

# Hypertensive Emergency

Blood pressure spikes so high that organs start getting damaged in real time.

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## THE STORY IN ONE SENTENCE

Blood pressure spikes so high that organs start getting damaged in real time.

“A with high blood pressure (hypertensive) emergency is dangerously high blood pressure — usually over 180/120 — that is actively hurting the brain, heart, kidneys, eyes, or blood vessels right now. The goal is to lower the pressure carefully with IV medicine while hunting for the root cause. Always watch the trend, because a stable vital sign can hide a patient sliding toward organ failure.”

## Normal anatomy — the body's job

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Your body keeps your brain, heart, kidneys, and eyes safe by autoregulating blood flow. Small arteries squeeze or relax to hold flow steady even when pressure goes up or down. After months or years of high blood pressure, this system resets to a higher baseline, so organs still work but the safety margin is thin. Keep that picture in your head, because when pressure suddenly spikes way beyond what even the reset system can handle, every weird finding you see is the body's alarm that autoregulation just failed and organs are getting crushed.

## BODY'S JOB

Small arteries (arterioles) have smooth muscle in their walls that can squeeze tighter (constrict) or relax wider (dilate) in response to signals from the endothelium (the inner lining of blood vessels), the sympathetic nervous system (the body's fight-or-flight wiring), and local chemical messengers like nitric oxide (a gas that relaxes vessels) and endothelin (a peptide that tightens them). This autoregulation keeps blood flow to the brain, heart, and kidneys constant across a wide range of pressures — so even if your systolic pressure swings from 100 to 150, flow stays steady. Chronic high blood pressure shifts the autoregulation curve rightward: the body recalibrates and defends a higher pressure as normal. That protects organs in the short run but leaves them vulnerable if pressure spikes suddenly or climbs so high that even the shifted curve cannot compensate.

1. Pressure in a large artery rises from the heart's pump or from resistance downstream.

2. Mechanoreceptors and chemoreceptors in the artery wall and the endothelium sense the stretch and release nitric oxide (a relaxing signal) and prostacyclin (another vasodilator).
3. Smooth muscle in the arteriole wall relaxes, the vessel widens, and resistance drops — this keeps flow and pressure inside the organ steady.
4. If pressure drops, the opposite happens: the sympathetic nervous system releases norepinephrine (the fight-or-flight hormone), the endothelium releases endothelin, vessels squeeze tighter, and resistance rises to keep flow steady.
5. Chronic high pressure remodels the vessel wall — smooth muscle gets thicker, the wall gets stiffer, and the autoregulation curve shifts right, so the new normal defended pressure is higher.
6. As long as pressure stays within the shifted autoregulation range (often 120–180 systolic in someone with chronic high blood pressure (hypertension)), organs get enough blood and you see no symptoms.

## What breaks

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When something suddenly pushes blood pressure far above the body's reset safety range — or when pressure climbs so high that even years of adaptation cannot hold it — the small arteries lose the fight. Their walls stretch, leak, tear, or spasm. Organs that depend on steady flow (brain, heart, kidneys, eyes) start dying in real time. That is the one upstream break that explains every weird finding you will see.

**CLINICAL TERM**

With high blood pressure (Hypertensive) emergency occurs when systolic blood pressure exceeds 180 mmHg or diastolic exceeds 120 mmHg (the exact threshold is less important than the presence of acute target-organ damage) and autoregulation fails. Common triggers: medication nonadherence (skipping antihypertensives, especially clonidine or beta-blockers, causes rebound surge), sympathomimetic drugs (cocaine, amphetamines, bath salts), pheochromocytoma (adrenal tumor secreting catecholamines), acute glomerulonephritis or renal-artery narrowing (stenosis) (kidney causes that spike renin and angiotensin), preeclampsia or eclampsia (pregnancy-induced endothelial dysfunction), thyrotoxicosis, or acute withdrawal from alcohol or sedatives. The break: pressure rises so high and fast that arteriolar autoregulation is overwhelmed — endothelial cells swell and die (endothelial injury), the blood-brain barrier breaks down (vasogenic swelling (edema)), fibrinoid tissue death (necrosis) occurs in vessel walls (protein and red cells leak into the wall, triggering clotting and starved blood flow (ischemia)), and microthrombi form. The downstream cascade: with high blood pressure encephalopathy (brain swelling, confusion, seizures), intracerebral or subarachnoid bleeding (hemorrhage), blood-starved (ischemic) stroke (paradoxically, because vessels spasm or clot), acute coronary syndrome or heart attack (myocardial infarction) (coronary demand exceeds supply), acute left-heart failure with pulmonary edema (fluid flooding the lungs) (afterload is too high for the left ventricle to pump against), acute aortic dissection (the inner layer of the aorta tears and blood tracks between layers), acute kidney injury (glomerular capillaries clot or burst, GFR drops, creatinine rises), and with high blood pressure retinopathy grade III or IV (flame hemorrhages, cotton-wool spots, macular star, or swelling of the optic nerve (papilledema) from optic-nerve swelling). The bedside signals: severe headache, blurred vision or sudden vision loss, chest pain (pressure-like or tearing), trouble breathing (dyspnea) (shortness of breath from pulmonary edema), altered mental status or focal neurologic deficit (one-sided weakness, slurred speech), low urine output (oliguria) or no urine output (anuria) (little or no urine) with rising creatinine, or blood in the urine (hematuria) (blood in urine from glomerular rupture).

## How the body compensates

The body tries to defend organ blood flow by shifting autoregulation upward over weeks to months, so chronic high pressure is tolerated without symptoms. In a with high blood pressure (hypertensive) emergency, that defense is overrun — pressure spikes so high or so fast that compensation fails and organs start dying. Vital signs can still look stable (alert, making urine, normal oxygen saturation) for hours while micro-damage piles up invisibly. The dangerous middle is missing the slide toward irreversible injury. The crash point to watch for is blood-starved (ischemic) stroke or heart attack (myocardial infarction) from dropping pressure too

fast (if you cut mean arterial pressure by more than 25% in the first hour, autoregulation cannot keep up and flow to the brain or heart falls below the blood-starved threshold), or continued bleeding (hemorrhage), pulmonary edema (fluid flooding the lungs), or kidney failure from dropping pressure too slowly. Each target organ has its own blood-pressure goal and time window — there is no one-size-fits-all number.

