure in acute stroke, blood pressure is elevated in about 75% of patients soon after ischemic stroke and in more than 80% of patients following intracerebral hemorrhage.[31,32] Managing hypertension in AIS can be challenging because elevated blood pressure may be a compensatory physiological response to inadequate cerebral perfusion pressure (CPP).[33] CPP is calculated as the difference between mean arterial pressure and intracranial pressure. Reducing mean arterial pressure, and consequently CPP, could lead to additional infarction by further reducing perfusion to ischemic tissues.[21] Higher blood pressure targets are thus permissible in this setting.

IV thrombolysis and mechanical thrombectomy. Patients with suspected ischemic stroke should be rapidly evaluated for IV thrombolysis and thrombectomy. Patients eligible for thrombolysis should have blood pressure quickly lowered to less than 185/110 mmHg and maintained at pressures below 180/105 mmHg for at least 24 hours following treatment in order to reduce risk of intracranial hemorrhage.[21] IV labetalol, clevidipine, and nicar-

For patients with transient ischemic attack and those ineligible for thrombolysis or thrombectomy, initial blood pressure as high as 220/120 mmHg can be considered in order to maintain perfusion to tissue with potentially reversible ischemia, followed by a gradual blood pressure reduction over the next 24 to 48 hours.[30] In some patients, blood pressure reduction may exacerbate ischemic symptoms, in which case the time frame for reduction should be extended.

INTRACRANIAL HEMORRHAGE

Patients with intracranial hemorrhage (ICH), like those with AIS, often present with extremely elevated blood pressure accompanied by focal neurologic abnormalities of sudden onset, including headache and reduced level of consciousness, though the latter occurs more often in ICH than in AIS and is frequently progressive in nature. Noncontrast computed tomography (CT) is both sensitive and specific for acute parenchymal hemorrhage, which in hypertensive ICH is frequently located in the basal ganglia, thalamus, pons, and cerebellum. However, in patients who have underlying vascular malformation, have cerebral amyloid angiopathy, or use

anticoagulants, ICH may also occur in the cerebral hemispheres.[18, 36] Since blood pressure and coagulopathy management for ICH and AIS are quite different, ICH-specific measures should only be started after the diagnosis is confirmed.

Results from recent large randomized controlled trials have been unable to clarify broadly applicable blood pressure targets for patients with spontaneous ICH.[37, 38]

INTERACT-2 (the second Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial), which enrolled patients within six hours of symptom onset, showed that reducing systolic blood pressure to less than 140 mmHg was as safe as reducing it to less than 180 mmHg.[37] While aggressive blood pressure lowering did not reduce the primary outcome of death or severe disability, functional outcomes were slightly but statistically significantly better in the aggressive treatment group.

ATACH-2 (the Antihypertensive Treatment of Acute Cerebral Hemorrhage II trial), by contrast, compared the same target systolic blood pressure levels (less than 140 mmHg versus the standard of less than 180 mmHg) in treating patients with ICH, but with the aim of achieving the targets more rapidly (within 4.5 hours of symptom onset). Results of the trial showed no difference in mortality or functional outcome between the two treatment groups, though the intensive treatment group had a significantly higher rate of renal dysfunction at seven days than the standard treatment group.[38] The risk–benefit of aggressive systolic blood pressure reduction to less than 140 mmHg in ICH thus remains unresolved.

Systolic blood pressure below 130 mmHg. A secondary analysis of INTERACT-2 found that achieved systolic blood pressure below 130 mmHg (the level at which the INTERACT-2 protocol called for cessation of IV antihypertensive treatment) was associated with increased risk of physical dysfunction compared with systolic blood pressure between 130 mmHg and 140 mmHg.[39] It is thus reasonable to conclude that overly intensive blood pressure reduction in the first few hours after symptom onset is inadvisable.

Patients presenting with sustained systolic blood pressure above 220 mmHg, those with severe ICH, and those requiring surgical decompression are poorly represented in the data, so optimal blood pressure targets have not been established for these groups. General principles of blood pressure lowering using IV infusions while avoiding sudden drops in blood pressure are still applicable.[1]

SYMPATHETIC CRISES

Marked hyperadrenergic states can result from ingestion of sympathomimetic medications (cocaine, methamphetamine, phencyclidine, lysergic acid diethylamide, and monoamine oxidase inhibitors with ingestion of food-containing tyramine), withdrawal of short-acting antihypertensive agents (clonidine and β-blockers), and endocrine disorders such as pheochromocytoma. Altered mental status, agitation, chest pain, palpitations, and seizures are the usual presenting symptoms.

The physical examination for hypertensive emergency should focus on identifying signs of target organ damage.

