BMJ Best Practice

Hypertensive emergencies

Straight to the point of care

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Table of Contents

Summary

Hypertensive emergency is severely elevated blood pressure (BP) associated with new or progressive target organ dysfunction.

If the clinical suspicion is high, treatment should be initiated immediately without waiting for further tests.

BP must be lowered over minutes to hours with parenteral medications in an intensive care setting.

The initial goal of therapy is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour). If the patient remains stable, further reduce the BP to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2 to 6 hours. Normal BP may be targeted over the next 24 to 48 hours.

Excessive falls in pressure may precipitate renal, cerebral, or coronary ischemia and so should be avoided.

Exceptions to this general rule are patients with aortic dissection, pheochromocytoma crisis, and severe preeclampsia or eclampsia. Selected patients with spontaneous intracerebral hemorrhage may also require acute blood pressure lowering; careful titration in these patients is required to ensure continuous smooth and sustained control of BP.

With appropriate treatment, prognosis is good.

Definition

Hypertensive emergency is defined as severely elevated blood pressure (BP) associated with new or progressive target organ dysfunction. Although the absolute value of the BP is not as important as the presence of end-organ damage, the systolic BP is usually >180 mmHg and/or the diastolic BP is >120 mmHg.[1]

Epidemiology

The worldwide prevalence of hypertension is around 31%, exceeding 1.3 billion people.[2] [3] [4] Of these, 1% to 2% will suffer a hypertensive crisis in their lifetime.[5] [6] Rates of hypertensive emergencies have increased over the past 20 years; however, mortality rates have decreased and range from 0.2% to 11.0%.[7] [8] [9]

Men may be more likely than women to suffer a hypertensive emergency. Hypertensive emergency is more common in older patients and in black people.[10] [11] [12] [13] Preeclampsia complicates 2% to 8% of pregnancies globally.[14] Preeclampsia is more prevalent among African American women than among white women.[15] [16] [17] Differences in prevalence may be, in part, due to African American women being disproportionately affected by risk factors for preeclampsia.[16] African American women also have case fatality rates related to preeclampsia three times higher than rates among white women.[16] Inequalities in access to adequate prenatal care may contribute to poor outcomes associated with preeclampsia in African American women.[16] However, UK data concerning ethnic differences in hypertension prevalence and complications are inconsistent.[18] [19] [20]

Lack of insurance or a primary care doctor and nonadherence to treatment all predispose toward development of hypertensive emergency.[21] [22] As populations age globally, the prevalence of hypertension and therefore hypertensive emergency is expected to increase.[3]

Etiology

Essential hypertension that is either undiagnosed or inadequately treated is a common cause of hypertensive emergency.[12] [21] [22] [23] Another common cause is secondary and resistant hypertension.

System disorders that can lead to a presentation of hypertensive emergency include:

- Renal disease (underlying chronic disease, renal artery stenosis, acute glomerulonephritis, collagen-vascular diseases, kidney transplantation)[24] [25] [26] [27] [28] [29] [30]
- Neurologic (head trauma, spinal cord injury, autonomic dysfunction)[31]
- Respiratory (obstructive sleep apnea)[32] [33] [34]
- Immunologic (scleroderma, vasculitis)[31]
- Endocrine (primary aldosteronism, pheochromocytoma, thyroid disorder, Cushing syndrome, acromegaly, hyperparathyroidism, carcinoid tumor, congenital adrenal hyperplasia, or renin-secreting tumor).[31] [35] [36][37]

Pregnancy-related preeclampsia, HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome, and eclampsia are also important causes of hypertensive emergency in women.[31]

Lifestyle choices should also be considered when trying to determine the potential cause of a hypertensive emergency as excessive dietary salt intake, obesity, and/or alcohol consumption can all contribute to hypertension.[38] A thorough medication history must also be obtained as hypertension can be induced or exacerbated by certain medications, including nonsteroidal anti-inflammatory drugs, oral contraceptives, sympathomimetics, illicit drugs, glucocorticoids, mineralocorticoids, calcineurin inhibitors, erythropoietin, herbal supplements, vascular endothelial growth factor inhibitors, and inadvertent drug or food interactions with monoamine oxidase inhibitors.[7][31] [39]

Pathophysiology

The factors that lead to the development of hypertensive emergency are poorly understood. A rise in systemic vascular resistance, resulting from a combination of humoral vasoconstrictor increase and autoregulatory failure, initiates the cycle. The subsequent increase in blood pressure generates mechanical stress and endothelial injury leading to increased permeability, activation of the coagulation cascade and platelets, deposition of fibrin, and inflammatory cytokine induction. These processes result in ischemia and the release of additional vasoactive mediators generating ongoing injury. Volume depletion caused by pressure natriuresis and activation of the renin-angiotensin system often lead to further vasoconstriction. Systemic vasoconstriction leads to decreased blood flow to vital organs and the subsequent end-organ injury that is the hallmark of hypertensive emergency. End-organ injury primarily affects the neurologic, cardiac, and renal systems.[39] [40] [41] [42] [43] [44] [45]

Case history

Case history #1

A 50-year-old black man with a history of untreated hypertension presents to the emergency room with substernal chest pressure. His symptoms started the previous day. The pain was initially intermittent in nature but has become constant and radiates to his jaw and left shoulder. He also complains of dizziness and some shortness of breath. Apart from a history of hypertension diagnosed 1 year ago, the patient denies any past medical history. He is not taking any antihypertensive medications. The patient denies smoking, or alcohol or drug use. Family history is unremarkable. His blood pressure (BP) is 230/130 mmHg with otherwise normal vital signs and no other significant findings. ECG shows diffuse T-wave inversion and ST depression in lateral leads. Laboratory testing is significant for elevated troponin, signaling myocardial infarction.

Case history #2

A 35-year-old woman presents at 37 weeks' gestation with severe headache and acute abdominal pain. She had a routine prenatal visit 4 days previously with no signs or symptoms reported or observed. On exam, her BP is 165/110 mmHg and urinalysis reveals proteinuria (3+). She is admitted to hospital and is started on labetalol.

Other presentations

In addition to acute coronary syndrome or severe preeclampsia/eclampsia, hypertensive emergency can present as new or progressive damage to the following target organs: brain (e.g., stroke, seizure, transient ischemic attack, cerebral infarction, intracerebral or subarachnoid bleed, hypertensive encephalopathy, posterior reversible leukoencephalopathy); heart/blood vessels (acute pulmonary edema, acute congestive heart failure, acute aortic dissection, microangiopathic hemolytic anemia); kidney (acute kidney injury); retina (papilledema, hemorrhages, retinal edema).

Approach

The key to diagnosis of hypertensive emergency is a rapid but thorough evaluation. The main areas of focus should be the neurologic, cardiovascular, and renal systems. Emergency treatment should be initiated while conducting a full diagnostic appraisal.

History

Any prior history of hypertension and previous treatment (including treatment adherence) should be identified.[7] [18] Prior or existing history of neurologic, cardiac, and renal impairment should also be determined.

Clinical features that may identify specific organ compromise include:[18] [24] [40][54]

- Neurologic compromise: for example, blurry vision, dizziness, headache, seizures, change in mental status from baseline, dysphagia, loss of sensation, paresthesia, or loss of movement
- Cardiac compromise: for example, chest pain, shortness of breath, diaphoresis, orthopnea, paroxysmal nocturnal dyspnea, palpitations, or edema
- Renal compromise: for example, decrease in urine output.

When appropriate, use of street drugs, particularly sympathomimetics (cocaine, amphetamines, phenylpropanolamine, phencyclidine, ecstasy, LSD) should be investigated.[7] [18]

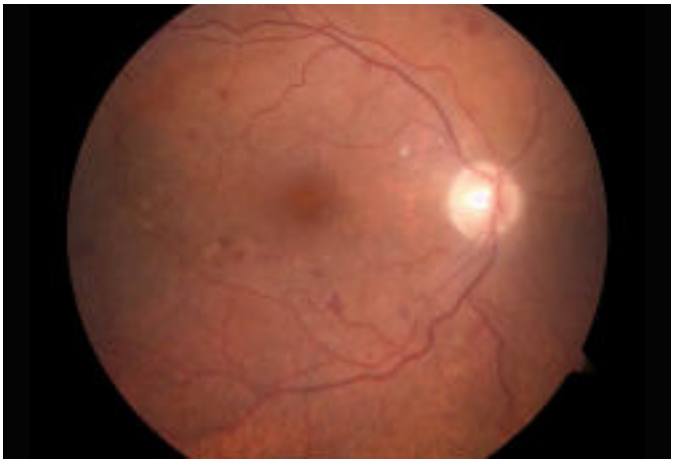
A diagnosis of preeclampsia or eclampsia should be considered in pregnant patients.[48] [55] Commonly described features of preeclampsia headache include severe bilateral frontal headache and blurry vision, which may progress to bilateral cortical blindness.[55] [56] The headache typically develops in temporal relation to the onset of preeclampsia, or substantially worsens or improves in parallel with worsening or improvement of preeclampsia.[55] In the setting of preeclampsia and headache, it is important to consider alternative secondary etiologies (e.g., reversible cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome, or infection) if accompanied by an altered level of consciousness, vomiting, or fever.[55]

Physical exam

An appropriately sized cuff should be used for blood pressure (BP) readings, so that the bladder encircles 80% of the arm.[1] [57] The arm should be supported at heart level during recordings. Using too large a cuff could result in an underestimation of BP; conversely, too small a cuff could lead to over-estimation. It should be noted if a larger- or smaller-than-normal cuff size is used.[1]

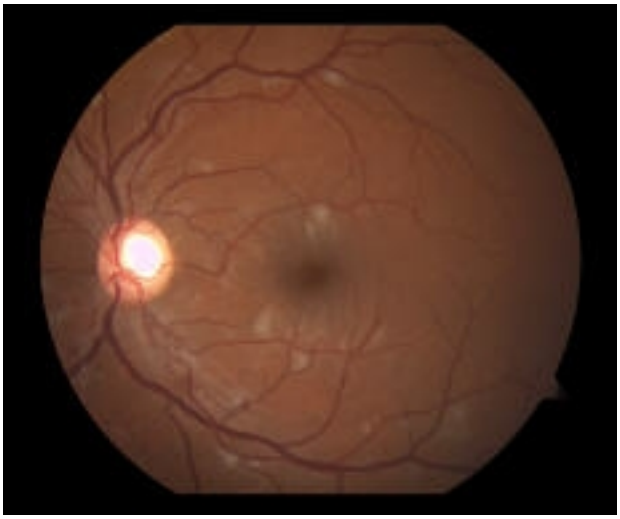
BP readings should be taken from both arms.[7] [54] Readings should be repeated after 5 minutes to confirm. If there is a more than 20 mmHg pressure difference between arms, aortic dissection should be considered.[58] [59] If blood pressure is elevated, a second measurement should be taken.[60]