1. {'what': 'Chronic adaptation shifts the autoregulation curve right', 'how': 'Over weeks to months of high blood pressure, arterioles remodel — smooth muscle hypertrophies, the media (middle layer) thickens, and the endothelium recalibrates. The lower limit of autoregulation (the pressure below which flow starts to drop) moves from around 60 mmHg mean arterial pressure up to around 100–120 mmHg in someone with long-standing high blood pressure (hypertension). This lets organs tolerate pressures of 160–180 systolic without symptoms.', 'cost': 'The safety margin shrinks. If pressure then drops suddenly (from overzealous treatment or hypovolemia), flow crashes and the brain or kidneys can a patch of dead tissue (infarct) even at pressures that would be normal for someone else.'}
2. {'what': 'Sympathetic surge tries to maintain cardiac output and blood flow (perfusion)', 'how': 'Baroreceptors in the carotid sinus and aortic arch sense high pressure and should tell the brainstem to dial down sympathetic tone — but in a with high blood pressure (hypertensive) emergency, this reflex is often blunted (from chronic resetting or direct brainstem starved blood flow (ischemia)). Instead, circulating catecholamines (epinephrine and norepinephrine from the adrenal medulla and sympathetic nerves) stay high, heart rate rises, and inotropy (squeeze strength) increases to keep cardiac output up.', 'cost': 'Myocardial oxygen demand spikes. A fast heart rate (Tachycardia) shortens diastole (the phase when coronaries fill), so the heart works harder but gets less fuel — this sets up acute coronary syndrome.'}
3. {'what': 'Renin–angiotensin–aldosterone system (RAAS) revs up', 'how': 'Low renal blood flow (perfusion) pressure (sensed by juxtaglomerular cells in the kidney) or high sympathetic tone triggers renin release. Renin cleaves angiotensinogen (from the liver) into angiotensin I. ACE (angiotensin-converting enzyme, mostly in the lungs) chops that into angiotensin II, a powerful vasoconstrictor that also tells the adrenal cortex to release aldosterone (a hormone that makes the kidney hold onto sodium and water). Volume and pressure rise further.', 'cost': 'This is helpful if pressure is truly low, but in a with high blood pressure (hypertensive) emergency it becomes a vicious cycle — more constriction, more volume, higher pressure, more end-organ damage.'}

4. {'what': 'Cerebral autoregulation attempts to hold brain blood flow constant', 'how': 'When mean arterial pressure climbs above the upper autoregulation limit (around 150–160 mmHg in most people, higher in chronic high blood pressure (hypertension)), cerebral arterioles are maximally constricted and cannot squeeze any tighter. Pressure forces blood through, the blood–brain barrier leaks, and vasogenic swelling (edema) (brain swelling from leaked fluid) begins. You see this as with high blood pressure (hypertensive) encephalopathy: headache, confusion, seizures, vision changes, and nausea.', 'cost': 'If you drop pressure too fast, you cross below the shifted lower autoregulation limit and cause blood-starved (ischemic) stroke — the brain cannot dilate vessels quickly enough to maintain flow. The safe drop is no more than 25% reduction in mean arterial pressure in the first hour, then cautious further lowering over 24–48 hours.'}
5. {'what': 'Natriuretic peptides try to offload volume', 'how': 'Atrial stretch (from volume overload and high filling pressures) triggers release of ANP (atrial natriuretic peptide) and BNP (B-type natriuretic peptide, mostly from the ventricles). These hormones tell the kidney to dump sodium and water, dilate arterioles, and blunt aldosterone and ADH (antidiuretic hormone). In theory, this should lower volume and pressure.', 'cost': 'In a with high blood pressure (hypertensive) emergency, especially with acute kidney injury, the kidney cannot respond well — GFR is falling, so sodium and water cannot be excreted effectively. Pulmonary edema (fluid flooding the lungs) worsens despite high natriuretic-peptide levels.'}
6. {'what': 'The body may look stable even as it slides toward the cliff', 'how': 'Blood pressure, heart rate, oxygen saturation, and even mental status can appear normal or only mildly abnormal for the first few hours. Creatinine takes 24–48 hours to rise after kidney injury begins (it is a lagging marker). Troponin (heart muscle damage marker) takes 3–6 hours to appear. The brain can swell silently until intracranial pressure suddenly herniates the brainstem. A patient who is alert, talking, and making urine can still be minutes away from a catastrophic bleed or a patch of dead tissue (infarct).', 'lesson': '"Do not wait for vital signs to crash. Look at trends: Is creatinine climbing? Is the patient's headache worsening? Is urine output dropping? Is the chest X-ray showing more fluid? A stable snapshot hides a failing movie. Start treatment based on the BP reading plus any sign of target-organ damage, not on how comfortable the patient looks."}'}

7. {'what': 'Watch for the two crash points: over-correction and under-treatment', 'how': 'Over-correction: dropping mean arterial pressure by more than 25% in the first hour (or dropping it below the patient's shifted autoregulation range) causes starved blood flow (ischemia) — brain (watershed-territory stroke, confusion, coma), heart (demand starved blood flow or a patch of dead tissue (infarct), ST depressions, troponin rise), or kidneys (acute tubular tissue death (necrosis), creatinine spikes further). Under-treatment: keeping pressure high out of fear lets ongoing bleeding (hemorrhage), pulmonary edema (fluid flooding the lungs), aortic dissection propagation, or glomerular clot formation (thrombosis) continue unchecked — the window to reverse damage closes within hours.', 'target': 'The goal is NOT a specific number but a safe rate of decline tailored to the injury. Acute blood-starved (ischemic) stroke: permissive high blood pressure (hypertension) unless BP >185/110 and you plan thrombolysis. Bleeding-driven (Hemorrhagic) stroke: target systolic 140 mmHg within 1 hour (INTERACT-2, ATACH-2). Aortic dissection: target systolic <120 mmHg and heart rate <60 bpm within 20 minutes. Acute pulmonary edema (fluid flooding the lungs): reduce by around 25% over 1 hour. Acute kidney injury without another acute target-organ injury: reduce by 10–20% over 1 hour, then more gradually. Preeclampsia or eclampsia: systolic <160, diastolic <110 with IV labetalol or hydralazine plus magnesium sulfate for seizure prevention (prophylaxis).'} }

