



**Objectives:** Define hypertensive urgency versus emergency, discuss end-organ effects of hypertensive emergency, explain diagnostic intervention, evaluate need for rapid versus delayed intervention in special scenarios.

### **Is hypertensive “urgency” really urgent?**

Let’s be honest. Hypertension is common. 1 in 3 adults >20 years old have it. It is estimated that most patients with BP >180/>120 have no major acute end-organ damage necessitating rapid lowering BP. In fact, it has been demonstrated that this might be more harm than good for the patient. This is deemed “hypertensive urgency,” where one has acutely elevated BP +/- mild headache in the setting of no end-organ ischemia (hypertensive encephalopathy, retinal hemorrhages, papilledema, or acute and subacute kidney injury). This definition has come under fire lately, and it seems more and more that it’s not an actual entity. There’s no high-quality evidence out there to guide the management of these patients.

We don’t believe there is such a pathology as hypertensive urgency, and we are against using it as a real medical term. In short, we don’t think it exists. Call it what it is, asymptomatic hypertension.

Those with elevated BP and who have no evidence of end-organ ischemia can follow up as an outpatient. We have noticed that even boards have now shifted away from aggressively lowering BP. In fact, boards want you to send these asymptomatic patients home. Be more minimalistic and do not treat aggressively if the patient is asymptomatic with an elevated BP. Place the patient in a quiet, undisturbed room with the lights dimmed, and remeasure their BP in 30 minutes. We guarantee in most instances, it will go down! In one study, for example, 30 minutes of rest in a quiet room produced a fall in blood pressure  $\geq 20/10$  mmHg in 32 percent of adults with severe asymptomatic hypertension.

Discharge these patients with close outpatient follow-up. If patients are in pain or anxious, treat those symptoms etiologies and recheck their blood pressures, don’t treat them with IV hydralazine or oral clonidine.

There are those who do have symptoms like chest pain, dyspnea, epigastric pain with nausea, stroke symptoms, seizures, or any neurological complaint with an elevated BP, however, needs to be worked up for...

### **What is Hypertensive Emergency?**

<1% of ER visits all year. Elevated BP with end-organ ischemia. This is such a lame term, and we really aren’t a fan of it because the term “hypertensive emergency” is a wastebasket category for basically any patient with elevated blood pressure and some underlying pathology. It is important to identify as the five-year mortality approached 100%.

We encourage you to think of hypertensive emergency as more of a “chief complaint” or “presentation” indicating a possible severe underlying disease (aortic dissection, intracranial bleeding, preeclampsia, SCAPE, etc.).

By itself, isolated hypertensive emergency was previously referred to as “malignant hypertension.” It can be any high blood pressure, contrary to popular belief. Naturally, the higher the BP, likely the higher the risk. It can also develop in patients without a history of hypertension.

We don’t really understand the pathophysiology of an isolated hypertensive emergency. Some initial, abrupt rise in vascular resistance likely occurs, resulting in downstream disruption of the arterial baroreflex and neurohormonal pathways.

In patients who are symptomatic with elevated blood pressure, a workup should be pursued. Usually, the cause is quickly identified (X-ray and story fitting SCAPE, CT showing ICH or aortic dissection, etc.).

Commonly ordered laboratory tests include +/- troponins, BMP (Cr), BNP, UA (blood), EKG, CXR, +/- CT head.

### **Management of hypertensive emergency**

This is the most high-yield portion and the most heavily tested area for exams.

Below are classic conditions that require emergent blood pressure lowering and addressing the underlying condition. We have separate, detailed guides on these conditions as well.

## Salt of the Earth: Hypertensive Emergencies

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**Posterior reversible encephalopathy syndrome (previously called hypertensive encephalopathy):** a disorder of reversible subcortical vasogenic edema in patients with acute neurologic symptoms and classic CT/MRI features. It is primarily a diagnosis of exclusion after other causes of central nervous system dysfunction are ruled out.

Classic symptoms include headache, vision changes, altered mental status, nausea/vomiting, and even seizures.

Aggressive BP management, sometimes by as little as 10-15%, usually results in rapid symptom improvement in patients. There are no randomized trials displaying a good target goal, but ~160/100 mmHg is what people seem to be quoting.