A fundoscopic exam should be performed, with the aid of slit lamp exam and pupillary mydriasis if necessary, looking for the presence of arteriolar spasm, retinal edema, retinal hemorrhages, retinal exudates, papilledema, or engorged retinal veins.[7] [18]



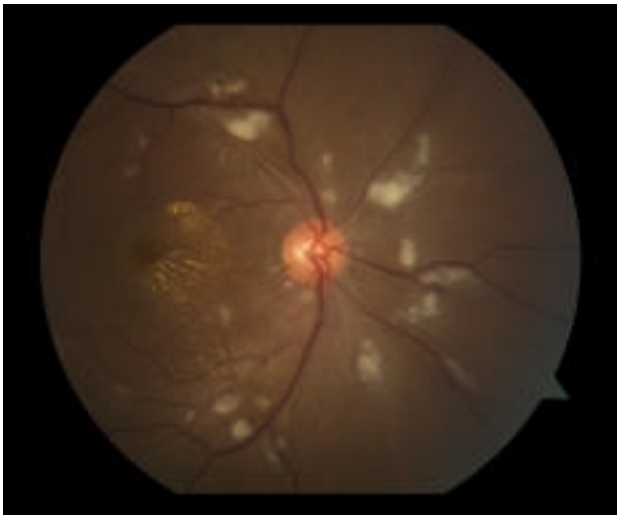
Fundus photograph of the right eye with multiple dot-blot hemorrhages typical of hypertensive retinopathy

Courtesy Angie Wen MD, Attending Faculty, New York Eye and Ear Infirmary, New York; used with permission



Fundus photograph of the left eye with multiple cotton-wool spots typical of hypertensive retinopathy

Courtesy Angie Wen MD, Attending Faculty, New York Eye and Ear Infirmary, New York; used with permission



Fundus photograph of the right eye centered on the optic nerve, showing multiple cotton-wool spots and macular exudates in a radiating star configuration around the fovea

Courtesy Angie Wen MD, Attending Faculty, New York Eye and Ear Infirmary, New York; used with permission

A rapid bedside neurologic exam is also required, including testing of cognition, cranial nerve function, dysarthria, motor strength, gross sensory function, upper extremity pronator drift, and gait.

Cardiopulmonary status should be assessed, examining in particular for the presence of new murmurs, friction rubs, additional heart sounds, lateral displacement of the apex beat, jugular venous distension, carotid or renal artery bruits, rales, and lower extremity edema.

Abdominal exam should be performed. Tenderness to palpation in the right upper quadrant is seen in severe preeclampsia and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome.[61] [62] Pheochromocytoma may be associated with an abdominal mass.

Renovascular hypertension should be suspected in patients with severe hypertension who have abdominal bruits and/or unexplained renal deterioration with angiotensin-converting enzyme inhibitor treatment, although the clinical presentation is variable.

During pregnancy, hypertension in a previously normotensive woman with proteinuria or evidence of systemic involvement (e.g., renal insufficiency, impaired liver function, neurologic complications, hematologic complications) is diagnostic of preeclampsia.[48] [61]

Preeclampsia should be considered in patients with headache who are at least 20 0/7 weeks of gestation, or within 6 weeks postpartum, and who have blood pressure ≥140 mmHg systolic or ≥90 mmHg diastolic.[55]

At least two measurements should be made, at least 4 hours apart.[48] The neurologic exam is typically normal in preeclampsia.[55]

See Preeclampsia (Diagnostic approach) .

Laboratory evaluation

Baseline blood and urine samples must be collected prior to administration of treatment. Laboratory evaluation should include the following:[7] [18] [54]

- Blood chemistry panel, including creatinine and electrolytes
- Complete blood count, including peripheral blood smear
- Urinalysis with microscopy.

In some circumstances, the following may also be indicated:

- Liver function tests, if preeclampsia or HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome are suspected.[48] [61]
- Cardiac enzymes and/or brain natriuretic peptide, if acute coronary syndrome or acute heart failure is suspected.[18]
- Coagulation profile, if disseminated intravascular coagulation is suspected.[18]
- Urine or serum pregnancy test (in women of childbearing age not known to be pregnant).[18]
- A urine drug screen, if illicit drug use is suspected.[63]
- Plasma renin activity and aldosterone levels, if primary aldosteronism is suspected (e.g., in patients with diastolic hypertension with persistent hypokalemia and metabolic alkalosis).
- Spot urine or plasma-free metanephrine levels, if pheochromocytoma is suspected (e.g., in patients with hypertension and palpitations, headaches, and/or diaphoresis, although clinical presentation is very variable).[18] [63] These tests need to be interpreted carefully, with consideration for possible confounding factors such as drugs (e.g., tricyclic antidepressants, clozapine, phenoxybenzamine, beta-blockers, sympathomimetics, buspirone), or major physiologic stress.
    - Do not use plasma catecholamines to rule out pheochromocytoma.[64] [65]
- Thyroid function tests, if signs of hypo- or hyperthyroidism.
- 24-hour urinary free cortisol, if Cushing syndrome is suspected.
- Sleep study, in cases of resistant hypertension and for patients with signs or symptoms of obstructive sleep apnea.[34]

Further investigation

ECG and chest x-ray should be strongly considered.[7] [54] If aortic dissection is considered possible, urgent thoracic computed tomography angiography (CTA) scan with contrast is recommended.[66] [67] For patients who cannot receive iodinated contrast, computed tomography (CT) without contrast is an acceptable alternative. Transthoracic echocardiography (TTE) may be used in the emergency department, intensive care unit (ICU), or operating room for acute proximal dissections if the patient is clinically unstable and there is any question about the diagnosis, or if CTA is unavailable or contraindicated.[66] [67] See Aortic dissection .

In clinical situations with high suspicion for renal artery disease, the use of doppler ultrasound, usually recommended as first-line imaging. This may be followed by magnetic resonance angiography and/or CTA.[68] Due to the potential risks with invasive procedures, angiography is generally limited to visualization and quantification of the stenosis before vascular intervention.[68]

If ischemic stroke or intracranial hemorrhage is suspected (e.g., in patients with decreased consciousness or those with focal neurological deficits), an urgent noncontrast CT scan of the head and/or a magnetic resonance imaging scan should be requested, depending on local availability.[18] Typically patients initially undergo a noncontrast head CT, in order to exclude a brain hemorrhage and guide treatment.[69] The mismatch between diffusion-weighted imaging and fluid-attenuated inversion recovery findings on magnetic resonance imaging (MRI) can be useful for selecting those who may benefit from intravenous thrombolysis.[69] However, MRI may take more than 30 minutes to complete, and is not universally available. See Ischemic stroke and Hemorrhagic stroke .

The American College of Obstetricians and Gynecologists recommends evaluating headaches in pregnancy that warrant brain or vascular imaging with magnetic resonance techniques that limit the use of gadolinium.[55]

History and exam

Key diagnostic factors

blood pressure (BP) >180/120 mmHg (common)

- BP is usually >180/120 mmHg in hypertensive emergencies; however, the key determinant is the presence of new or worsening end-organ damage.[1] [60] During pregnancy, hypertension in a previously normotensive woman with proteinuria or evidence of systemic involvement (e.g., renal insufficiency, impaired liver function, neurologic complications, hematologic complications) is diagnostic of preeclampsia.[48] [61] Preeclampsia should be considered in patients with headache who are at least 20 0/7 weeks of gestation, or within 6 weeks postpartum, and who have blood pressure ≥140 mmHg systolic or ≥90 mmHg diastolic.[55] At least two measurements should be made, at least 4 hours apart.[48]

Other diagnostic factors

neurologic symptoms (common)

- Neurologic abnormalities, such as vision changes, dizziness, headache, dysarthria, seizures, change in mental status, dysphagia, loss of sensation or paresthesia, and loss of movement, are symptoms often associated with hypertensive emergency.[40] Commonly described features of preeclampsia headache include severe bilateral frontal headache and blurry vision, which may progress to bilateral cortical blindness.[55] [56] The headache typically develops in temporal relation to the onset of preeclampsia, or substantially worsens or improves in parallel with worsening or improvement of preeclampsia.[55]

cardiac symptoms (common)

- Cardiac abnormalities (e.g., chest pain, shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, palpitations, edema) are frequently associated with hypertensive emergency.[40]

abnormal cardiopulmonary exam (common)

- The presence of new murmurs, friction rub, S3, jugular venous distension, rales, or lower extremity edema may be found.

abnormal abdominal exam (common)

- Tenderness to palpation in the right upper quadrant is seen in severe preeclampsia and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome.[61] [62] Pheochromocytoma may be associated with an abdominal mass. Renovascular hypertension should be suspected in patients with severe hypertension who have abdominal bruits and/or unexplained renal deterioration with angiotensin-converting enzyme inhibitor treatment, although the clinical presentation is variable.

oliguria or polyuria (common)

- Any changes in renal output can be indicative of renal damage.[24]

abnormal fundoscopic exam (common)

- The following signs are indicative of hypertensive retinopathy: arteriolar spasm, retinal edema, retinal hemorrhages, retinal exudates, papilledema, engorged retinal veins.[7][71]

abnormal neurologic exam (common)

- Abnormal findings in cognition, cranial nerve function, motor strength, gross sensory function, and gait can frequently result from hypertensive crisis.