## Domino effect

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1. A trigger — stopped blood-pressure pills (especially clonidine or beta-blockers), cocaine, pheochromocytoma, kidney artery narrowing (stenosis), acute glomerulonephritis, preeclampsia — causes blood pressure to spike rapidly or climb to extreme levels (systolic >180, diastolic >120). — Each cause has a mechanism. Stopping clonidine removes central alpha-2 agonism (the brake on sympathetic outflow in the brainstem), so norepinephrine floods out and pressure surges (rebound high blood pressure (hypertension)). Cocaine and amphetamines block reuptake of norepinephrine and dopamine at nerve terminals, so sympathetic tone skyrockets. Pheochromocytoma dumps catecholamines directly into the blood. Kidney causes (stenosis, glomerulonephritis) cut renal blood flow (perfusion) or damage glomeruli, spiking renin and angiotensin II. Preeclampsia injures the endothelium systemwide through placental factors like sFlt-1 (soluble fms-like tyrosine kinase, which mops up VEGF and prevents vessel repair). The common endpoint is extreme systemic vascular resistance and pressure that exceeds the body's autoregulation ceiling.

2. Autoregulation fails — small arteries cannot squeeze tight enough to protect organs from the pressure wave, and the endothelium (inner lining) starts to break down. — Normally, arterioles constrict when pressure rises, keeping flow and downstream capillary pressure steady. But there is an upper limit — mean arterial pressure around 150–160 mmHg in most people, higher in chronic high blood pressure (hypertension). Above that, shear stress tears endothelial cells off the wall, nitric oxide production stops, and vessels spasm instead of dilating. Plasma and red blood cells leak into the vessel wall (fibrinoid tissue death (necrosis)), and microthrombi form. This is the microscopic break that will show up as gross organ damage within hours.
3. The brain swells or bleeds — you see severe headache (often occipital, worst ever), blurred or lost vision, confusion, seizures, or focal weakness (hemiparesis, speech trouble). — The blood-brain barrier is made of capillary endothelial cells with tight junctions that normally keep plasma proteins and fluid out of brain tissue. When cerebral blood flow (perfusion) pressure exceeds the autoregulation ceiling, those junctions pop open — fluid floods into the white matter (vasogenic swelling (edema)), intracranial pressure rises, and you see with high blood pressure (hypertensive) encephalopathy (headache, nausea, altered mental status, seizures, vision changes). At the same time, arterioles can rupture outright (intracerebral bleeding (hemorrhage), often in the basal ganglia, thalamus, pons, or cerebellum) or spasm so hard they clot off (ischemic stroke) — both can happen in the same patient. On exam, you may see swelling of the optic nerve (papilledema) (optic disc swelling from high intracranial pressure) or focal neurologic deficits. Head CT will show swelling (hypodense white matter, sulci effaced) or blood (hyperdense on non-contrast CT).
4. The heart fails or infarcts — you see crushing chest pain, severe shortness of breath, pink frothy coughed-up mucus (sputum) (from pulmonary edema (fluid flooding the lungs)), or a new heart murmur (acute mitral regurgitation from papillary-muscle starved blood flow (ischemia)). — The left ventricle suddenly faces massive afterload (the resistance it must pump against). Stroke work (pressure  $\times$  volume) spikes, myocardial oxygen demand soars, but diastolic filling time (when coronaries perfuse) shortens because heart rate is high. The mismatch causes demand starved blood flow (ischemia) or outright heart attack (myocardial infarction) (you will see ST elevations or depressions, troponin rise). Meanwhile, the ventricle cannot keep up with the load — end-diastolic pressure climbs, backs up into the left atrium and pulmonary veins, and pulmonary capillary pressure exceeds oncotic pressure (around 18–20 mmHg). Fluid leaks into alveoli (acute cardiogenic pulmonary edema (fluid flooding the lungs)). Chest X-ray shows perihilar fluffy infiltrates (bat-wing pattern), Kerley B lines (interstitial swelling (edema)), and cephalization (upper-lobe vessels are larger than lower because pressure redistributed blood upward). The patient gasps, sits upright, and may cough up pink foam.

5. The aorta can tear — you see sudden tearing chest pain radiating to the back, a blood-pressure difference between arms ( $>20$  mmHg), a new diastolic murmur (aortic regurgitation if the dissection involves the aortic root), or signs of end-organ starved blood flow (ischemia) (stroke, mesenteric starved blood flow, limb starved blood flow) if branch vessels are sheared off. — Acute aortic dissection is a tear in the intima (inner layer) that lets blood track into the media (middle muscular layer), creating a false lumen. High pressure is the number-one trigger — it generates shear stress on the wall, especially at the aortic root and the ligamentum arteriosum (just distal to the left subclavian artery). Stanford type A involves the ascending aorta (surgical emergency — mortality is around 1–2% per hour untreated). Type B involves the descending aorta distal to the left subclavian (managed medically unless complicated). The dissection can propagate forward (cutting off coronary or carotid flow, leading to MI or stroke) or backward (into the pericardium, causing cardiac tamponade (the heart squeezed by fluid around it)). Diagnosis: CT angiography of the chest shows the flap and false lumen. Treatment: immediate beta-blockade (to drop heart rate and  $dP/dt$ , the rate of pressure rise) then vasodilator (nicardipine or nitroprusside) to bring systolic  $<120$  mmHg, plus cardiothoracic surgery consult for type A.
6. The kidneys shut down — urine output drops (oliguria  $<400$  mL/day, no urine output (anuria)  $<50$  mL/day), creatinine rises, and you may see blood in the urine (hematuria) (tea-colored or cola-colored urine) or proteinuria. — Glomerular capillaries (the filtration units) are delicate, high-flow, low-resistance vessels. Extreme pressure causes thrombotic microangiopathy — endothelial cells swell and die, platelets clump, fibrin strands form, and red blood cells shatter as they squeeze through (you see schistocytes or helmet cells on the peripheral smear, like in destroying red blood cells (hemolytic)-uremic syndrome or thrombotic thrombocytopenic purple bleeding blotches (purpura)). GFR (glomerular filtration rate) plummets. The kidney cannot excrete creatinine, urea, potassium, or acid — you see acute kidney injury (creatinine rising  $\geq 0.3$  mg/dL in 48 hours or  $\geq 1.5\times$  baseline), high potassium (hyperkalemia) (peaked T waves on ECG, risk of fatal arrhythmia), and metabolic acidosis (acid building up in the blood) (low bicarbonate, high anion gap if severe waste buildup in the blood (uremia) is severe). Urinalysis shows blood in the urine (hematuria) (red blood cells, RBC casts from glomerular bleeding) and proteinuria. This is malignant high blood pressure (hypertension) (the old term for with high blood pressure (hypertensive) emergency with renal and retinal injury).