Hypertension from cocaine or amphetamine intoxication can be treated effectively with benzodiazepines to reduce the stimulant effects and with nitrates or calcium channel blockers to control blood pressure.[11, 40] With the use of β-blockers in sympathomimetic drug overdose, there has historically been a concern for exacerbating coronary ischemia; this view however has come into question recently since supportive data seem to be weak.[40]

ENDOCRINE HYPERTENSIVE EMERGENCIES

Pheochromocytomas are neuroendocrine catecholamine-producing tumors originating from chromaffin cells of the adrenal medulla or extraadrenal paraganglia, which produce varying amounts of epinephrine, norepinephrine, or dopamine.[41]

The clinical presentation is highly variable and often appears as a mimic for stress-related disorders. Symptoms of catecholamine excess can include headache, diaphoresis, palpitations, and panic attacks in addition to severe hypertension.[42] Both plasma and urine tests for free normetanephrine, a major metabolite of norepinephrine, and metanephrine, a major metabolite of epinephrine, are commonly used for screening of this condition.[41] Both plasma and urine tests for free metabolites

have negative predictive values greater than 99% and specificities of 94%, though false positives may occur in critically ill patients.[41]

Imaging studies like contrast-enhanced CT or magnetic resonance imaging instead of or in addition to biochemical testing can improve sensitivity and specificity of diagnosis.[41]

Definitive therapy for pheochromocytoma is the surgical removal of the tumor, but presurgical administration of α-adrenergic receptor blockers is considered a first-choice treatment to prevent hypertensive crisis in the perioperative period from a massive release of catecholamines during tumor removal.[41] Frequently prescribed α-blockers include phenoxybenzamine and doxazosin.[41] Labetalol should not be used without prior adequate α-blockade in patients with hyperadrenergic states, such as pheochromocytoma, because α-adrenergic activity can increase blood pressure if β-blockade is not complete.[22]

Eclampsia is the occurrence of generalized seizures not attributable to other causes in a patient with preeclampsia.

In pregnant women with preexisting chronic hypertension, acute exacerbation of hypertension can occur due to inadequate medical treatment or from superimposed preeclampsia.[17] Regardless of nomenclature, acute-onset severe systolic hypertension (160 mmHg or above), severe diastolic hypertension (110 mmHg or above), or both, occurring during pregnancy is a hypertensive emergency.[17] It's inadvisable to adhere too strictly to blood pressure thresholds, because target organ damage can occur with milder hypertension in preeclampsia, and the condition of patients with preeclampsia can deteriorate rapidly without warning.[17]

Management involves lowering blood pressure to a systolic range of 140 to 150 mmHg and a diastolic range of 90 to 100 mmHg.[45] Preferred agents include IV labetalol, IV hydralazine, or immediate-release oral nifedipine.[45] IV magnesium sulfate should be administered concurrently to reduce the risk of seizures.[45] Additional considerations are monitoring of fetal heart rate for bradycardia with β-blocker use and evaluation for delivery.

Blood pressure measurement in both arms, if found to be significantly different, can raise suspicion of aortic dissection.

Hypertension in primary hyperaldosteronism can also result in vascular target organ damage to the heart, kidney, and arterial walls.[43] This results not only from high blood pressure but also from aldosterone-induced endothelial dysfunction, microvascular inflammation, and fibrosis.

Cushing syndrome during pregnancy is both rare and associated with high maternal and fetal mortality rates.[44] Its symptoms, however, can mimic those of a normal pregnancy and its occurrence during pregnancy with uncontrolled hypertension can be misdiagnosed as preeclampsia.[44]

OBSTETRIC HYPERTENSIVE EMERGENCIES

Preeclampsia refers to the onset of hypertension (sustained systolic blood pressure of 140 mmHg or above or diastolic blood pressure of 90 mmHg or above) after 20 weeks' gestation in a previously normotensive woman, in conjunction with one or more of the following[17]:

- proteinuria
- thrombocytopenia
- renal insufficiency
- impaired liver function
- pulmonary edema
- neurologic symptoms, such as intractable headache, visual scotomata, convulsions, altered mental status, blindness, stroke, or clonus

THROMBOTIC MICROANGIOPATHY AND ACUTE RENAL FAILURE

Thrombotic microangiopathy (TMA) can result from hypertensive endothelial injury, involving platelet aggregation, coagulation activation, and inhibition of fibrinolysis.[7] In hypertensive emergency, very high blood pressure can cause progressive vascular injury, acute renal failure, and TMA.[7] It's important to distinguish hypertension-induced TMA and renal failure from thrombotic thrombocytopenic purpura (TTP) and acute renal failure from hemolytic uremic syndrome (HUS) because, while antihypertensive treatment will usually improve TMA and associated renal failure in hypertensive emergency, other treatments may be required for TTP and HUS.

ACUTE PERIOPERATIVE HYPERTENSION

Perioperative hypertensive emergencies can result from adrenergic stimulation from the surgical event, changes in intravascular volume, postoperative

pain, or anxiety. If untreated, perioperative hypertension can result in new target organ damage, increased risk of bleeding, and myocardial infarction.[46] To identify possible risk factors for hypertensive emergencies in preoperative patients with hypertension, a careful preoperative assessment is essential. When blood pressure is above 180/110 mmHg, urgent treatment or postponement of surgery should be considered on a case-by-case basis to avoid the risk of hypertensive emergencies in the perioperative or postoperative period.[46] For each patient, comorbidities and specific blood pressure goals should be discussed with the surgeon, considering the patient's type of hypertension.