Risk factors

Strong

inadequately treated hypertension

- A history of inadequately treated hypertension is commonly seen.[18] [21] [22] [23]

chronic kidney disease

- Chronic kidney disease is a strong risk factor for hypertension and progression to hypertensive emergencies in both adults and children.[13] [18][24] [25] [26] [27]

renal artery stenosis

- Renal artery stenosis is strongly associated with secondary hypertension.[28]

renal transplant

- Renal transplantation is commonly associated with hypertension, with graft failure most commonly responsible.[29] Transplant renal artery stenosis accounts for between 1% and 5% of hypertension after transplantation.[30] Anti-rejection medication (e.g., calcineurin inhibitors) may also play a role.[7] [31] [39]

endocrine disorders with known hypertensive effects

- There are a number of endocrine disorders that are associated with hypertensive emergencies. These include: primary aldosteronism, pheochromocytoma, thyroid disorder, Cushing syndrome, acromegaly, hyperparathyroidism, carcinoid tumor, congenital adrenal hyperplasia, or renin-secreting tumor.[31] [35] [36][37] The treatment of certain endocrine disorders may also precipitate a hypertensive emergency. For example, the use of beta-blocker medication before the administration of an alpha-adrenergic receptor blocker in a patient with a pheochromocytoma may lead to a hypertensive crisis.[46][47]

pregnancy

- Preeclampsia, eclampsia, and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome can all result in a hypertensive emergency.[31] [48]

Weak

older age

- Older age predisposes to hypertensive emergency.[10] [11] [12] [13]

black ethnicity

- Black people are predisposed to hypertensive emergency, compared with white people.[11] [12]

male sex

- Men may be more likely than women to suffer a hypertensive emergency.[11] [12]

use of sympathomimetic drugs

- Use of sympathomimetic street drugs (e.g., cocaine, LSD, amphetamines, ecstasy) predisposes to hypertensive emergency.[7][18]

pharmacotherapy with known hypertensive effect

- Many medications can induce or exacerbate hypertension, leading to a hypertensive emergency. These include nonsteroidal anti-inflammatory drugs, oral contraceptives, sympathomimetics, illicit drugs, glucocorticoids, mineralocorticoids, calcineurin inhibitors, erythropoietin, herbal supplements, vascular endothelial growth factor inhibitors, and inadvertent drug or food interactions with monoamine oxidase inhibitors (MAOIs).[7][31] [39] If foods high in tyramine are ingested by patients taking MAOIs, this can precipitate a hypertensive emergency (the so called 'tyramine reaction').

obstructive sleep apnea

- Obstructive sleep apnea is associated with secondary hypertension which, if left untreated, may precipitate a hypertensive emergency.[32] [33] [34]

vasculitis and connective tissue diseases

- Multiple vasculitides and connective tissue disorders are associated with hypertension and hypertensive emergencies. These include scleroderma, systemic lupus erythematosus, Takayasu's arteritis, and giant cell arteritis.[31] [49] [50] [51]

Tests

1st test to order

Test	Result
blood chemistry • Acute kidney injury may be the only sign of hypertensive emergency.	**may reveal elevated creatinine and BUN**
CBC with smear • Microangiopathic hemolytic anemia may occur in patients with hypertensive emergency and increases the risk of developing acute kidney injury.[72] Additional evidence for hemolysis may be obtained by checking a serum LDH, haptoglobin, and indirect bilirubin.	**may reveal schistocytes (red cell fragments) indicating the presence of hemolysis**
urinalysis with microscopy • Acute kidney injury as manifested by hematuria and proteinuria may be the only sign of hypertensive emergency.	**may reveal presence of red cells and protein**
ECG • If the patient has chest pain and there is ST elevation on the ECG, the patient should be sent for emergency revascularization. • If the ECG is abnormal but the ST segment is not elevated, biomarkers (high-sensitivity troponin), and echocardiogram are the first-line investigations in all patients to rule out ongoing ischemia or infarction. • If the ECG is normal, aortic dissection should be considered in the context of unexplained chest pain.	**may reveal evidence of ischemia or infarct such as ST- or T-wave changes**
chest x-ray • A chest x-ray is useful to assess for pulmonary edema, left ventricular hypertrophy, and aortic dissection. • A plain chest x-ray is neither sufficiently sensitive nor specific for aortic dissection to be used as a diagnostic tool. If aortic dissection is suspected, urgent computed tomography angiography (CTA) with contrast should be ordered.	**may reveal evidence of pulmonary edema indicating left ventricular failure or widened mediastinum indicating possible aortic dissection**

Test to avoid

Recommendations	Rationale
plasma catecholamines • Do not use plasma catecholamines to rule out pheochromocytoma as a cause of hypertension. Use urine or plasma metanephrine instead.[64] [65]	• **Measurement of metabolites including metanephrine provides a more sensitive diagnostic test than catecholamines.[65] [73]**

Other tests to consider

Test	Result
thyroid function tests • Indicated if signs/symptoms of hypothyroidism or hyperthyroidism.	**thyroid-stimulating hormone (TSH) high and thyroxine (T4) low in primary hypothyroidism; TSH low/normal and T4 low in central hypothyroidism; TSH low and T4 high in primary hyperthyroidism (e.g., Graves disease); TSH high and T4 high in central hyperthyroidism (e.g., in rare pituitary tumors)**
liver function tests • Recommended in all women with suspected preeclampsia. Useful indicator of disease progression. Increased transaminase levels are partly diagnostic for HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome.	**may be abnormal**
cardiac enzymes • Cardiac enzymes should be performed if acute coronary syndrome is suspected.	**elevated in acute coronary syndrome**
N-terminal pro-B-type natriuretic peptide (NT-proBNP) • BNP should be measured if acute heart failure is suspected. If NT-proBNP levels are normal it is unlikely that the patient has heart failure.	**may be elevated in acute heart failure**
coagulation profile • Coagulation profile should be performed if disseminated intravascular coagulation is suspected. Prolonged prothrombin time/partial thromboplastin time in addition to thrombocytopenia is indicative of progression to disseminated intravascular coagulation.	**may be abnormal**
urine or serum pregnancy test • Performed in women of childbearing age not known to be pregnant. All pregnant women presenting with hypertension and either proteinuria or evidence of systemic involvement require close assessment and monitoring for preeclampsia and its complications.	**positive in pregnancy**
urine toxicology screen • Performed in patients with suspected ingestion of illicit substances.	**may be positive for illicit substances**
computed tomography angiography (CTA) scan • If aortic dissection is considered possible, an urgent thoracic CTA scan with contrast is recommended.[66] [67] For patients who cannot receive iodinated contrast, computed tomography (CT) without contrast is an acceptable alternative. • Transthoracic echocardiography (TTE) may be used in the emergency department, intensive care unit (ICU), or operating room for acute proximal dissections if the patient is clinically unstable and there is any question about the diagnosis, or if CTA is unavailable or contraindicated.[66] [67]	**evidence of two separate aortic lumens with dividing intimal flap in aortic dissection**

Test	Result
transthoracic echocardiography (TTE) • TTE may be used in the emergency department, ICU, or operating room for acute proximal dissections if the patient is clinically unstable and there is any question about the diagnosis, or if CTA is unavailable or contraindicated.[66] [67]	**evidence of two separate aortic lumens with dividing intimal flap in aortic dissection**
renal ultrasound with Doppler • Doppler ultrasound is usually the first-line imaging in clinical situations with high suspicion for renal artery disease. This may be followed by magnetic resonance angiography and/or CTA.[68] Due to the potential risks with invasive procedures, angiography is generally limited to visualization and quantification of the stenosis before vascular intervention.[68]	**may reveal increased renal artery resistive indices**
head CT without contrast • Indicated if neurologic complications are suspected. In patients suspected of having a stroke, CT or magnetic resonance imaging (MRI) of the brain is recommended to confirm the diagnosis of symptomatic ischemic cerebral vascular disease. Typically patients initially undergo a noncontrast head CT, in order to exclude a brain hemorrhage and guide treatment.[69]	**may reveal evidence of infarct or hemorrhage**
head MRI • In patients suspected of having a stroke, CT or MRI of the brain is recommended to confirm the diagnosis of symptomatic ischemic cerebral vascular disease. The mismatch between diffusion-weighted imaging and fluid-attenuated inversion recovery findings on MRI can be useful for selecting those who may benefit from intravenous thrombolysis.[69] However, MRI may take more than 30 minutes to complete, and is not universally available. The American College of Obstetricians and Gynecologists recommends evaluating headaches in pregnancy that warrant brain or vascular imaging with magnetic resonance techniques that limit the use of gadolinium.[55]	**may reveal evidence of infarct or hemorrhage**
plasma renin activity and aldosterone level • This test is an indirect measure of the activity of renin through measurement of the rate of production of angiotensin I, which increases as a result of renin stimulation. Aldosterone levels are usually measured at the same time. High plasma renin activity suggests hypertension from the vasoconstrictive effects of angiotensin.	**in primary hyperaldosteronism, renin activity will be decreased and aldosterone levels increased; in secondary hyperaldosteronism, both renin activity and aldosterone levels will be increased**
spot urine or plasma metanephrine • May be useful before initiation of drug therapy to rule out pheochromocytoma. However, these tests need to be interpreted carefully, with consideration for possible confounding factors such as drugs (e.g., tricyclic antidepressants, clozapine, phenoxybenzamine, beta-blockers, sympathomimetics, buspirone) or major physiologic stress. Do not use plasma catecholamines to rule out pheochromocytoma.[64] [65]	**may reveal elevated metanephrine levels**
24-hour urine free cortisol • Indicated when stigmata of Cushing syndrome present.	**elevated in Cushing syndrome**

Test	Result
sleep study • Sleep study may be considered in cases of resistant hypertension and also for patients with signs or symptoms of obstructive sleep apnea.[34]	**may show results consistent with obstructive sleep apnea**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Hypertensive urgency	• BP is above 180/120 mmHg but the patient is stable and there is no organ dysfunction.	• History, physical exam, laboratory tests, and imaging show no evidence of end-organ damage.
Uncontrolled essential hypertension	• Asymptomatic elevated BP. • BP is less than 180/120 mmHg.	• History, physical exam, laboratory tests, and imaging show no evidence of end-organ damage.

Approach

If hypertensive emergency is suspected, treatment should not be delayed while conducting a full diagnostic evaluation.

Appropriate facilities

Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure (BP) and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][18] [74] Other supportive measures that may be required include intracranial pressure monitoring (in rare cases of increased intracranial pressure), noninvasive ventilation or intubation (in cases of respiratory distress), or dialysis (in case of severe acute kidney injury).

Choice of agents and route of administration

The specific parenteral agents used for treating a hypertensive emergency should be dictated by the end-organ systems that have been damaged, patient comorbidities, and overall clinical condition. Oral therapies are generally discouraged as first-line treatment options.[1] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7] There are very few randomized controlled trials studying different parenteral agents in hypertensive emergency. Published guidelines are therefore based on common clinical experience and practice.

Rate of BP reduction

The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

Exceptions to the above recommendation include:[1] [75]

- Patients with an ischemic stroke, as there is no clear evidence from clinical trials to support the use of immediate antihypertensive treatment
- Patients who are candidates for thrombolytic therapy (typically those with ischemic stroke), who should have their BP slowly lowered to systolic BP (SBP) <185 mmHg and diastolic BP <110 mmHg before intravenous tissue plasminogen activator is initiated
- Patients with severe preeclampsia, eclampsia, or pheochromocytoma crisis, in whom SBP should be reduced to <140 mmHg in the first hour
- Patients with aortic dissection, in whom accepted practice is reduction of SBP to <120 mmHg in the first 20 minutes, although evidence to support this timescale is lacking.