7. The eyes show the damage — fundoscopy (looking through the pupil with an ophthalmoscope) reveals flame hemorrhages (bleeding in the nerve-fiber layer), cotton-wool spots (fluffy white patches where the retina is blood-starved (ischemic)), a macular star (hard exudates radiating from the fovea), or swelling of the optic nerve (papilledema) (the optic disc is swollen, margins are blurred, veins are engorged). — The retina is the only place you can see arterioles and capillaries directly without cutting the body open. Changes here mirror what is happening in the brain and kidneys. Flame hemorrhages mean a retinal arteriole ruptured. Cotton-wool spots are microinfarcts — axonal starved blood flow (ischemia) causes axoplasm to pile up, looking like cotton. Swelling of the optic nerve (Papilledema) means intracranial pressure is up (from brain swelling (edema)) or the optic nerve head itself is blood-starved (ischemic). Macular hard exudates (lipid leaked from damaged capillaries) arrange in a star because of the radial anatomy of Henle's layer. This is Keith-Wagener-Barker grade III (hemorrhages and exudates without swelling of the optic nerve) or grade IV (papilledema present). The presence of these findings confirms target-organ damage and mandates immediate IV treatment.

8. You must lower pressure carefully with IV medicine (nicardipine, labetalol, clevidipine, esmolol, nitroprusside, or fenoldopam — NEVER sublingual nifedipine, because it drops pressure unpredictably and can cause stroke) AND hunt for the root cause at the same time, because treating the number alone is just a bandaid — you must fix the upstream trigger to stop the cascade. — The pressure is the gun, but the cause pulled the trigger. If you bring pressure down but the patient goes home and uses cocaine again, or stops taking pills, or has an undiagnosed pheochromocytoma still pumping out catecholamines, the emergency will repeat. Workup: detailed medication reconciliation and adherence history (most common cause is nonadherence). Urine drug screen (cocaine, amphetamines). Basic metabolic panel (creatinine for kidney injury, potassium). Urinalysis (hematuria, proteinuria, casts). Chest X-ray (pulmonary swelling (edema), widened mediastinum if dissection). ECG (ischemia, left-ventricular hypertrophy). Troponin if chest pain. Head CT non-contrast if neuro symptoms (to rule out bleed before considering thrombolysis). CT angiography chest if dissection suspected. Renal-artery duplex or CT-angiography renal arteries if young patient or refractory high blood pressure (hypertension) (stenosis). Plasma or urine metanephrines if paroxysmal symptoms or resistant high blood pressure (pheochromocytoma). Pregnancy test in any woman of childbearing age (preeclampsia). Treatment: ICU admission, continuous BP monitoring (arterial line if available), IV antihypertensive with titratable infusion (not boluses), and gradual reduction — target 10–20% drop in the first hour (NOT more than 25% or you risk starved blood flow (ischemia)), then toward 160/100 over the next 2–6 hours, then normal over 24–48 hours. Exceptions: aortic dissection requires systolic <120 mmHg within minutes; acute blood-starved (ischemic) stroke allows permissive high blood pressure unless thrombolysis planned. Choice of agent: nicardipine (dihydropyridine calcium-channel blocker, dilates arterioles, start 5 mg/h IV, titrate by 2.5 mg/h every 5–15 min, max 15 mg/h; avoid in acute heart failure because it can cause reflex a fast heart rate (tachycardia)). Labetalol (combined alpha- and beta-blocker, lowers pressure and heart rate, 10–20 mg IV bolus or 0.5–2.0 mg/min infusion; avoid in asthma or after cocaine because unopposed alpha-constriction can worsen pressure and cause coronary vasospasm). Clevidipine (ultra-short-acting dihydropyridine, start 1–2 mg/h, double every 90 seconds up to 21 mg/h; expensive, useful when you need tight second-to-second control). Esmolol (ultra-short-acting beta-blocker, 500–1000 mcg/kg load then 50–300 mcg/kg/min; ideal for aortic dissection, first-line to drop heart rate and dP/dt before adding vasodilator). Nitroprusside (direct NO donor, venous and arterial dilator, 0.3–10 mcg/kg/min; powerful but causes cyanide toxicity if used >24–48 hours or in renal failure; mostly second-line now). Fenoldopam (dopamine-1 receptor agonist, dilates renal and splanchnic beds, may protect kidney function, 0.1–0.3 mcg/kg/min; useful if concern for kidney injury). Monitor neuro exam, urine output, oxygen saturation, and serial labs (creatinine, troponin) every 4–6 hours. If pressure drops too fast and patient develops new confusion, focal weakness, or chest pain, stop the drip and allow pressure to drift back up slightly — you have crossed below their autoregulation threshold.

The art is walking the tightrope: fast enough to stop ongoing damage, slow enough to keep blood flowing to the brain and heart. There is no cookbook — every patient's curve is different.

## Symptoms · what the patient feels, and why

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- Blood pressure above 180/120 mmHg with signs that an organ is being hurt right now: When pressure inside the blood vessels stays dangerously high, it forces the heart to pump harder and crushes delicate tissues in the brain, kidneys, eyes, and blood-vessel walls, causing them to break down or bleed.
- Severe headache that feels explosive or the worst of your life: The extreme pressure inside the arteries stretches and irritates the blood vessels in the brain and the meninges (the protective coverings around the brain and spinal cord), triggering pain signals; sometimes pressure builds inside the skull itself.
- Blurred vision, seeing double, or sudden loss of part of your sight: The tiny blood vessels in the retina (the back layer of the eye that senses light) swell, leak, or bleed under the high pressure, scrambling the signals your eye sends to your brain.
- Chest pain or pressure, especially a squeezing or heavy feeling: The heart muscle needs more oxygen because it is working overtime against the high pressure, but the coronary arteries (the pipes feeding the heart) may not deliver enough blood, causing starved blood flow (ischemia) (not enough blood reaching the tissue to feed it) or even a heart attack.
- Trouble breathing or feeling like you cannot catch your breath: Fluid backs up into the lungs (pulmonary swelling (edema)) because the left ventricle (the main pumping chamber of the heart) cannot pump hard enough against the extreme pressure, so blood pools in the lung vessels and water leaks into the alveoli (the tiny air sacs where oxygen moves into your blood).
- Confusion, slurred speech, weakness on one side of the body, or trouble moving your face, arm, or leg: The brain is either not getting enough blood because an artery has torn or clogged (stroke), or pressure has damaged the blood-brain barrier (the wall that protects brain tissue) and caused swelling called with high blood pressure (hypertensive) encephalopathy (brain swelling from extreme pressure), scrambling the brain's ability to think and control the body.
- Nausea and vomiting: Rising pressure inside the skull presses on the brainstem (the lower part of the brain that controls automatic body functions like breathing and vomiting), or kidney damage releases waste into the blood that triggers the chemoreceptor trigger zone (the brain's nausea center).

