

Managing hypertensive emergencies in the ED

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The following 5 patients arrive at various times during your shift in a community emergency department (ED).

- Patient A is a 65-year-old man with nausea, vomiting, and confusion.
- Patient B is a 73-year-old woman with sudden shortness of breath, pink sputum, and heavy chest pain.
- Patient C is a 56-year-old man with sharp, tearing chest and back pain.
- Patient D is a 64-year-old woman with a 6-hour history of right-sided weakness.
- Patient E is a 51-year-old woman with a mild headache, concerned about her history of hypertension.

Interestingly, all 5 patients arrive with identical vital signs: heart rate of 100 beats/min, blood pressure (BP) of 209/105 mm Hg, respiration rate of 20 breaths/min, and temperature of 36.9°C. Each time the nurse expresses concern over the elevated BP. Which of the 5 patients require emergent hypertension treatment?

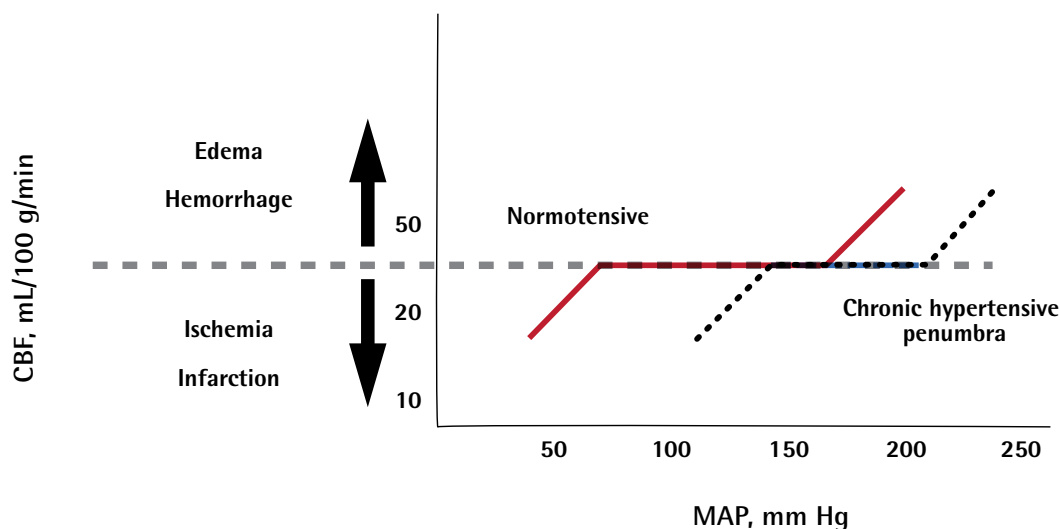
Discussion

Markedly elevated BP is a source of anxiety, not only for patients, but also for the doctors and nurses who care for them. However, even with large fluctuations in BP, the body's normal autoregulatory mechanisms maintain a relatively constant perfusion pressure to vital organs (**Figure 1**).¹ When do we need to intervene and consider lowering a patient's BP emergently?

A *hypertensive emergency* is defined as a rapid and progressive decompensation of vital organ function caused by an inappropriately increased BP. It is not defined by a specific number, but rather by evidence of acute dysfunction in cardiovascular, neurologic, or renal systems.

If end-organ dysfunction exists, emergent treatment with parenteral medication is indicated. There are multiple drug classes that can be used, each with a different mechanism of action (**Table 1**). The drug of choice in each hypertensive emergency should target the specific organ dysfunction involved. Regardless of which drug

Figure 1. Defective autoregulation in the ischemic brain: MAP versus CBF versus ischemia.



CBF—cerebral blood flow, MAP—mean arterial pressure.
Adapted from Varon and Marik.¹

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Table 1. Drugs of choice for hypertensive emergencies

MECHANISM OF ACTION	DRUGS
Sympathetic blockers	
• α	Phentolamine
• β	Metoprolol, esmolol
• α and β	Labetalol
Vasodilators	
• Arterial	Hydralazine
• Venous	Nitroglycerin
• Both	Nitroprusside
Angiotensin-converting enzyme inhibitors	Enalapril (intravenous) Captopril (sublingual)
Calcium channel blockers	Nicardipine (intravenous) NOT nifedipine (sublingual)

you choose, the goal is a controlled decrease of mean arterial pressure (MAP) over hours.² We need only return the BP to a level where autoregulation restores normal perfusion pressure to vital organs, not to “normal” BP levels.