Accelerated (malignant) hypertension, hypertensive encephalopathy or intracranial hemorrhage

The term "accelerated hypertension" (also known as malignant hypertension) is a subcategory of hypertensive emergency where severe hypertension occurs with retinopathy of grade III (flame hemorrhages, dot and blot hemorrhages, hard and soft exudates) or grade IV (papilledema).[18] [54]

Hypertensive encephalopathy encompasses the transient neurologic symptoms (lethargy, seizures, cortical blindness, and coma) that occur with malignant hypertension, which are usually reversed by prompt treatment and lowering of BP.[18] [54] Encephalopathy should improve once the BP is lowered. If there is no improvement despite a decrease in BP, an alternative diagnosis should be considered.

In the management of intracerebral hemorrhage (ICH), the patient's ideal BP should be based on individual factors, including baseline BP, presumed cause of hemorrhage, age, elevated intracranial pressure, and interval since onset. In cases of large or severe ICH, or an initial SBP ≥220 mmHg, cautious BP lowering should be pursued.[76] In patients with initial SBP ≥220 mmHg, early intensive BP reduction, compared with standard BP lowering, was associated with higher rates of renal adverse events in one post-hoc analysis of a large randomized clinical trial.[76] [77]

While elevated BP could in theory increase the risk of ongoing bleeding from ruptured small arteries and arterioles, the relationship between BP, intracranial pressure, and volume of hemorrhage is complex and not yet fully understood.

The rationale for lowering BP is to minimize further hemorrhage: for example, from a ruptured aneurysm or arteriovenous malformation. However, the evidence for the effectiveness and safety of rapid BP lowering in the management of intracerebral hemorrhage remains inconclusive.[76] [78][79] [80]

For the management of patients with spontaneous ICH, recommendations from the American Heart Association and American Stroke Association (AHA/ASA) include the following, based on two of the largest trials for early intensive BP lowering after ICH (INTERACT2, ATACH-2), meta-analyses, and several post-hoc analyses of these two trials:[76] [78] [79]

- In patients with spontaneous ICH requiring acute BP lowering, careful titration to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP, can be beneficial for improving functional outcomes.
- Initiating blood pressure treatment within 2 hours of ICH onset, and reaching target within 1 hour, can be beneficial to reduce the risk of hematoma expansion and improve functional outcomes.
- In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mmHg, acute lowering of SBP to a target of 140 mmHg, with the goal of maintaining it within the range of 130-150 mmHg, is safe and may be reasonable for improving functional outcomes. Acute lowering of SBP to <130 mmHg is potentially harmful in these patients.
- In patients with spontaneous ICH presenting with large or severe ICH, SBP >220 mmHg, more than 6 hours after symptom onset, or in those requiring surgical decompression, the safety and efficacy of intensive BP lowering is not well established.

See Hemorrhagic stroke (Management approach) .

Treatment options include the following.

- First-line

- Labetalol is the first-line treatment for accelerated (malignant) hypertension, hypertensive encephalopathy, or intracranial hemorrhage.[18] [39] [41] [42]

- Second-line
  - Nicardipine is a second-line agent.[18] One randomized controlled trial found that intravenous nicardipine significantly increased the proportion of people who reached physician-specified target range SBP within 30 minutes compared with intravenous labetalol.[81] Nicardipine is especially useful in the presence of cardiac disease due to coronary vasodilatory effects.[82] [83]
  - If patients do not have evidence of raised intracranial pressure, nitroprusside is a second-line treatment choice.[39] [41] However, if raised intracranial pressure is present or suspected, nitroprusside is contraindicated and another agent should be used.[18] [84] Nitroprusside decreases cerebral blood flow while increasing intracranial pressure, effects that are particularly disadvantageous in patients with hypertensive encephalopathy or following a stroke.[85] [86] [87] It should also be avoided in patients with renal or hepatic insufficiency.
- Third-line
  - The third-line treatment choice is fenoldopam, a selective peripheral dopamine-1-receptor agonist with arterial vasodilator effects.[39] [41] [42] [88] [89] This drug is particularly useful in patients with renal insufficiency, where the use of nitroprusside is restricted due to the risk of thiocyanate poisoning.

Acute ischemic stroke

Treating a hypertensive emergency with an associated acute ischemic stroke warrants greater caution in reducing BP than in other types of hypertensive emergency. Overly rapid or large reductions of mean arterial pressure may decrease cerebral perfusion pressure to a level that could theoretically worsen brain injury. However, AHA/ASA guidelines recommend early treatment of hypertension when required by comorbid conditions, including preeclampsia/eclampsia.[69] The following may be used as guidance.

If the SBP is >220 mmHg or the diastolic BP is >120 mmHg, it may be reasonable to lower the BP by 15% during the first 24 hours after the onset of stroke.[1] [90]

If the SBP is <220 mmHg and the diastolic BP is <120 mmHg, then it is reasonable to maintain close observation without direct intervention to reduce BP, unless:[1] [91] [92]

- There is other end-organ involvement such as aortic dissection, renal failure, or acute myocardial infarction
- The patient is to receive thrombolysis, in which case the target SBP should be <185 mmHg and diastolic BP <110 mmHg. The BP should be maintained <185/105 mmHg for at least 24 hours after initiating intravenous thrombolysis.

If the SBP is >220 mmHg or diastolic BP is between 121-140 mmHg, then labetalol, nicardipine, or clevidipine should be used to achieve a 10% to 15% reduction in BP in 24 hours.[39] [41] [42] [93] [94]

If diastolic BP is >140 mmHg, then nitroprusside is used to achieve a 10% to 15% reduction over 24 hours.[39] [41] [95]

Myocardial ischemia/infarction

First-line treatment of hypertensive emergency complicated by myocardial ischemia or infarction is the combination of esmolol (a selective beta-blocker) plus nitroglycerin (a peripheral vasodilator, which affects venous vessels more than arterial).[1] [39] [41] [42] [95] [96]

Esmolol acts to reduce the heart rate and nitroglycerin acts to decrease preload and cardiac output, and increases coronary blood flow.

Second-line treatment choice would be labetalol plus nitroglycerin.[39] [41] [42] [95] [96]

Contraindications to beta-blockers include moderate-to-severe left ventricular failure with pulmonary edema, bradycardia, hypotension, poor peripheral perfusion, second- or third-degree heart block, and reactive airway disease.[1]

The third-line treatment choice would be nitroprusside.[39] [41] [95]

Left ventricular failure and/or pulmonary edema

First-line treatment of hypertensive emergency with left ventricular failure and/or pulmonary edema is nitroglycerin or clevidipine.[39] [41] [42] [96]

Nitroprusside (a potent arterial and venous vasodilator that decreases after-load and preload) is the second-line treatment choice in this situation.[39] [41] [95]

If the patient is not already on a loop diuretic, one should be started (e.g., furosemide). Beta-blockers are contraindicated in moderate-to-severe left ventricular failure with pulmonary edema.[1]

Suspected aortic dissection

If aortic dissection is suspected in a hypertensive emergency, the BP should be lowered quite aggressively, typically with a target of reducing the SBP to <120 mmHg within 20 minutes, although evidence to support this timescale is lacking.[1] [75]

Medical therapy aims to both lower the BP and decrease the velocity of left ventricular contraction, so decreasing aortic shear stress and minimizing the tendency for propagation of the dissection.

First-line treatment choice is beta-blockers, either labetalol or esmolol, administered intravenously.[39] [41] [42] [97]

If there is no significant improvement, nitroprusside or nicardipine can be added to the beta-blocker.[39] [41] [42] [97] The beta-blockade should precede vasodilator (nicardipine or nitroprusside) administration to prevent reflex tachycardia and worsen shear stress on the intimal flap.[1] See Aortic dissection .

Acute kidney injury

Fenoldopam is the first-line treatment choice of hypertensive emergency complicated by acute kidney injury.[39] [41] [42] [88] [89] This drug (a selective peripheral dopamine-1-receptor agonist with arterial vasodilator effects) is particularly useful in renal insufficiency because it acts to both decrease afterload and increase renal perfusion. Other potential first-line agents are dihydropyridine calcium-channel blockers (e.g., nicardipine, clevidipine), which increase stroke volume and have strong cerebral and coronary vasodilatory activity.[1] [39] [41] [42] [93]

Hyperadrenergic states

Hyperadrenergic states include:

- Pheochromocytoma
- Sympathomimetic drug use: for example, cocaine, amphetamines, phenylpropanolamine, phencyclidine, or the combination of monoamine oxidase inhibitors with foods rich in tyramine
- Following abrupt discontinuation of a short-acting sympathetic blocker.

If the hyperadrenergic state is due to sympathomimetic drug use, the first-line agents are benzodiazepines, and antihypertensive medications are given only if the BP response is inadequate.[18] [54] Benzodiazepines reduce agitation and prevent neurologic complications such as seizures by their action on gamma-aminobutyric acid receptors.[18] In all other clinical situations, the first-line treatment choice is phentolamine (which acts by blocking alpha-adrenoceptors) or calcium-channel blockers (clevidipine and nicardipine).[1] [39] [41] [42] A beta-blocker (such as labetalol) can be added after sufficient alpha-adrenoceptor blockade. The administration of a beta-blocker alone is contraindicated, since inhibition of beta-adrenoceptor-induced vasodilation results in unopposed alpha-adrenergic vasoconstriction and a further rise in BP.[54]

Severe hypertension in pregnancy (preeclampsia and eclampsia)

For acute-onset, severe hypertension managed in a critical care setting, intravenous labetalol, intravenous hydralazine, or oral nifedipine can be used first line. Labetalol is usually considered the antihypertensive of choice, and is effective as monotherapy in 80% of pregnant women.[48] [61][98] [99] It seems to be safe and effective in pregnant women for the management of preeclampsia; however, it should be avoided in women with asthma or any other contraindication to its use.[48] Immediate-release oral nifedipine may also be considered first-line therapy, particularly when intravenous access is not available.[48] Although intravenous hydralazine is still widely used, particularly in North America, it can produce an acute fall in BP.[63] The consequences of this are mostly related to maternal hypotension, including a greater risk of cesarean section, more frequent placental abruption, more maternal oliguria, and fetal tachycardia, suggesting the need for close monitoring of maternal BP and fetal wellbeing during its use.[63] The UK guidelines recommend to consider volume expansion with crystalloid fluid before or at the same time as the first dose of intravenous hydralazine in the prenatal period.[61]

If second-line alternatives are required, the choice of agent should be discussed with an appropriate subspecialist in fetal-maternal medicine or critical care. Availability of drugs and local experience with individual drugs are likely to influence the choice of treatment.[54]

In pregnancy, ACE inhibitors or angiotensin-II receptor antagonists are avoided due to potential teratogenic effects, and nitroprusside is avoided due to its potential for fetal cyanide poisoning.[1] Renin inhibitors are also contraindicated.[1]

The American College of Obstetricians and Gynecologists (ACOG) recommends antihypertensive therapy for women with preeclampsia and a sustained SBP ≥160 mmHg or diastolic BP ≥110 mmHg.[48] However, thresholds for treatment vary internationally, with lower thresholds recommended by a number of societies.[100] The UK National Institute for Health and Care Excellence recommends treatment if BP remains above 140/90 mmHg.[61]

Although some of the available literature suggests that antihypertensive agents should be administered within 30-60 minutes, it is recommended that antihypertensive therapy begin as soon as reasonably possible after the criteria for acute onset severe hypertension are met.[48] It should be noted, however,

that there are no trials supporting these suggested thresholds, and treatments should be tailored to individual patient circumstances. Specialist advice should be sought.