- Back pain between the shoulder blades that feels tearing or ripping: The wall of the aorta (the body's largest artery, which carries blood from the heart to the rest of the body) may be tearing apart under the extreme pressure in a condition called aortic dissection, and the tear stretches pain nerves in the vessel wall and surrounding tissue.
- Decreased urine output or blood in the urine: The glomeruli (the kidneys' tiny filters that clean the blood) are crushed or torn by the high pressure, damaging their ability to make urine and sometimes causing bleeding that leaks into the urine.
- Severe anxiety, restlessness, or feeling like something terrible is about to happen: The body senses danger from the extreme pressure and releases epinephrine (adrenaline, the fight-or-flight hormone) and norepinephrine (a related stress hormone), which activate the amygdala (the brain's alarm center that flags danger) and speed up the heart and breathing.

## Focused exam

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- Blood pressure 180/120 mmHg or higher: This number separates with high blood pressure (hypertensive) emergency from with high blood pressure urgency. The high pressure itself squeezes and tears delicate blood vessels. But the real danger is whether organs — brain, heart, kidneys, eyes — are being damaged right now. Without that organ damage, it is urgency, not emergency.
- Swelling of the optic nerve (Papilledema) (swelling of the optic disc where the nerve enters the back of the eye) or retinal hemorrhages (flame-shaped bleeding in the retina): The tiny blood vessels in the eye are the same size and build as vessels in the brain and kidneys. When you see swelling or bleeding here, it means the pressure has climbed high enough to burst or stretch these delicate vessels. This tells you organ damage is happening right now, not someday.
- Focal neurologic deficits: weakness on one side, slurred speech, confusion, or loss of consciousness: The brain has lost its ability to protect itself from the high pressure. Normally the brain squeezes its blood vessels when pressure climbs, keeping flow steady — a process called autoregulation, controlled by the smooth muscle in the walls of brain arteries. When pressure climbs too high or too fast, autoregulation breaks, blood vessels leak fluid into brain tissue, and the brain swells. This can also mean a stroke from a burst vessel or a clot. Either way, brain cells are dying.
- Elevated serum creatinine or urinalysis showing blood (hematuria) or protein (proteinuria): The kidneys filter blood through millions of tiny clusters of vessels called glomeruli. High pressure damages these filters, making them leak protein and red blood cells into the urine. Waste products like creatinine pile up in the blood because the kidneys cannot clear them. This means the kidneys are failing right now.

- Chest pain or shortness of breath, with an S3 gallop on heart listening with a stethoscope (auscultation) or crackles (rales) in the lungs: The left ventricle (the heart's main pumping chamber) is struggling to push blood against the very high pressure. When it cannot keep up, blood backs up into the lungs, leaking fluid into the air sacs and making it hard to breathe. The S3 gallop is the sound of a failing, overfilled ventricle. This is acute heart failure.
- Severe, sudden-onset headache that feels different from any prior headache: The blood vessels in the brain are stretching and leaking fluid into the surrounding tissue, causing the brain to swell. This headache is not just discomfort — it is a sign that the brain is under attack from pressure and swelling right now.

## Diagnostics · every test answers a question

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- Basic metabolic panel (BMP) with serum creatinine and electrolytes → Are the kidneys still filtering waste, or are they failing? Are electrolytes dangerously out of balance?
- Urinalysis with microscopy → Are the kidney filters leaking blood or protein?
- Troponin I or T → Is the heart muscle being damaged by the strain of pumping against high pressure?
- Brain natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) → Is the heart stretched and failing from pressure overload?
- Electrocardiogram (ECG or EKG) → Is the left ventricle thickened from chronic high pressure, or is there a new heart attack?
- Chest X-ray → Is fluid backing up into the lungs? Is the aorta widened from a tear?
- Non-contrast head CT scan → Is there bleeding in the brain, stroke, or swelling?
- CT angiography of the chest (CTA chest) if aortic dissection is suspected → Is there a tear in the wall of the aorta?

## Treatment logic

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- IV labetalol (alpha and beta blocker) or nicardipine (calcium channel blocker), titrated to reduce mean arterial pressure by 10–20% in the first hour, then another 5–15% over the next 23 hours → Labetalol blocks both alpha receptors (which tighten blood vessels) and beta receptors (which speed the heart and increase pumping force). Nicardipine blocks calcium channels in vessel walls, stopping them from squeezing tight. This brings pressure down fast enough to stop ongoing organ damage but slow enough that the brain, kidneys, and heart do not lose blood flow suddenly. Organs that have adapted to high pressure can be hurt by pressure dropping too fast.

