Chapter 58
Approach to Acute Severe Hypertension in the ICU

58.1 Introduction

Hypertension is a common condition encountered in acute care settings, particularly within the intensive care unit (ICU). While elevated blood pressure is frequent among critically ill patients, not all instances necessitate immediate intervention. Hypertension in the ICU can be reversible, often resulting from transient factors such as pain, anxiety, or agitation, or irreversible when linked to persistent pathological conditions. Understanding the nuances of hypertension in critically ill patients is essential for effective management and improving patient outcomes [1, 2]. [Ref: Algorithm 58.1].

58.2 Classification of Hypertension in the ICU

The classification of hypertension in the ICU is crucial for guiding management strategies. It is categorized based on blood pressure levels and the presence or absence of target-organ damage:

- Hypertensive Emergency: Characterized by severe elevations in blood pressure—typically a systolic blood pressure (SBP) exceeding 180 mm Hg or a diastolic blood pressure (DBP) over 110–120 mm Hg—accompanied by new or worsening target-organ damage. Immediate medical intervention is required to prevent morbidity and mortality.
- Asymptomatic Markedly Elevated Blood Pressure: Patients exhibit significantly elevated blood pressure (SBP/DBP >180/110–120 mm Hg) without any signs of target-organ damage. While immediate organ damage is not present, these patients require careful monitoring and intervention to prevent progression.

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- Asymptomatic Elevated Blood Pressure: Defined as SBP/DBP ≥130/80 mm Hg without evidence of organ involvement. Although less severe, sustained elevations can lead to long-term complications and warrant management.

It is important to note that target-organ damage can occur even at lower blood pressure thresholds, affecting vital organs such as the brain, heart, kidneys, arteries, and retina [3].

58.3 Malignant Hypertension

Malignant hypertension is a severe form of hypertension with distinct vascular pathology, including fibrinoid necrosis of arterioles and small arteries, particularly in the kidneys, and severe hypertensive retinopathy. This condition signifies advanced vascular damage and is associated with high morbidity and mortality. Prompt recognition and aggressive management are imperative to prevent irreversible organ damage.

58.4 Mechanisms and Triggers of Hypertensive Crises

Understanding the underlying mechanisms and triggers of hypertensive crises in the ICU is essential for prevention and management.

58.4.1 Pathophysiology and Triggers

Hypertensive crises may be precipitated by:

- Noncompliance with Antihypertensive Therapy: Sudden cessation or irregular intake of antihypertensive medications can lead to rebound hypertension.
- Use of Sympathomimetic Drugs: Substances like cocaine, amphetamines, or certain over-the-counter medications can induce severe hypertension.
- Acute Physical Stressors: Conditions such as infections, surgical procedures, pain, or agitation can elevate blood pressure due to increased sympathetic activity.

58.4.2 Pathogenesis of End-Organ Damage

In hypertensive emergencies, increased vascular wall stress leads to endothelial injury, initiating a cascade of pathological events:

- Endothelial Dysfunction: Mechanical stress damages the endothelium, disrupting its barrier function and promoting inflammation.
- Inflammatory Response: Endothelial injury triggers the release of inflammatory mediators, leading to leukocyte adhesion and further vascular damage.
- Increased Vascular Permeability: Damage to the endothelium increases permeability, resulting in edema and microhemorrhages within organs.
- Hypoperfusion and Ischemia: The combination of vasoconstriction and vascular injury leads to reduced blood flow and oxygen delivery to vital organs.
- Fibrinoid Necrosis: Particularly noted in malignant hypertension, fibrinoid necrosis involves the deposition of fibrin-like material in the vessel walls, leading to vessel obstruction and organ ischemia.

Understanding these mechanisms underscores the importance of prompt and effective blood pressure control to prevent irreversible organ damage.