Magnesium sulfate is not recommended as an antihypertensive agent, but remains the drug of choice for treatment of eclampsia and/or seizure prophylaxis for women with acute-onset severe hypertension during pregnancy and the postpartum period.[48]

See Preeclampsia (Management approach) .

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)

accelerated (malignant) hypertension or hypertensive encephalopathy or intracranial hemorrhage		
▪ increased intracranial pressure or renal disease	1st	labetalol
	2nd	nicardipine
	3rd	fenoldopam
▪ normal intracranial pressure and renal function	1st	labetalol
	2nd	nitroprusside or nicardipine
	3rd	fenoldopam
acute ischemic stroke		
▪ SBP ≤220 mmHg and DBP ≤120 mmHg	1st	close observation ± blood pressure reduction
▪ SBP >220 mmHg or DBP 121-140 mmHg	1st	labetalol
	1st	nicardipine or clevidipine
▪ DBP >140 mmHg	1st	nitroprusside
myocardial ischemia/infarction		
	1st	esmolol and nitroglycerin
	2nd	labetalol and nitroglycerin
	3rd	nitroprusside
left ventricular failure and/or pulmonary edema		
	1st	nitroglycerin + furosemide
	1st	clevidipine + furosemide
	2nd	nitroprusside + furosemide
aortic dissection		
	1st	labetalol or esmolol

Acute (summary)		
	adjunct	nitroprusside or nicardipine
acute kidney injury		
	1st	fenoldopam
	1st	nicardipine or clevidipine
hyperadrenergic state		
▪ sympathomimetic drug use	1st	benzodiazepine
	2nd	phentolamine
	2nd	nicardipine or clevidipine
▪ no sympathomimetic drug use	1st	phentolamine
	adjunct	labetalol
	1st	nicardipine or clevidipine
severe hypertension in pregnancy (preeclampsia and eclampsia)		
	1st	labetalol or hydralazine or nifedipine

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

accelerated (malignant) hypertension or hypertensive encephalopathy or intracranial hemorrhage

increased intracranial pressure or renal disease — **1st** — **labetalol**

Primary options

» labetalol: 20 mg intravenously every 10 minutes according to response, maximum 300 mg total dose; or 0.5 to 2 mg/minute intravenous infusion

» Labetalol is the drug of choice in situations characterized by markedly elevated intracranial pressure.[18] [39] [41] [42]

» Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Encephalopathy is usually reversed by prompt treatment and lowering of BP.[18] [54] If there is no improvement despite a decrease in BP, an alternative diagnosis should be considered.

» Dose should be adjusted to maintain BP in desired range and is continued until BP controlled on oral agents.

» In the management of intracerebral hemorrhage (ICH), the patient's ideal BP should be based on individual factors, including baseline BP, presumed cause of hemorrhage,

Acute

age, elevated intracranial pressure, and interval since onset. In cases of large or severe ICH, or an initial systolic BP (SBP) ≥220 mmHg, cautious BP lowering should be pursued.[76] In patients with initial SBP ≥220 mmHg, early intensive BP reduction, compared with standard BP lowering, was associated with higher rates of renal adverse events in one post-hoc analysis of a large randomized clinical trial.[76] [77]

» For the management of patients with spontaneous ICH, the American Heart Association and American Stroke Association (AHA/ASA) recommend careful titration in patients requiring acute BP lowering, to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP. This can be beneficial for improving functional outcomes.[76] Initiating treatment within 2 hours of ICH onset, and reaching target within 1 hour, can be beneficial to reduce the risk of hematoma expansion and improve functional outcome.[76] In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mmHg, acute lowering of SBP to a target of 140 mmHg, with the goal of maintaining it within the range of 130-150 mmHg, is safe and may be reasonable for improving functional outcomes.[76] In patients with spontaneous ICH presenting with large or severe ICH, SBP >220 mmHg, more than 6 hours after symptom onset, or in those requiring surgical decompression, the safety and efficacy of intensive BP lowering is not well established.[76]

» These recommendations from the AHA/ASA are based on two of the largest trials for early intensive BP lowering after ICH (INTERACT2, ATACH-2), meta-analyses, and several post-hoc analyses of these two trials.[76] [78] [79]

» See Hemorrhagic stroke (Management approach) .

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

2nd **nicardipine**

Primary options

MANAGEMENT

Acute

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

» Nicardipine is a second-generation dihydropyridine derivative calcium-channel blocker with high vascular selectivity and strong cerebral and coronary vasodilatory activity.[18] The onset of action of intravenous nicardipine is from 5-15 minutes, with a duration of action of 4-6 hours.

» Nicardipine is especially useful in the presence of cardiac disease due to coronary vasodilatory effects.[82] [83]

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» In the management of intracerebral hemorrhage (ICH), the patient's ideal BP should be based on individual factors, including baseline BP, presumed cause of hemorrhage, age, elevated intracranial pressure, and interval since onset. In cases of large or severe ICH, or an initial SBP ≥220 mmHg, cautious BP lowering should be pursued.[76] In patients with initial SBP ≥220 mmHg, early intensive BP reduction, compared with standard BP lowering, was associated with higher rates of renal adverse events in one post-hoc analysis of a large randomized clinical trial.[76] [77]

» For the management of patients with spontaneous ICH, the AHA/ASA recommend careful titration in patients requiring acute BP lowering, to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP. This can be beneficial for improving functional outcomes.[76] Initiating treatment within 2 hours of ICH onset, and

Acute

reaching target within 1 hour, can be beneficial to reduce the risk of hematoma expansion and improve functional outcome.[76] In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mmHg, acute lowering of SBP to a target of 140 mmHg, with the goal of maintaining it within the range of 130-150 mmHg, is safe and may be reasonable for improving functional outcomes.[76] In patients with spontaneous ICH presenting with large or severe ICH, SBP >220 mmHg, more than 6 hours after symptom onset, or in those requiring surgical decompression, the safety and efficacy of intensive BP lowering is not well established.

» These recommendations from the AHA/ASA are based on two of the largest trials for early intensive BP lowering after ICH (INTERACT2, ATACH-2), meta-analyses, and several post-hoc analyses of these two trials.[76] [78] [79]

» See Hemorrhagic stroke (Management approach) .

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

3rd **fenoldopam**

Primary options

» fenoldopam: 0.1 to 0.3 micrograms/kg/minute intravenously initially, increase by 0.05 to 0.1 micrograms/kg/minute increments every 15 minutes according to response, maximum 1.6 micrograms/kg/minute

» Fenoldopam is especially useful in renal insufficiency, where the use of nitroprusside is restricted because of the risk of thiocyanate poisoning.

» It acts as a selective peripheral dopamine-1-receptor agonist with arterial vasodilator effects. Its hemodynamic effects are a decrease in afterload and an increase in renal perfusion.

» Onset of action: 5 minutes. Duration of action: 30 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention,

Acute

Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Encephalopathy is usually reversed by prompt treatment and lowering of BP.[18] [54] If there is no improvement despite a decrease in BP, an alternative diagnosis should be considered.

» Continue until the BP is controlled on oral agents.

» For the management of patients with spontaneous ICH, the AHA/ASA recommend careful titration in patients requiring acute BP lowering, to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP. This can be beneficial for improving functional outcomes.[76] Initiating treatment within 2 hours of ICH onset, and reaching target within 1 hour, can be beneficial to reduce the risk of hematoma expansion and improve functional outcome.[76] In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mmHg, acute lowering of SBP to a target of 140 mmHg, with the goal of maintaining it within the range of 130-150 mmHg, is safe and may be reasonable for improving functional outcomes.[76] In patients with spontaneous ICH presenting with large or severe ICH, SBP >220 mmHg, more than 6 hours after symptom onset, or in those requiring surgical decompression, the safety and efficacy of intensive BP lowering is not well established.[76]

» These recommendations from the AHA/ASA are based on two of the largest trials for early intensive BP lowering after ICH (INTERACT2, ATACH-2), meta-analyses, and several post-hoc analyses of these two trials.[76] [78] [79]

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure

Acute

and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

normal intracranial pressure and renal function

1st **labetalol**

Primary options

» labetalol: 20 mg intravenously every 10 minutes according to response, maximum 300 mg total dose; or 0.5 to 2 mg/minute intravenous infusion

» Labetalol is the drug of choice.

» Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

» In cases of intracranial hemorrhage, treatment should commence if the initial SBP is above 220 mmHg. The American College of Cardiology/ American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Encephalopathy is usually reversed by prompt treatment and lowering of BP.[18] [54] If there is no improvement despite a decrease in BP, an alternative diagnosis should be considered.

» Dose should be adjusted to maintain BP in desired range and is continued until BP controlled on oral agents.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

2nd **nitroprusside or nicardipine**

Primary options

Acute

» nitroprusside: 0.3 to 0.5 micrograms/kg/minute intravenously initially, increase by 0.5 micrograms/kg/minute increments according to response, maximum 10 micrograms/kg/minute

OR

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

» Nitroprusside acts as a potent arterial and venous vasodilator thereby reducing afterload and preload. Its hemodynamic effects are to decrease mean arterial pressure, with a modest increase or no change in cardiac output. Onset of action: immediate. Duration of action: 3-5 minutes.

» Patients should be monitored by drawing thiocyanate levels after 48 hours of therapy (levels kept at <12 mg/dL). The maximum dose should be delivered for less than 10 minutes to decrease the chance of cyanide toxicity.

» Nicardipine is a second-generation dihydropyridine derivative calcium-channel blocker with high vascular selectivity and strong cerebral and coronary vasodilatory activity. The onset of action of intravenous nicardipine is from 5-15 minutes, with a duration of action of 4-6 hours. It is especially useful in the presence of cardiac disease due to coronary vasodilatory effects.[82] [83]

» In cases of intracranial hemorrhage, treatment should commence if the initial SBP is above 220 mmHg. The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

Acute

» Encephalopathy should improve once the BP is lowered. If there is no improvement despite a decrease in BP, an alternative diagnosis should be considered.