- Identify and treat the root cause: restart home antihypertensive medications, stop sympathomimetic drugs (cocaine, methamphetamine), screen for secondary high blood pressure (hypertension) (renal artery narrowing (stenosis), pheochromocytoma, primary aldosteronism), deliver the baby if preeclampsia or eclampsia, or start renal replacement therapy if kidneys have failed → Removes the reason pressure climbed out of control in the first place. For example, restarting an ACE inhibitor (which blocks the enzyme that makes angiotensin II, a powerful vessel-tightening hormone) or an ARB (which blocks angiotensin II receptors) helps the kidneys hold steady pressure long-term. Delivering the placenta in preeclampsia removes the source of the anti-angiogenic proteins that damage blood vessels. Finding and removing a pheochromocytoma stops the flood of epinephrine and norepinephrine (adrenaline hormones). Without fixing the root cause, pressure will climb back up.
- Intensive care unit (ICU) admission with continuous arterial blood pressure monitoring, hourly urine output measurement, and serial labs (creatinine, electrolytes, troponin) every 4–6 hours → Catches worsening organ injury early. Rising creatinine means the kidneys are failing further. Falling urine output (less than 0.5 mL/kg/hour) means kidneys are shutting down. Rising troponin means the heart is under too much strain. Continuous BP monitoring from an arterial line lets you adjust IV drip rates minute-by-minute to keep pressure in the safe zone.
- If aortic dissection is confirmed or suspected: IV esmolol (ultra-short-acting beta blocker) first to lower heart rate below 60 beats per minute, then add IV nicardipine or nitroprusside to drop systolic BP below 120 mmHg within 20 minutes → Esmolol blocks beta-1 receptors in the heart, slowing heart rate and reducing the force of each heartbeat. This lowers the shear stress (the twisting and tearing force) on the torn aortic wall with each beat. Adding nicardipine or nitroprusside (which releases nitric oxide, a powerful vessel relaxer) drops the pressure wave hitting the tear. This stops the tear from ripping further down the artery, which would block blood flow to the brain, kidneys, or legs, or cause the aorta to rupture and bleed out.
- In pregnancy with severe preeclampsia or eclampsia: IV labetalol or IV hydralazine (direct vasodilator) to keep systolic BP 140–150 mmHg and diastolic 90–100 mmHg. Avoid ACE inhibitors, ARBs, and nitroprusside. Give IV magnesium sulfate 4–6 g loading dose, then 1–2 g/hour infusion to prevent seizures. Plan delivery of the baby and placenta. → Labetalol and hydralazine (which relaxes vessel walls directly) lower blood pressure safely without harming the fetus. ACE inhibitors and ARBs cause kidney damage and death in the fetus; nitroprusside releases cyanide, which poisons the baby. Magnesium sulfate blocks NMDA receptors (excitatory receptors) in the mother's brain, calming overexcited neurons and preventing eclamptic seizures. Delivery of the placenta is the only definitive cure for preeclampsia because the placenta releases sFlt-1 and soluble endoglin (anti-angiogenic proteins that damage blood vessel linings and drive the high pressure).

- Supplemental oxygen (nasal cannula 2–6 L/min or non-rebreather mask 10–15 L/min if oxygen saturation < 90%), elevate head of bed 30–45 degrees, restrict sodium intake to less than 2 grams per day and restrict fluids to 1–1.5 liters per day if pulmonary edema (fluid flooding the lungs) or volume overload is present → Oxygen keeps tissues alive while the heart struggles to pump. Elevating the head helps gravity pull fluid out of the lungs and makes breathing easier. Restricting salt keeps the kidneys from holding onto extra water (because sodium pulls water with it). Restricting fluids reduces the workload on the failing heart by keeping fluid from building up further in the lungs and body.
- Loop diuretic (IV furosemide 20–80 mg) if pulmonary edema (fluid flooding the lungs) is present → Furosemide blocks the sodium-potassium-chloride cotransporter in the thick ascending limb of the loop of Henle (the part of the kidney tubule that reabsorbs the most salt). This makes the kidneys dump salt and water into the urine, pulling fluid out of the lungs and the bloodstream. This relieves shortness of breath and reduces the volume the failing heart has to pump.

## Medications

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### Labetalol (IV) · Combined alpha- and beta-blocker

Labetalol blocks two kinds of landing spots in the body. It blocks beta-1 receptors (spots on heart muscle that normally let adrenaline speed up the heart) and alpha-1 receptors (spots on artery walls that normally tell vessels to squeeze tight). By covering both, labetalol slows the heart a bit and relaxes arteries so blood flows through wider pipes, which drops pressure smoothly.

1. Labetalol enters the bloodstream and travels to the heart and arteries.
2. It sits on beta-1 receptors in the heart, blocking adrenaline from making the heart beat faster or harder.
3. It also sits on alpha-1 receptors in artery walls, blocking signals that squeeze vessels.
4. With less squeezing and gentler pumping, blood pressure falls in a controlled way within 5 to 10 minutes.

#### SAFETY WATCH

Can slow the heart too much (bradycardia) or drop pressure too fast—check heart rate and blood pressure every 5 to 10 minutes at first. · Avoid in severe asthma or COPD flare-ups, because blocking beta receptors can tighten airways and make breathing harder. · Avoid in acute heart failure with a very weak pump (severe systolic dysfunction), because the heart may need adrenaline's help to squeeze hard enough. · Watch for dizziness or feeling faint when standing, which means pressure dropped too low. · Safe in pregnancy, including preeclampsia and eclampsia.

**Nicardipine (IV) · Calcium channel blocker (dihydropyridine)**

Nicardipine blocks calcium channels in the muscle walls of arteries. Normally calcium rushes into those muscle cells through tiny gates and makes them squeeze tight. When nicardipine closes the gates, calcium stays out, the muscle relaxes, and the artery widens. Wider arteries mean less resistance, so pressure drops smoothly and quickly.

1. Nicardipine is infused into a vein and circulates to arteries all over the body.
2. It plugs L-type calcium channels in artery muscle cells, stopping calcium from rushing in.
3. Without calcium, the muscle cells relax instead of squeezing.
4. Arteries widen, resistance falls, and blood pressure drops within 5 to 15 minutes.

**SAFETY WATCH**

Can drop pressure too fast if the dose climbs too quickly—titrate slowly and check pressure every 5 to 15 minutes. · May cause headache or flushing (warm, red face) because blood vessels in the head widen too. · Watch for reflex fast heart rate (tachycardia) as the body tries to keep up when pressure falls. · Safe in most cases, but slow the dose if pressure is already falling or the patient feels dizzy.

**Clevidipine (IV) · Calcium channel blocker (dihydropyridine, ultra-short-acting)**

Clevidipine works just like nicardipine—it blocks calcium channels in artery muscle so vessels relax and widen. The key difference is that clevidipine breaks down in the blood in about one minute. Enzymes in the blood called esterases chop it apart. So if pressure drops too much, turning off the drip brings pressure back up fast. This makes it very easy to fine-tune.

1. Clevidipine is infused continuously into a vein.
2. It plugs L-type calcium channels in artery muscle, stopping calcium entry.
3. Muscles relax, arteries widen, and pressure drops within 2 to 4 minutes.
4. Esterases (enzymes in the blood) chop the drug apart in about one minute, so its effect fades quickly when the drip stops.