Pro tip: Many of these patients are in a volume-contracted state, and if they can tolerate IV fluids this might be greatly beneficial for them. Think about it: initiating IV antihypertensives force the circulatory system to relax suddenly, decreasing blood volume to already-damaged organs. Think about 500-1000 mL of lactated Ringers to help patients out before aggressive IV antihypertensive therapy.

Agents used include clevidipine > nicardipine > nitroprusside

Clevidipine has the advantage of having fast onset, and easier titration. At many shops it is typically cheaper as well.

**Aortic dissection:** This is the truest form of hypertensive emergency. *Rapid* lowering is needed as fast as possible in order to prevent false lumen expansion. Target: "as low as possible", but realistically 100-120 systolic in <30 minutes.

Agent of choice: Esmolol > Labetalol > Diltiazem > Nicardipine/Clevidipine > Nitroprusside

Beta blockers are the first line agents, any IV beta-blocker you can get your hands on- use it! Titrate to HR <60 bpm. Be aggressive.

Fentanyl boluses need to be occurring simultaneously with BP control as it reduces sympathetic pain response which increases HR and BP.

Agents NOT to use: Nitroglycerin, hydralazine, or other vasodilators which cause increase in inotropy.

Check out our [full review on aortic dissection here](#).

**Subarachnoid hemorrhage:** not clear and still debated. Goal of SBP <160 is reasonable. Labetalol, nicardipine, clevidipine are preferred. The benefit of lowering BP might be offset by risk of infarction (CPP = MAP - ICP). i.e. if ICP is high then the only variable maintaining perfusion is MAP. The patient's consciousness might be a helpful marker: alert and oriented = SBP ~140. Patient impaired = SBP ~160.

Agents of choice: nicardipine, enalapril

Management of vasospasm: a devastating complication of SAH. Oral nimodipine should be given.

Check out our [full review of SAH here](#).

**Non-traumatic intracerebral hemorrhage:** this is still a matter of debate, but let's try to keep it somewhat grounded.

SBP >220, aggressive reduction of BP is warranted with IV medication to ~140 SBP.

SBP >220-150 and evidence of increased ICP, reduce BP using intermittent/continuous IV meds to modest range ~140-160 SBP over the course of the first few hours. It might improve functional outcomes, but it has not been shown to reduce mortality and might increase risk of AKI.

Agent of choice: nicardipine > labetalol, clevidipine, esmolol, enalaprilat

**Ischemic stroke:** Broken down into the following-

tPA candidates- BP needs to be <185/110 prior to tPA administration. No aggressive lowering is warranted if BP is already <185/110.

Non-tPA candidates- BP needs to be <220/120. No aggressive lowering is warranted if <220/120.

Agent of choice: clevidipine > nicardipine. A couple of labetalol pushes may be all that is needed in some patients.

Check out our [full review guide on acute stroke management here](#).

**SCAPE:** the goal is to reduce afterload and relieve volume excess. Diuretics can be mainstay, but they take a while to work and minimally affect BP in the acute setting. *Nitroglycerin is first line*. Hydralazine should not be used as its effect is unpredictable and can increase inotropy.

Check out our [full review guide on SCAPE here](#). People really mess up the management on this condition quite often.

**Acute coronary syndrome:** nitroglycerin, nicardipine, clevidipine, metoprolol/esmolol.

Check out our [full review on ACS management here](#).

Nitroglycerin should be used with caution or avoided in the setting of right ventricular infarction, severe aortic stenosis, and PDEi use within the previous 24 hours.

**Pregnancy hypertensive emergency:** BP  $\geq$  140/90 in pregnancy, especially after the 20<sup>th</sup> week, should raise concern. Agents of choice: methyl dopa, hydralazine, labetalol. Magnesium is absolutely needed and is the mainstay therapy in suspected pre-eclampsia or eclampsia. HELLP syndrome is a life-threatening complication of pregnancy that is considered to be a variant of pre-eclampsia: Hemolysis, ELevated liver enzymes, Low Platelets. It typically occurs during the later stages of pregnancy but can occur postpartum.

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