In each of the following scenarios, consider 2 questions: *Should I manage this patient's BP emergently? If yes, what is the optimal treatment plan?*

Patient A: 65-year-old man with nausea, vomiting, and confusion. The patient is uncooperative and has an altered level of consciousness. Physical examination reveals papilledema but no obvious focal neurologic deficit.

Hypertensive encephalopathy is a severe presentation of end-organ dysfunction. Cerebral autoregulation is overwhelmed, resulting in a combination of vasodilation, edema, and increased intracranial pressure. Clinically, patients present with headache, visual changes, nausea, and vomiting. They might complain of transient and migrating nonfocal neurologic deficits, and might progress to seizures and coma.

Controlled BP reduction results in rapid improvement of neurologic symptoms. Pure vasodilators like nitroprusside have theoretical risks of intracranial shunting, which could increase intracranial pressure. Labetalol allows measured reduction by titrated boluses or continuous drip. The patient requires monitoring in the intensive care unit, and other causes of encephalopathy should be investigated if rapid improvement is not seen.

Drug of choice: Intravenous labetalol, bolus or infusion.

Target: Reduce MAP by 20% to 25% over 2 to 8 hours.

Patient B: 73-year-old woman with sudden shortness of breath, pink sputum, and heavy chest pain. Patient B is having difficulty speaking in full sentences. Physical examination reveals bilateral crackles in her lungs, an elevated jugular venous pressure, and no heart

murmurs. An electrocardiogram reveals signs of hypertrophy but no acute ischemic change.

Unlike primary cardiogenic shock, acute pulmonary edema often presents with extreme hypertension, which overloads cardiac reserve. These patients require emergent management of elevated BP to reduce cardiac preload and afterload.

Vasodilation with nitroglycerin is the treatment of choice in acute pulmonary edema. It primarily reduces preload, decreasing the workload of the heart. An intravenous nitroglycerin drip can be rapidly titrated up to numerical and symptomatic end points. Repeated sprays of sublingual nitroglycerin can also be effective. In addition, acute use of angiotensin-converting enzyme inhibitors is appropriate to reduce afterload and improve cardiac output.³ Only after these measures and positive pressure ventilation have been started should consideration be given to loop diuretics. Furosemide has little effect in the initial, emergent phase until BP control reduces stress on the heart.

Drug of choice: Nitroglycerin infusion; intravenous enalaprilat or sublingual captopril.

Target: Reduce MAP by 20% to 25% and symptomatic improvement.

Patient C: 56-year-old man with sharp, tearing chest and back pain. Patient C's pain started suddenly and he complains of weakness and paresthesia in his left arm. Physical examination reveals differential BPs and evidence of a new aortic insufficiency murmur.

Aortic dissection is largely a disease of hypertension. A sudden increase in BP and heart rate combine to create shear stress that tears into the intimal layer of the aorta. The tear propagates down the aorta, resulting in migrating pain, neurovascular symptoms (eg, obstructing vascular branches, emboli), or sudden death (eg, cardiac tamponade, aortic rupture). Diagnosis requires a high index of suspicion and urgent imaging (eg, computed tomography, magnetic resonance imaging, transesophageal echocardiography).⁴

Treatment of aortic dissection requires simultaneous control of BP and heart rate to control shear stress. Unlike other hypertensive emergencies, the goal is to reduce BP rapidly, as long as there is no evidence of resulting hypoperfusion. Nitroprusside has a rapid onset of action, and provides both arterial and venous dilation. Heart rate control with β -blockers must be initiated first to avoid reflex tachycardia that will propagate the dissection. Alternatively, labetalol, with its α -blocking and β -blocking properties, is a relatively user-friendly option for controlling heart rate and BP simultaneously.