58.5 Assessment of End-Organ Damage

A comprehensive evaluation of target-organ damage is critical in patients with hypertensive emergencies. The mnemonic BARKH serves as a useful tool to assess the affected organs:

- Brain: Assess for hypertensive encephalopathy, acute ischemic stroke (AIS), posterior reversible encephalopathy syndrome (PRES), and intracerebral hemorrhage (ICH).
- Arteries: Evaluate for aortic dissection, preeclampsia, eclampsia, and HELLP (hemolytic anemia, elevated liver enzymes, and low platelet) syndrome.
- Retina: Conduct fundoscopic examinations for signs of hypertensive retinopathy.
- Kidneys: Monitor for acute kidney injury (AKI) and thrombotic microangiopathy.
- Heart: Investigate for acute heart failure, pulmonary edema, and acute coronary syndromes (ACS).

58.6 Diagnostic Evaluation

Specific imaging and laboratory tests are essential to confirm end-organ involvement:

- Neurological Assessment: Utilize computed tomography (CT) or magnetic resonance imaging (MRI) to detect cerebral edema, infarcts, or hemorrhages.
- Cardiac Evaluation: Perform electrocardiograms (ECG) and measure cardiac biomarkers (e.g., troponin levels) to identify myocardial ischemia or infarction.

- Renal Function Tests: Assess serum creatinine, blood urea nitrogen (BUN), and perform urinalysis to evaluate kidney function.
- Ophthalmologic Examination: A fundoscopic exam can reveal retinal hemorrhages, exudates, or papilledema indicative of hypertensive retinopathy [4].

58.7 Target Blood Pressure Reduction Strategies

Effective management of hypertensive emergencies involves careful and controlled reduction of blood pressure to prevent further organ damage while avoiding hypoperfusion.

58.7.1 Organ-Specific Blood Pressure Goals

- Brain (e.g., Hypertensive Encephalopathy): Aim to reduce diastolic blood pressure to 100–110 mm Hg within the first few hours. This target helps stabilize cerebral perfusion while avoiding hypoperfusion-related injury.
  - AIS: Reduces MAP by 15%. SBP between 130 and 180 mm Hg.
  - ICH: Reduces MAP by 15% in the first hour.
  - PRES: MAP declines by 20–25% as early as possible.
- Aortic Dissection: Rapidly lower SBP to less than 120 mm Hg and heart rate below 60 beats per minute to minimize aortic wall stress.
  - Pulmonary Edema and ACS: Aim for an SBP of less than 140 mm Hg to decrease cardiac workload and improve oxygenation in. Target SBP < 180 mm Hg or reduce MAP by 25% in the first hour; target 160/100 mm Hg in next 2–6 hours if stable, then to normal in 24–48 hours in acute heart failure.
- Preeclampsia, HELLP, and Eclampsia: Reduce SBP to less than 160 mm Hg and DBP to less than 105 mm Hg to prevent maternal and fetal complications.
- Retina:
  - Target SBP <180 mm Hg/ MAP reduction by 15%.
  - Kidney:
  - Gradual reduction of MAP by 20–25%.

58.7.2 Caution Against Overly Rapid Reduction

Excessive and rapid lowering of blood pressure can lead to ischemic complications, particularly in the brain and heart. Cerebral autoregulation may be compromised, increasing the risk of cerebral hypoperfusion and subsequent infarction. Therefore, gradual reduction with close monitoring is essential.

58.8 Management Approach: Assess, Identify, Modify (AIM)

An effective management strategy can be encapsulated in the AIM framework:

- Assess: Obtain accurate blood pressure measurements and evaluate the severity of hypertension and presence of end-organ damage. Ensure correct reading, assess volume status, review home medications (e.g., abrupt stoppage of beta blockers) are some of the steps to be followed.
- Identify: Determine the underlying cause and affected organ systems through clinical evaluation and diagnostic testing.
- Modify: Implement appropriate pharmacological interventions and address modifiable factors contributing to hypertension.

58.9 Monitoring and Device Accuracy

In hypertensive emergencies, precise blood pressure monitoring is critical.