» Continue until the BP is controlled on oral agents.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

3rd **fenoldopam**

Primary options

» fenoldopam: 0.1 to 0.3 micrograms/kg/minute intravenously initially, increase by 0.05 to 0.1 micrograms/kg/minute increments every 15 minutes according to response, maximum 1.6 micrograms/kg/minute

» Fenoldopam acts as a selective peripheral dopamine-1-receptor agonist with arterial vasodilator effects. Its hemodynamic effects are a decrease in afterload and an increase in renal perfusion.

» Onset of action: 5 minutes. Duration of action: 30 minutes.

» In cases of intracranial hemorrhage, treatment should commence if the initial SBP is above 220 mmHg. The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Encephalopathy should improve once the BP is lowered. If there is no improvement despite a decrease in BP, an alternative diagnosis should be considered.

Acute

» Continue until the BP is controlled on oral agents.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

acute ischemic stroke

SBP ≤220 mmHg and DBP ≤120 mmHg — **1st**

close observation ± blood pressure reduction

» Treatment of hypertension with an associated acute ischemic stroke warrants greater caution in reducing blood pressure (BP) than with other types of hypertensive emergency. Overly rapid or too great a reduction of mean arterial pressure may decrease cerebral perfusion pressure to a level that could theoretically worsen brain injury (e.g., through watershed infarcts). However, American Heart Association/American Stroke Association guidelines recommend early treatment of hypertension when required by comorbid conditions, including preeclampsia/eclampsia.[69]

» If the systolic BP (SBP) is below 220 mmHg and the diastolic BP is below 120 mmHg, there is no evidence of end organ involvement or intracranial hemorrhage and thrombolytic treatment is not proposed, then it is reasonable to maintain close observation without direct intervention to reduce BP.

» If there is other end-organ involvement such as aortic dissection, acute kidney injury, or acute myocardial infarction, or the patient is to receive thrombolysis, the target SBP should be below 185 mmHg and diastolic BP should be below 110 mmHg. The BP should be maintained below 185/105 mmHg for at least 24 hours after initiating intravenous thrombolysis.

» The choice of agent to reduce BP depends on the associated end-organ involvement.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

Acute

SBP >220 mmHg or DBP 121-140 mmHg

1st **labetalol**

Primary options

» labetalol: 20 mg intravenously every 10 minutes according to response, maximum 300 mg total dose; or 0.5 to 2 mg/minute intravenous infusion

» If the SBP is above 220 mmHg or the diastolic BP is between 121-140 mmHg, labetalol can be used to achieve a 10% to 15% reduction in BP within 24 hours.[39] [41] [42]

» Labetalol acts as an alpha-1-blocker and nonselective beta-blocker and its hemodynamic effects include decreasing systemic vascular resistance, mean arterial pressure, and heart rate, accompanied by a slight decrease or minimal change in cardiac output.

» Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

» Continue until the BP is controlled on oral agents.

» If there is other end-organ involvement such as aortic dissection, acute kidney injury, or acute myocardial infarction, or the patient is to receive thrombolysis, the target SBP should be below 185 mmHg and diastolic BP should be below 110 mmHg. The BP should be maintained below 185/105 mmHg for at least 24 hours after initiating intravenous thrombolysis.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

1st **nicardipine or clevidipine**

Primary options

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

OR

» clevidipine: 1-2 mg/hour intravenously initially, dose may be doubled every 90 seconds initially until blood pressure reaches

MANAGEMENT

Acute

target, usual dose 4-6 mg/hour, maximum 32 mg/hour (maximum duration 72 hours)

» If the SBP is above 220 mmHg or the diastolic BP is between 121-140 mmHg, nicardipine or clevidipine can be used to achieve a 10% to 15% reduction in BP within 24 hours.[39] [41] [42] [93] [94]

» Nicardipine and clevidipine are dihydropyridine calcium-channel blockers, which increase stroke volume and have strong cerebral and coronary vasodilatory activity.

» Nicardipine onset of action: 5-10 minutes. Duration of action: 2-4 hours.

» Clevidipine onset of action: 2-4 minutes. Duration of action: 5-15 minutes.

» Continue until the BP is controlled on oral agents.

» If there is other end-organ involvement such as aortic dissection, acute kidney injury, or acute myocardial infarction, or the patient is to receive thrombolysis, the target SBP should be below 185 mmHg and diastolic BP should be below 110 mmHg. The BP should be maintained below 185/105 mmHg for at least 24 hours after initiating intravenous thrombolysis.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

DBP >140 mmHg **1st** **nitroprusside**

Primary options

» nitroprusside: 0.3 to 0.5 micrograms/kg/minute intravenously initially, increase by 0.5 micrograms/kg/minute increments according to response, maximum 10 micrograms/kg/minute

» If the diastolic BP is above 140 mmHg, nitroprusside may be used to achieve a 10% to 15% reduction over 24 hours.[39] [41] [95]

» Nitroprusside acts as a potent arterial and venous vasodilator, thereby reducing afterload and preload. Its hemodynamic effects are to decrease mean arterial pressure, with a modest increase or no change in cardiac output.

Acute

» Onset of action: immediate. Duration of action: 3-5 minutes.

» Patients should be monitored by drawing thiocyanate levels after 48 hours of therapy (levels maintained <12 mg/dL). The maximum dose should be delivered for less than 10 minutes to decrease the chance of cyanide toxicity.

» Continue until the BP is controlled on oral agents.

» If there is other end-organ involvement such as aortic dissection, acute kidney injury, or acute myocardial infarction, or the patient is to receive thrombolysis, the target SBP should be below 185 mmHg and diastolic BP should be below 110 mmHg. The BP should be maintained below 185/105 mmHg for at least 24 hours after initiating intravenous thrombolysis.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arrterial lines are preferred for the use of intravenous antihypertensive medications.[7]

myocardial ischemia/infarction

1st **esmolol and nitroglycerin**

Primary options

» esmolol: 50-100 micrograms/kg/minute intravenously
-and-
» nitroglycerin: 5-100 micrograms/minute intravenously

» Esmolol is a selective beta-blocker that acts to reduce the heart rate.

» Contraindications to beta-blockers include moderate-to-severe left ventricular failure with pulmonary edema, bradycardia, hypotension, poor peripheral perfusion, second- or third-degree heart block, and reactive airway disease.[1]

» Esmolol onset of action: 1-5 minutes. Duration of action: 5 minutes.

» Nitroglycerin acts as a peripheral vasodilator, with greater action on the venous vessels than on arterial vessels. It causes a decrease

Acute

in preload and cardiac output and increases coronary blood flow. Onset of action: immediate. Duration of action: 3-5 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Continue until the BP is controlled on oral agents.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

2nd **labetalol and nitroglycerin**

Primary options

» labetalol: 20 mg intravenously every 10 minutes according to response, maximum 300 mg total dose; or 0.5 to 2 mg/minute intravenous infusion
-and-
» nitroglycerin: 5-100 micrograms/minute intravenously

» Labetalol is an alpha-1-blocker and nonselective beta-blocker, which decreases systemic vascular resistance, mean arterial pressure, and heart rate, and causes a decrease or no change in cardiac output. Contraindications to beta-blockers include moderate-to-severe left ventricular failure with pulmonary edema, bradycardia, hypotension, poor peripheral perfusion, second-or third-degree heart block, and reactive airway disease.[1]

» Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

Acute

» Nitroglycerin acts as a peripheral vasodilator, with greater action on the venous vessels than on arterial vessels. It causes a decrease in preload and cardiac output and increases coronary blood flow. Onset of action: immediate. Duration of action: 3-5 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Continue until the BP is controlled on oral agents.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

3rd **nitroprusside**

Primary options

» nitroprusside: 0.3 to 0.5 micrograms/kg/minute intravenously initially, increase by 0.5 micrograms/kg/minute increments according to response, maximum 10 micrograms/kg/minute

» Nitroprusside acts as a potent arterial and venous vasodilator, thereby reducing afterload and preload. Its hemodynamic effects are to decrease mean arterial pressure, with a modest increase or no change in cardiac output.

» Onset of action: immediate. Duration of action: 3-5 minutes.

» Patients should be monitored by drawing thiocyanate levels after 48 hours of therapy

Acute

(levels maintained <12 mg/dL). The maximum dose should be delivered for less than 10 minutes to decrease the chance of cyanide toxicity.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Continue until the BP is controlled on oral agents.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

left ventricular failure and/or pulmonary edema

1st **nitroglycerin + furosemide**

Primary options

» nitroglycerin: 5-100 micrograms/minute intravenously
-and-
» furosemide: 40-80 mg intravenously initially, increase according to response

» Nitroglycerin acts as a peripheral vasodilator, with greater action on the venous vessels than on arterial vessels.

» It causes a decrease in preload and cardiac output and increases coronary blood flow.

» Onset of action: immediate. Duration of action: 3-5 minutes.

» Continue until the blood pressure (BP) is controlled on oral agents.

Acute

» If the patient is not already on a loop diuretic, one should be started (e.g., furosemide).

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

1st **clevidipine + furosemide**

Primary options

» clevidipine: 1-2 mg/hour intravenously initially, dose may be doubled every 90 seconds initially until blood pressure reaches target, usual dose 4-6 mg/hour, maximum 32 mg/hour (maximum duration 72 hours)
-and-
» furosemide: 40-80 mg intravenously initially, increase according to response

» Clevidipine is a dihydropyridine calcium-channel blocker, which increases stroke volume and has strong cerebral and coronary vasodilatory activity.

» Onset of action: 2-4 minutes. Duration of action: 5-15 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then,

Acute

if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Continue until the BP is controlled on oral agents.

» If the patient is not already on a loop diuretic, one should be started (e.g., furosemide).

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

2nd **nitroprusside + furosemide**

Primary options

» nitroprusside: 0.3 to 0.5 micrograms/kg/minute intravenously initially, increase by 0.5 micrograms/kg/minute increments according to response, maximum 10 micrograms/kg/minute
-and-
» furosemide: 40-80 mg intravenously initially, increase according to response

» Nitroprusside acts as a potent arterial and venous vasodilator, thereby reducing afterload and preload. Its hemodynamic effects are to decrease mean arterial pressure, with a modest increase or no change in cardiac output.

» Patients should be monitored by drawing thiocyanate levels after 48 hours of therapy (levels maintained <12 mg/dL). The maximum dose should be delivered for less than 10 minutes to decrease the chance of cyanide toxicity.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then,

Acute

if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Continue until the BP is controlled on oral agents.