**SAFETY WATCH**

Can drop pressure too fast—watch the monitor closely and adjust the drip every few minutes. · Comes dissolved in a fat emulsion (like IV nutrition), so avoid long or high-dose infusions in patients who cannot handle extra fat (severe pancreatitis or very high triglycerides over 400 mg/dL). · May cause headache or a warm, flushed feeling. · Reflex fast heart rate is common—pair with a beta-blocker if the heart is racing and the patient has aortic dissection.

**Esmolol (IV) · Beta-1 blocker (ultra-short-acting, cardioselective)**

Esmolol blocks beta-1 receptors mainly in the heart. These receptors are landing spots for adrenaline and noradrenaline (the body's main fight-or-flight chemicals). When adrenaline lands, the heart beats faster and harder, which raises pressure. Esmolol sits on those spots so adrenaline cannot land. The heart slows down and pumps with less force, and pressure falls. Esmolol breaks down in red blood cells in about 9 minutes, so its effect fades fast when you stop the drip.

1. Esmolol is infused into a vein and reaches the heart quickly.
2. It covers beta-1 receptors so adrenaline and noradrenaline cannot attach.
3. The heart slows and each beat is gentler, so less blood is pushed out per minute.
4. Pressure drops, especially the top number (systolic), within 1 to 2 minutes.
5. Esterases (enzymes) in red blood cells chop esmolol apart in under 10 minutes, so the effect reverses fast when the drip stops.

**SAFETY WATCH**

Can slow the heart too much (bradycardia) or drop pressure too low—monitor heart rate and blood pressure every 5 minutes at first. · Avoid in severe asthma or COPD flare, because even though esmolol targets the heart, it can still tighten airways at high doses. · Avoid in acute heart failure with a very weak pump (severe systolic dysfunction), because blocking adrenaline may make the heart too weak to pump enough blood. · Watch for dizziness or confusion if pressure or heart rate fall too far. · First choice (with a vasodilator) in aortic dissection to slow the heart and protect the aorta from tearing further.

**Sodium nitroprusside (IV) · Direct nitric-oxide donor (vasodilator)**

Nitroprusside releases nitric oxide inside artery and vein walls. Nitric oxide is a gas molecule that tells smooth muscle to relax. When the muscle relaxes, both arteries and veins widen. This drops resistance in arteries (lowers pressure) and eases the load on the heart. Nitroprusside works in seconds, so it is very powerful but must be watched closely.

1. Nitroprusside is infused into a vein and breaks apart in the blood, releasing nitric oxide.
2. Nitric oxide drifts into smooth muscle cells and turns on an enzyme called guanylate cyclase.
3. Guanylate cyclase makes a messenger called cyclic GMP (cGMP).
4. cGMP tells the muscle to relax.
5. Both arteries and veins widen, resistance falls, and pressure drops within seconds to 2 minutes.

**SAFETY WATCH**

Extremely powerful—pressure can crash in seconds, so check the monitor every 2 to 5 minutes and use an arterial line (a small tube in an artery for continuous pressure reading) when possible. · Breaks down into cyanide in the blood; the liver turns cyanide into thiocyanate, which the kidneys clear. In long infusions (more than 24 to 48 hours) or high doses (more than 2 micrograms per kilogram per minute for many hours), cyanide or thiocyanate can build up and poison cells. Watch for unexplained metabolic acidosis (acid building up in the blood) (high acid in the blood), confusion, seizures, or rising lactate. · Avoid in severe kidney failure unless you can measure thiocyanate levels, because the kidneys clear thiocyanate and it can build up. · Light breaks the drug down, so wrap the IV bag and tubing in foil or opaque material. · In aortic dissection, never use nitroprusside alone—always pair with a beta-blocker first to protect the aorta from extra stress. · Rarely used now; nicardipine and clevidipine are safer and easier to control.

**Hydralazine (IV or IM) · Direct arterial vasodilator**

Hydralazine relaxes the smooth muscle in artery walls, though the exact way it does this is still not completely clear. It may open potassium channels in the muscle cell membrane, letting potassium leak out. That makes the cell less excitable and the muscle relaxes. It may also release a little nitric oxide. Either way, arteries widen, resistance drops, and pressure falls. Hydralazine is slower and less predictable than nicardipine or labetalol, but it has decades of safety data in pregnancy.

1. Hydralazine is injected into a vein or muscle and travels to artery walls.
2. It triggers muscle relaxation—likely by letting potassium flow out of cells or by releasing nitric oxide.
3. Arteries widen, lowering resistance.
4. Pressure drops over 10 to 30 minutes (slower than most IV agents).

**SAFETY WATCH**

Pressure drop is less predictable—sometimes very little, sometimes a big drop—so recheck pressure every 5 to 15 minutes. · Can cause reflex fast heart rate and headache. · Repeated doses can cause fluid retention (the body holds on to salt and water as pressure falls). · Preferred in pregnancy (especially severe preeclampsia and eclampsia) because decades of use show it is safe for mother and baby, but always pair with magnesium sulfate (IV) to prevent seizures. · ACOG guidelines list hydralazine and labetalol as first-line IV agents for acute severe high blood pressure (hypertension) in pregnancy.

**Phentolamine (IV) · Alpha-blocker (non-selective alpha-1 and alpha-2)**

Phentolamine blocks alpha receptors on artery walls. Adrenaline, noradrenaline, and similar chemicals (such as those released by cocaine or a pheochromocytoma tumor) normally land on alpha receptors and squeeze vessels tight. Phentolamine covers those receptors so the squeeze signal cannot get through. Vessels relax, resistance drops, and pressure falls. It works fast and is the go-to choice when too much adrenaline-like activity is the problem.

1. Phentolamine is injected into a vein and circulates to arteries.
2. It sits on alpha receptors, blocking adrenaline, noradrenaline, and related molecules.
3. Without the squeeze signal, artery muscle relaxes and vessels widen.
4. Resistance falls and pressure drops within 1 to 2 minutes.

**SAFETY WATCH**

Can drop pressure very fast, especially if the patient is dry (low on fluid)—check pressure every 5 minutes at first. · Reflex a fast heart rate (tachycardia) (fast heart rate) is common—the heart speeds up as vessels open. Pair with a beta-blocker if needed, but in pheochromocytoma give phentolamine (alpha-blocker) first. Giving a beta-blocker alone can make pressure worse by leaving alpha receptors unopposed. · Watch for dizziness or fainting if pressure falls too far. · Mainly used for cocaine toxicity, pheochromocytoma crisis, or tyramine crisis (from certain foods such as aged cheese with an MAOI antidepressant).