Drug of choice: Nitroprusside or esmolol infusion; labetalol boluses or infusion.

Target: Rapidly reduce systolic BP to 110 mm Hg if there is no evidence of hypoperfusion.

Patient D: 64-year-old woman with a 6-hour history of right-sided weakness. Patient D is aphasic and unable to walk. On physical examination, marked right-sided hemiplegia is noted.

Both hemorrhagic and embolic strokes can be related to markedly elevated BP. In either case, the injured brain shifts the cerebral autoregulation curve to the right. As a result, a higher MAP is *essential* to maintaining adequate cerebral blood flow and not extending the affected stroke territory. Therefore, BP should *not* be lowered in the acute period except in extreme situations (ie, BP >220/120 mm Hg in embolic cerebrovascular accidents or >180/100 in hemorrhagic cerebrovascular accidents).⁵ If acute hypertension management is necessary, care should be taken to reduce BP gradually and only to just below these levels.

What if she had presented within 2 hours? Thrombolysis of embolic strokes is contraindicated in the presence of extreme hypertension owing to increased risk of bleeding. Current stroke guidelines allow attempts to reduce the BP acutely to 185/110 mm Hg before treatment, and to maintain BP at less than 180/105 mm Hg after thrombolytic treatment.⁵

Drug of choice: Labetalol; nicardipine; hydralazine.

Target: If no thrombolytic is given, reduce BP only if it is greater than 220/120 mm Hg (embolic) or greater than 180/100 mm Hg (hemorrhagic); if a thrombolytic is given, reduce BP to 180/105 mm Hg before treatment and 180/100 mm Hg after treatment.

Patient E: 51-year-old woman with a mild headache, concerned about her history of hypertension. Without evidence of end-organ dysfunction, acute treatment of hypertension in the ED should be avoided. Reactive hypertension (eg, pain, anxiety) usually resolves with management of the precipitating stimulus. Those with chronic hypertension, known or undiagnosed, might have shifted their autoregulation curve to the right, tolerating a higher baseline MAP. These patients require gradual BP reduction over time on an outpatient basis and should be directed to consult their regular primary care physicians. Acutely elevated BP that persists in the ED with evidence of end-organ damage (left ventricular hypertrophy, proteinuria or elevated creatinine levels, retinopathy, etc), but not dysfunction, should have more urgent follow-up arranged with the patient's own physician or at a local hypertension clinic. However, immediate reduction of BP before

Further reading and resources

For physicians

Marik PE, Varon J. Hypertensive crisis challenges and management. *Chest* 2007;131(6):1949-62.

Decker WW, Godwin SA, Hess EP, Lenamond CC, Jagoda AS; American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Asymptomatic Hypertension in the ED. Clinical policy: critical issues in the evaluation and management of patients with asymptomatic hypertension in the emergency department. *Ann Emerg Med* 2006;47(3):237-49.

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For patients

Heart and Stroke Foundation [website]. *My blood pressure action plan*. Ottawa, ON: Heart and Stroke Foundation; 2011. Available from: http://heartandstroke.ca/hs_bp2.asp?media=bp.

Hypertension Canada [website]. *Education: public recommendations*. Calgary, AB: Hypertension Canada; 2010. Available from: www.hypertension.ca/education.

discharge from the ED risks hypoperfusion and should only be done with caution.^{6,7}

Conclusion

Hypertension is a common presentation in the ED, but true hypertensive emergencies are more rare. Physicians should look for evidence of acute end-organ dysfunction rather than focus on specific numbers. The goal of treatment should be controlled reduction of BP with parenteral agents to avoid harming those we are trying to help. Follow-up for all hypertensive patients who do not require admission should be arranged to address long-term complications.



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Competing interests

None declared

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: BOTTOM LINE

- Hypertensive emergencies are defined by evidence of end-organ dysfunction *not* by a specific number.
- Physicians should consider the risks and benefits of lowering blood pressure emergently in individual patient scenarios.
- Multiple antihypertensive drug classes exist. Choose one that targets the specific organ dysfunction involved.
- Follow-up for all hypertensive patients who are not admitted to hospital is advised for long-term control and risk assessment.

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