» If the patient is not already on a loop diuretic, one should be started (e.g., furosemide).

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

aortic dissection

1st **labetalol or esmolol**

Primary options

» labetalol: 20 mg intravenously every 10 minutes according to response, maximum 300 mg total dose; or 0.5 to 2 mg/minute intravenous infusion

OR

» esmolol: 50-100 micrograms/kg/minute intravenously

» Medical therapy of aortic dissection involves lowering the blood pressure (BP) and decreasing the velocity of left ventricular contraction, which decreases aortic shear stress and minimizes the tendency for propagation of the dissection.

» SBP should be reduced to <120 mmHg in the first 20 minutes or as tolerated by the patient.[1] [75]

» Labetalol is an alpha-1-blocker and nonselective beta-blocker, which decreases systemic vascular resistance, mean arterial pressure, and heart rate, and causes a decrease or no change in cardiac output. Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

Acute

» The mechanism of action of esmolol is as a selective beta-blocker, producing a decrease in heart rate. Onset of action: 1-5 minutes. Duration of action: 5 minutes.

» The dose should be adjusted to maintain the BP in the desired range. This should be continued until the patient has undergone surgical repair/evaluation and is stable on oral therapy.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

adjunct **nitroprusside or nicardipine**

Treatment recommended for SOME patients in selected patient group

Primary options

» nitroprusside: 0.3 to 0.5 micrograms/kg/minute intravenously initially, increase by 0.5 micrograms/kg/minute increments according to response, maximum 10 micrograms/kg/minute

OR

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

» If there is no significant improvement with beta-blocker monotherapy, nitroprusside or nicardipine can be added to the beta-blocker.[39] [41] [42] [97]

» Nitroprusside acts as a potent arterial and venous vasodilator, thereby reducing afterload and preload. Its hemodynamic effects are to decrease mean arterial pressure, with a modest increase or no change in cardiac output.

» Nitroprusside onset of action: immediate. Duration of action: 3-5 minutes.

» Patients should be monitored by drawing thiocyanate levels after 48 hours of therapy (levels maintained <12 mg/dL). The maximum dose should be delivered for less than 10 minutes to decrease the chance of cyanide toxicity.

Acute

» Alternatively, nicardipine can be used.[1] Nicardipine is a dihydropyridine calcium-channel blocker, which increases stroke volume and has strong cerebral and coronary vasodilatory activity.

» Nicardipine onset of action: 5-10 minutes. Nicardipine duration of action: 2-4 hours.

» Nitroprusside or nicardipine must be administered after a beta-blocker, as nitroprusside-induced or calcium-channel blocker-induced vasodilation would otherwise induce a compensatory tachycardia and worsen shear stress.[1]

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

acute kidney injury

1st **fenoldopam**

Primary options

» fenoldopam: 0.1 to 0.3 micrograms/kg/minute intravenously initially, increase by 0.05 to 0.1 micrograms/kg/minute increments every 15 minutes according to response, maximum 1.6 micrograms/kg/minute

» Fenoldopam is useful in renal insufficiency because it causes an increase in blood flow to the kidneys.

» It acts as a selective peripheral dopamine-1-receptor agonist with arterial vasodilator effects. Its hemodynamic effects are a decrease in afterload and an increase in renal perfusion.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

Acute

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

1st **nicardipine or clevidipine**

Primary options

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

OR

» clevidipine: 1-2 mg/hour intravenously initially, dose may be doubled every 90 seconds initially until blood pressure reaches target, usual dose 4-6 mg/hour, maximum 32 mg/hour (maximum duration 72 hours)

» Nicardipine and clevidipine are dihydropyridine calcium-channel blockers, which increase stroke volume and have strong cerebral and coronary vasodilatory activity.

» Nicardipine onset of action: 5-10 minutes. Duration of action: 2-4 hours.

» Clevidipine onset of action: 2-4 minutes. Duration of action: 5-15 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

Acute

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

hyperadrenergic state

sympathomimetic drug use

1st

benzodiazepine

Primary options

» lorazepam: 1 mg intravenous bolus initially, repeated every 10-15 minutes according to response, maximum 8 mg

OR

» diazepam: 5 mg intravenous bolus initially, repeated every 5-10 minutes according to response, maximum 50 mg

» Sympathomimetic drug use includes cocaine, amphetamines, phenylpropanolamine, phencyclidine, or the combination of a monoamine oxidase inhibitor with foods rich in tyramine.

» If the patient is agitated, benzodiazepines can be given first.[18] [54] Benzodiazepines reduce agitation and prevent neurologic complications such as seizures by their action on gamma-aminobutyric acid receptors.[18]

» Lorazepam should be used with caution in patients with renal or hepatic impairment, myasthenia gravis, organic brain syndrome, or Parkinson disease. Excess central nervous system or respiratory depression can occur with higher doses, and should be watched for.

» Antihypertensive agents can be given if the blood pressure (BP) response to benzodiazepines is inadequate.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial BP by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

Acute

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

2nd **phentolamine**

Primary options

» phentolamine: 5-15 mg intravenous bolus

» Phentolamine acts to block alpha-adrenoceptors. Its main hemodynamic effects are to increase heart rate and contractility.

» Onset of action: 1-2 minutes. Duration of action: 3-10 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

2nd **nicardipine or clevidipine**

Primary options

Acute

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

OR

» clevidipine: 1-2 mg/hour intravenously initially, dose may be doubled every 90 seconds initially until blood pressure reaches target, usual dose 4-6 mg/hour, maximum 32 mg/hour (maximum duration 72 hours)

» Nicardipine and clevidipine are dihydropyridine calcium-channel blockers, which increase stroke volume and have strong cerebral and coronary vasodilatory activity.

» Nicardipine onset of action: 5-10 minutes. Duration of action: 2-4 hours.

» Clevidipine onset of action: 2-4 minutes. Duration of action: 5-15 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74]

no sympathomimetic drug use — **1st** — **phentolamine**

Primary options

» phentolamine: 5-15 mg intravenous bolus

» Causes of hyperadrenergic states include pheochromocytoma and abrupt discontinuation of a short-acting sympathetic blocker.

Acute

» Phentolamine acts to block alpha-adrenoceptors. Its main hemodynamic effects are to increase heart rate and contractility.

» Onset of action: 1-2 minutes. Duration of action: 3-10 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours. In patients with pheochromocytoma crisis, SBP should be reduced to <140 mmHg in the first hour.[1]

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [7][74] Arterial lines are preferred for the use of intravenous antihypertensive medications.[7]

adjunct **labetalol**

Treatment recommended for SOME patients in selected patient group

Primary options

» labetalol: 20 mg intravenously every 10 minutes according to response, maximum 300 mg total dose; or 0.5 to 2 mg/minute intravenous infusion

» Labetalol is an alpha-1-blocker and nonselective beta-blocker, which decreases systemic vascular resistance, mean arterial pressure, and heart rate, and causes a decrease or no change in cardiac output. Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

» The administration of a beta-blocker alone is contraindicated, since inhibition of beta-adrenoceptor-induced vasodilation results in

Acute

unopposed alpha-adrenergic vasoconstriction and a further rise in BP.

1st **nicardipine or clevidipine**

Primary options

» nicardipine: 5 mg/hour intravenously initially, increase by 2.5 mg/hour increments every 15 minutes according to response, maximum 15 mg/hour

OR

» clevidipine: 1-2 mg/hour intravenously initially, dose may be doubled every 90 seconds initially until blood pressure reaches target, usual dose 4-6 mg/hour, maximum 32 mg/hour (maximum duration 72 hours)

» Nicardipine and clevidipine are dihydropyridine calcium-channel blockers, which increase stroke volume and have strong cerebral and coronary vasodilatory activity.

» Nicardipine onset of action: 5-10 minutes. Duration of action: 2-4 hours.

» Clevidipine onset of action: 2-4 minutes. Duration of action: 5-15 minutes.

» The American College of Cardiology/American Heart Association Task Force on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure states that the initial goal of therapy in hypertensive emergencies is to reduce mean arterial blood pressure (BP) by no more than 25% (within minutes to 1 hour), then, if stable, to 160 mmHg systolic and 100-110 mmHg diastolic within the next 2-6 hours.[1]

» Excessive falls in pressure that may precipitate renal, cerebral, or coronary ischemia should be avoided.[1] [57]

» If the initial level of reduced BP is well tolerated and the patient is clinically stable, further gradual reductions toward a normal BP can be implemented over the next 24-48 hours.

severe hypertension in pregnancy (preeclampsia and eclampsia)

1st **labetalol or hydralazine or nifedipine**

Primary options

» labetalol: 10-20 mg intravenously initially, followed by 20-80 mg every 10-30 minutes according to response, maximum 300 mg/

MANAGEMENT

Acute

total dose; or 1-2 mg/minute intravenous infusion

OR

» nifedipine: 10-20 mg orally (immediate-release) initially, repeat in 20 minutes if needed, followed by 10-20 mg every 2-6 hours according to response, maximum 180 mg/day

OR

» hydralazine: 5 mg intravenously initially, followed by 5-10 mg every 20-40 minutes according to response, maximum 20 mg/total dose; or 0.5 to 10 mg/hour intravenous infusion

» The American College of Obstetricians and Gynecologists (ACOG) recommends antihypertensive therapy for women with preeclampsia and a sustained systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg.[48] However, thresholds for treatment vary internationally, with lower thresholds recommended by a number of societies.[100] The UK National Institute for Health and Care Excellence recommends treatment if BP remains above 140/90 mmHg.[61]

» Although some of the available literature suggests that antihypertensive agents should be administered within 30-60 minutes, it is recommended that antihypertensive therapy begin as soon as reasonably possible after the criteria for acute onset severe hypertension are met.[48] It should be noted, however, that there are no trials supporting these suggested thresholds, and treatments should be tailored to individual patient circumstances. Specialist advice should be sought.

» For acute-onset, severe hypertension managed in a critical care setting, intravenous labetalol, intravenous hydralazine, or oral nifedipine can be used first line.

» Labetalol is usually considered the antihypertensive of choice, and is effective as monotherapy in 80% of pregnant women.[48][61][98][99] It seems to be safe and effective in pregnant women for the management of preeclampsia; however, it should be avoided in women with asthma or any other contraindication to its use.[48]

Acute

» Labetalol acts as an alpha-1-blocker and nonselective beta-blocker and its hemodynamic effects include decreasing systemic vascular resistance, mean arterial pressure, and heart rate, accompanied by a slight decrease or minimal change in cardiac output. Onset of action: 5-10 minutes. Duration of action: 3-8 hours.