**Fenoldopam (IV) · Selective dopamine-1 receptor agonist**

Fenoldopam turns on dopamine-1 (D1) receptors in kidney tubules and artery walls. When these receptors switch on, arteries relax and widen, and the kidneys flush out more salt and water in the urine. Both actions lower pressure. Fenoldopam is especially helpful when you want to protect the kidneys during a with high blood pressure (hypertensive) emergency, because it increases blood flow through the kidney filters (glomeruli) and may help preserve kidney function.

1. Fenoldopam is infused into a vein and reaches arteries and kidneys.
2. It activates dopamine-1 receptors, which relax artery smooth muscle and open vessels.
3. At the same time, kidney tubules let more sodium (salt) and water escape into urine instead of being reabsorbed into the blood.
4. Wider arteries and less fluid in the blood both lower pressure within 5 to 15 minutes.

**SAFETY WATCH**

Can drop pressure too fast—titrate slowly and check every 5 to 15 minutes. · Reflex a fast heart rate (tachycardia) (fast heart rate) and headache are common as vessels widen. · May raise pressure inside the eye (intraocular pressure), so use carefully in patients with glaucoma. · Helps protect kidneys by increasing renal blood flow, so it is often chosen when creatinine is rising or urine output is low, but it does not replace treating the underlying kidney problem. · Evidence for kidney protection is mixed; some studies show benefit in acute kidney injury, but it is not proven to prevent dialysis or improve long-term kidney outcomes.

**Magnesium sulfate (IV) · N-methyl-D-aspartate (NMDA) receptor antagonist and calcium modulator**

Magnesium sulfate has two jobs in a with high blood pressure (hypertensive) emergency caused by preeclampsia or eclampsia. First, it prevents and stops seizures by blocking NMDA receptors in the brain (receptors that normally let nerve cells fire rapidly) and by competing with calcium at nerve and muscle junctions, which calms overactive nerves. Second, it gently lowers blood pressure by relaxing artery walls—magnesium blocks calcium entry into smooth muscle cells, so vessels widen a bit. The seizure-prevention effect is much stronger than the blood-pressure effect, so magnesium is always paired with another drug (such as labetalol or hydralazine) to control pressure.

1. Magnesium sulfate is infused into a vein, usually as a loading dose followed by a continuous drip.
2. It travels to the brain and blocks NMDA receptors, reducing the risk of seizures caused by preeclampsia or eclampsia.
3. It also competes with calcium at nerve terminals and in artery muscle cells, calming overactive nerves and gently relaxing arteries.
4. Seizure protection begins within minutes and lasts as long as the drip continues; blood pressure may drop slightly but not enough to replace a dedicated antihypertensive.

**SAFETY WATCH**

Can build up in the blood and cause magnesium toxicity, especially if kidney function is poor. Early signs are loss of deep tendon reflexes (the knee-jerk reflex disappears), then muscle weakness, trouble breathing, very slow heart rate (bradycardia), and finally cardiac arrest. Check reflexes and breathing every hour, and stop the drip if reflexes disappear. · Monitor urine output—if the patient is making less than 30 mL per hour, magnesium may build up because the kidneys clear it. · Can cause flushing (warm, red skin), nausea, and feeling very warm. · Keep calcium gluconate (IV) at the bedside as an antidote—it reverses magnesium toxicity in seconds by competing with magnesium at receptors. · ACOG strongly recommends magnesium sulfate for seizure prevention in severe preeclampsia and for stopping seizures in eclampsia. It is more effective than other seizure medicines (such as phenytoin or diazepam) for eclampsia.

## Red flags

- Blood pressure above 180/120 with any chest pain, shortness of breath, back pain, headache, vision changes, weakness, numbness, or confusion — call 911 immediately
- Signs of stroke: sudden face droop, arm weakness, or slurred speech — every minute counts
- Seizure, loss of consciousness, or severe agitation with sky-high blood pressure
- Chest pain or back pain that feels like tearing or ripping and may move between the chest and back — may be aortic dissection
- Pulmonary edema (fluid flooding the lungs): gasping for air, coughing up pink frothy spit, unable to lie flat

## Nursing priorities

1. Place the patient on continuous BP monitoring with an arterial line or automatic cuff cycling every 5 to 15 minutes — You need to see BP changes minute by minute because dropping pressure too fast can cause stroke or heart attack, while dropping too slow leaves organs getting damaged.
2. Start IV access with at least one large-bore IV and prepare titratable BP medicine like nicardipine or labetalol — IV medicines work in minutes and you can adjust the dose up or down to hit the target of 20 to 25 percent BP reduction in the first hour without overshooting.
3. Measure urine output every hour with a Foley catheter and monitor for less than 30 mL per hour — The kidneys show you if organs are getting enough blood flow; dropping BP too fast can stop urine production, meaning you need to slow down the BP reduction.

4. Perform and document a full neurologic exam every hour, checking pupil size, grip strength, speech, and orientation — The brain is very sensitive to BP changes; new confusion or weakness means stroke or brain bleeding, requiring immediate CT scan and possible BP target adjustment.
5. Keep the head of the bed at 30 degrees and maintain a quiet, dim environment — Elevating the head reduces brain swelling pressure, and calm surroundings lower stress hormones like adrenaline that push BP higher.
6. Hold all oral BP medicines until the doctor gives new orders after reviewing the home medication list — Adding oral medicines on top of IV drips causes unpredictable stacking that can drop pressure too far; you restart home pills only after finding the right IV dose.
7. Monitor continuous EKG and watch for ST changes, new T-wave flips, or arrhythmias — High pressure forces the heart to pump against resistance, causing heart attacks or dangerous rhythms; EKG changes mean you need troponin blood tests and cardiology consultation.
8. Draw labs including creatinine, BUN, electrolytes, troponin, and urinalysis at admission and recheck creatinine in 6 to 12 hours — Rising creatinine shows kidney damage progression; low potassium or high potassium changes your medicine choices; troponin elevation confirms heart damage needing different treatment intensity.

## Sources

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- ACC/AHA Guideline on the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults (2017)
- American Heart Association Scientific Statement on Hypertensive Crisis
- European Society of Cardiology / European Society of Hypertension Guidelines for the Management of Arterial Hypertension
- UpToDate

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