ACS, such as raised jugular venous pressure, crackles, third heart sound, or gallop.

The neurologic examination should assess the level of consciousness, signs of meningeal irritation like photophobia and neck stiffness, and note the presence of visual field defects, localized weakness or numbness, uncoordinated limb movements, dysarthria, and language deficits.[48]

Initial diagnostic tests may include renal function, electrolytes, complete blood count (including peripheral smear for signs of hemolysis), ECG, chest X-ray, and urine analysis, depending on patient presentation. Of note, during hypertensive emergency, abnormalities seen on ECG may result not only

When transitioning patients from IV infusions to oral medications, allow sufficient overlap to reduce the risk of rebound hypertension.

NURSING CONSIDERATIONS IN HYPERTENSIVE EMERGENCIES

Initial evaluation. A thorough history should incorporate details of the duration and severity of preexisting hypertension and the presence of previous end-organ damage, especially renal, cardiac, and cerebrovascular disease. It should also include details of antihypertensive medications; level of blood pressure control; intake of over-the-counter drugs, such as sympathomimetic agents; and any use of illicit drugs. Document all information about ongoing or impending end-organ compromise, including but not limited to such symptoms as chest pain (associated with ACS and acute aortic dissection); back pain (as can occur with aortic dissection); dyspnea (a potential sign of pulmonary edema or congestive heart failure); and neurologic symptoms, such as seizures, altered consciousness, or hypertensive encephalopathy.

The physical examination should focus on identifying signs of target organ damage. If possible, blood pressure should be measured when the patient is in both supine and standing positions, so as to assess for volume depletion due to pressure natriuresis, which can sustain a cycle of renal ischemia, vasoconstriction, and progressively increasing hypertension.[47] Blood pressure measurement in both arms, if found to be significantly different, can raise suspicion of aortic dissection. A fundoscopic assessment can reveal such signs of severe hypertension as retinal hemorrhages, exudates, or papilledema.

The cardiovascular assessment should focus on evaluating the patient for signs of heart failure or

from ACS but also from acute neurologic conditions associated with a reversible myocardial dysfunction termed "neurogenic stunned myocardium."[49]

Therapeutic considerations and safety monitoring. Adequate IV access should be established for medication administration and volume infusion. Preparation for intraarterial blood pressure monitoring may be necessary for medication adjustment. Nurses should document any precipitous drop in blood pressure as this may aggravate cerebral, myocardial, or renal ischemia. If there is evidence of volume depletion, IV saline may be administered to restore perfusion in advance of antihypertensive treatment.[47] In the case of pregnancy-related hypertensive emergency, fetal monitoring may be necessary.

When transitioning patients from IV infusions to oral medications, allow sufficient overlap to reduce the risk of rebound hypertension. The specific time frame required will depend on the pharmacodynamics of the drug being titrated downward and the drug being initiated. Continuous ECG monitoring is necessary to detect arrhythmias and cardiac ischemia. Document any changes in the patient's level of consciousness, mood, or orientation; patient reports of headache or visual changes; any vomiting; and all intake and output measurements, which can signal both cardiac and renal complications. Unrelieved pain should be promptly addressed, as it may not only exacerbate hypertension but also indicate target organ damage. Similarly, treating anxiety in patients with acute hypertension has been shown to significantly reduce blood pressure in those without target organ damage,

though the safety of such therapy has not been studied in patients with hypertensive emergency.[50]

PATIENT EDUCATION AND DISCHARGE PLANNING

Patient education. When preparing patients for discharge, nurses should emphasize preventive measures in patient teaching. The Dietary Approaches to Stop Hypertension (DASH) trial has shown that a low-sodium diet combined with the "DASH" diet, which is rich in fruits, vegetables, legumes, and low-fat dairy products and low in sweets, saturated fat, and total fat, can substantially aid in lowering blood pressure.[51] This approach is recommended in the 2020 International Society of Hypertension Global Hypertension Practice Guidelines.[52] Dietary advice should take into account comorbid conditions that may require additional modifications, such as diabetes or kidney disease. Recommended lifestyle modifications include smoking cessation, regular physical activity, and weight reduction when appropriate.[52]

Discharge planning should include information concerning the impact of long-term hypertension on various organ systems, as well as the importance of blood pressure control, adherence to the prescribed medication regimen, and regular follow-up with an established provider. For patients who lack requisite resources, consider social work consultation to find financial support, low-cost drug programs, and generic medication substitutions.[48] Although data on long-term functional outcomes following discharge of patients after hypertensive emergency are sparse, specific rehabilitative and equipment needs may be determined by the sequelae of target organ damage in clinical situations, such as stroke, heart failure, and renal failure. Coordination of care between the nurse, hospitalist, specialist services, therapy services, and social work is essential for safe hospital discharge and appropriate follow-up.[53] ▼

For 19 additional nursing continuing professional development activities related to hypertension, go to www.nursingcenter.com.

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