» Hydralazine is an arterial vasodilator with minimal effects on the fetus. Onset of action: 10-20 minutes. Duration of action: 3-8 hours. Its main hemodynamic effects are a decrease in systemic vascular resistance, an increase in heart rate, and an increase in intracranial pressure. Although intravenous hydralazine is still widely used, particularly in North America, it can produce an acute fall in BP. The consequences of this are mostly related to maternal hypotension, including a greater risk of cesarean section, more frequent placental abruption, more maternal oliguria, and fetal tachycardia, suggesting the need for close monitoring of maternal BP and fetal wellbeing during its use.[63] UK guidelines recommend to consider volume expansion with crystalloid fluid before or at the same time as the first dose of intravenous hydralazine in the prenatal period.[61]

» Immediate-release oral nifedipine may also be considered first-line therapy, particularly when intravenous access is not available.[48] Nifedipine is a dihydropyridine calcium-channel blocker, which increases stroke volume and has strong cerebral and coronary vasodilatory activity. Onset of action: 30-45 minutes. Duration of action: 4-6 hours.

» Therapy should be continued until the fetus has been delivered and the patient is stable on oral therapy.

» Patients with hypertensive emergencies should be admitted to an intensive care unit for continuous monitoring of blood pressure and target organ damage and for parenteral administration of appropriate therapeutic agent(s).[1] [74] See Preeclampsia (Management approach) .

Primary prevention

The mainstay of primary prevention is appropriate screening and treatment of essential hypertension.

In the US, around 25% of all hypertensive individuals are unaware of their illness, 35% are not being treated, and 52% of those being treated are not at goal blood pressure levels.[52] [53] See Essential hypertension (Prevention) .

The American College of Obstetricians and Gynecologists recommends low-dose aspirin for preeclampsia prophylaxis. This should be initiated between 12 weeks and 28 weeks of gestation (optimally before 16 weeks of gestation) and continued until delivery.[48]

- Any of the high-risk factors for preeclampsia: previous pregnancy with preeclampsia, multifetal gestation, renal disease, autoimmune disease, type 1 or type 2 diabetes mellitus, and chronic hypertension.
- More than one of the moderate-risk factors: first pregnancy, maternal age ≥35 years, body mass index >30, family history of preeclampsia, sociodemographic characteristics, and personal history factors.

See Preeclampsia (Prevention) .

Secondary prevention

Major lifestyle modifications shown to lower blood pressure include the Dietary Approaches to Stop Hypertension (DASH) eating plan, dietary sodium reduction, weight reduction in overweight patients, physical activity, and moderation of alcohol consumption.[38] [103] For women with severe preeclampsia, low-dose aspirin (starting at 12-14 weeks' gestation) is recommended in subsequent pregnancies. See Preeclampsia (Prevention) .

Patient discussions

Patients should be reminded of the importance of taking medications as directed and not missing doses. Patients should be advised to call their doctor or an ambulance immediately if they experience any dizziness, loss of sensation or mobility, blurred vision, chest pain, shortness of breath, or any other relevant symptoms.

FOLLOW UP

Monitoring

Monitoring

The patient should return for a follow-up visit and blood pressure (BP) check within 1 week of discharge. During the follow-up visit, BP should be checked by a medical professional in both arms and with the appropriate cuff size. The target BP should be below 130/80 mmHg. Regular and frequent follow-up (monthly) is recommended until target BP and ideally regression of hypertension-mediated organ damage has been achieved.[54] Patients should return for follow-up visits once a month, or more frequently, until the target BP is achieved. Once the target BP is achieved, the patient should be monitored every 3 to 6 months (or more frequently based on comorbidities). Serum potassium and creatinine should be measured twice a year. For patients with preeclampsia, see Pre-eclampsia (Monitoring) .

Complications

Complications	Timeframe	Likelihood
cardiac impairment	**variable**	**high**
Myocardial damage and subsequent heart failure is a frequent complication and cause of death in hypertensive emergency.[101] [102]		
neurologic deficit	**variable**	**medium**
Permanent neurologic compromise may occur after stroke, hemorrhage, or hypertensive encephalopathy and is a frequent cause of death.[101] [102]		
acute kidney injury	**variable**	**medium**
Acute kidney injury is both a frequent cause and complication of hypertensive emergency.[101] [102]		

Prognosis

Without therapy, the prognosis of hypertensive emergencies is poor with 1-year survival rates of 10% to 20%.[1] However, current antihypertensive therapy has greatly improved survival, with 5-year survival rates around 70% in patients who receive appropriate treatment. The presence of acute kidney injury upon diagnosis of hypertensive emergency increases the mortality rate.[101] [102]

Preeclampsia is a self-limiting condition of pregnancy that usually resolves once the placenta has been delivered, although it may persist for a few days post delivery. There are few long-term sequelae; however, there are some long-term disease associations. See Preeclampsia (Prognosis) .

Diagnostic guidelines

International

Headaches in pregnancy and postpartum: clinical practice guideline (https://www.acog.org/clinical/clinical-guidance/clinical-practice-guideline) [55]

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Prevention, detection, evaluation, and management of high blood pressure in adults (https://www.acc.org/guidelines) [1]

Published by: American College of Cardiology; American Heart Association **Last published:** 2017

Global hypertension practice guidelines (https://ish-world.com/ish-global-hypertension-practice-guidelines) [54]

Published by: International Society of Hypertension **Last published:** 2020

Hypertension in adults: diagnosis and management (https://www.nice.org.uk/guidance/ng136) [60]

Published by: National Institute for Health and Care Excellence **Last published:** 2023

Management of hypertensive crisis: position document (https://bihsoc.org/guidelines) [18]

Published by: British and Irish Hypertension Society **Last published:** 2022

Treatment guidelines

International

The management of elevated blood pressure in the acute care setting (https://professional.heart.org/en/guidelines-and-statements) [7]

Published by: American Heart Association **Last published:** 2024

Management of patients with spontaneous intracerebral hemorrhage (https://professional.heart.org/en/guidelines-and-statements) [76]

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Headaches in pregnancy and postpartum: clinical practice guideline (https://www.acog.org/clinical/clinical-guidance/clinical-practice-guideline) [55]

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Hypertension in adults: diagnosis and management (https://www.nice.org.uk/guidance/ng136) [60]

Published by: National Institute for Health and Care Excellence **Last published:** 2023

Management of hypertensive crisis: position document (https://bihsoc.org/guidelines) [18]

Published by: British and Irish Hypertension Society **Last published:** 2022

Key articles

- Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2018 May 15;71(19):e127-248. Full text (https://www.sciencedirect.com/science/article/pii/S0735109717415191?via%3Dihub) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29146535?tool=bestpractice.bmj.com)

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- Mancia G, Kreutz R, Brunström M, et al. 2023 ESH Guidelines for the management of arterial hypertension the Task Force for the management of arterial hypertension of the European Society of Hypertension: endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens. 2023;41(12):1874-2071. Full text (https://journals.lww.com/jhypertension/fulltext/2023/12000/2023_esh_guidelines_for_the_management_of_arterial.2.aspx) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/37345492?tool=bestpractice.bmj.com)

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Images

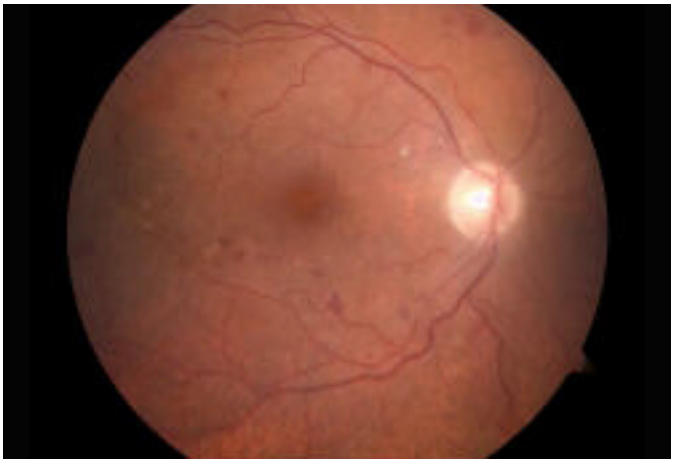

Figure 1: Fundus photograph of the right eye with multiple dot-blot hemorrhages typical of hypertensive retinopathy

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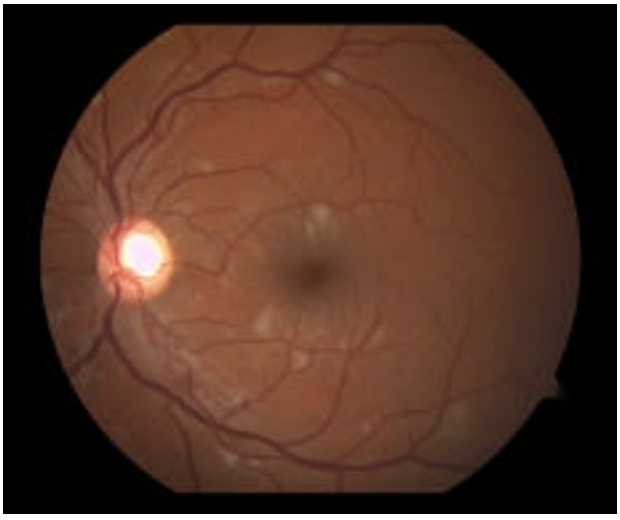

Figure 2: Fundus photograph of the left eye with multiple cotton-wool spots typical of hypertensive retinopathy

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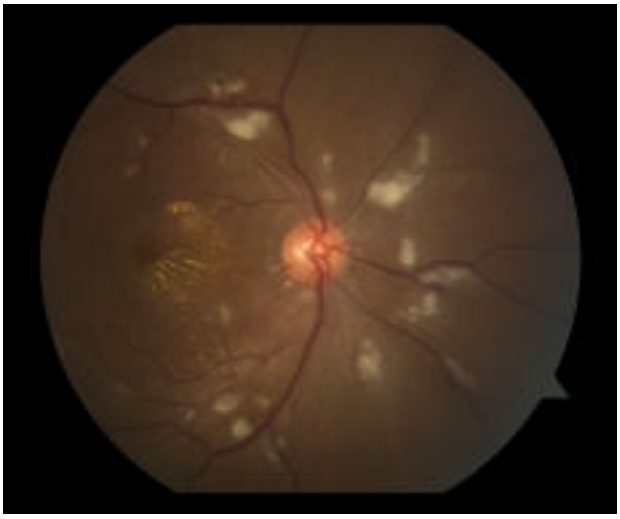

Figure 3: Fundus photograph of the right eye centered on the optic nerve, showing multiple cotton-wool spots and macular exudates in a radiating star configuration around the fovea

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Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

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