- Arterial Lines: Use intra-arterial lines for continuous and accurate measurements, which are essential for titrating intravenous antihypertensive medications.
- Noninvasive Monitoring: For patients without arterial lines, blood pressure should be measured every 15–30 min initially. Utilize automated blood pressure devices with appropriate cuff sizes, and confirm readings manually to ensure accuracy, especially in critically unstable patients.

58.10 Drug Titration

Careful titration of antihypertensive medications is crucial to achieve target blood pressure goals without causing rapid reductions that may lead to hypoperfusion.

- Start Low, Go Slow: Initiate therapy with lower doses and adjust gradually based on the patient's response and tolerability.
- Frequent Monitoring: Reassess blood pressure at appropriate intervals (e.g., every 5–15 min during titration) to guide dosage adjustments.
- Individualized Approach: Consider patient-specific factors such as age, comorbidities, and the presence of organ dysfunction when titrating medications [5].

58.11 Drug Choices Based on Organ Impact

Selection of antihypertensive agents should be tailored to the patient's specific clinical scenario and target-organ involvement.

58.11.1 Brain-Related Crises

Labetalol: An intravenous beta-blocker that offers controlled reduction of blood pressure without significant effects on cerebral blood flow.

- Dosage and Titration: Start with 20 mg IV over 2 min. Additional doses of 20–80 mg can be administered every 10 min until target blood pressure is achieved, or a continuous infusion of 1–8 mg/min can be used. Monitor blood pressure every 5–10 min during titration to avoid rapid drops. (European) Initial 0.3–1.0-mg/kg dose (maximum 20 mg), slow IV injection every 10 min or 0.4–1.0-mg/kg/h IV infusion up to 3 mg/kg/h. Adjust rate up to total cumulative dose of 300 mg. This dose can be repeated every 4–6 h. (AHA 2017).

Nicardipine: A calcium channel blocker effective in lowering blood pressure with the advantage of preserving cerebral autoregulation.

- Dosage and Titration: Initiate at 5 mg/hour IV infusion, increasing by 2.5 mg/hour every 5–15 min as needed, up to a maximum of 15 mg/hour. Adjust titration intervals carefully to prevent overshooting the target blood pressure.

58.11.2 Heart-Related Crises

Nitroglycerin (NTG): Ideal for patients with ACS or pulmonary edema, as it reduces preload and afterload, improving myocardial oxygen supply and decreasing cardiac workload.

- Dosage and Titration: Start at 5 mcg/min IV infusion, increasing by 5 mcg/min every 3 to 5 min until desired blood pressure or symptom relief is achieved, to a maximum of 20 mcg/min. Frequent monitoring is necessary due to its rapid onset and short duration of action.

Avoidance of Nitroprusside: Due to risks of cyanide toxicity and potential worsening of renal function, nitroprusside is generally avoided, especially in patients with renal impairment. However, it can be used for acute pulmonary edema. Initial 0.3–0.5 mcg/kg/min; increase in increments of 0.5 mcg/kg/min to achieve BP target; maximum dose 10 mcg/ kg/min; duration of treatment as short as possible. For infusion rates ≥4–10 mcg/kg/min or duration >30 min, thiosulfate can be co-administered to prevent cyanide toxicity.

Esmolol: is also the agent of choice along with NTG for ACS.

- Dosage and titration: Loading dose 500–1000 mcg/kg/min over 1 min followed by a 50-mcg/kg/min infusion. For additional dosing, the bolus dose is repeated and the infusion increased in 50-mcg/kg/min increments as needed to a maximum of 200 mcg/kg/min.
- Caution: Avoid in bradycardia, heart block, decompensated heart failure.

58.11.3 Preeclampsia and Eclampsia Management

In pregnant patients with hypertensive crises:
Magnesium Sulfate: First-line therapy for the prevention and treatment of eclampsia seizures.

- Dosage: Administer a loading dose of 4–6 grams IV over 15–20 min, followed by a maintenance infusion of 1–2 grams per hour.

Blood Pressure Control: Use hydralazine or labetalol for acute blood pressure management.

- Hydralazine Dosage: Start with 5–10 mg IV over 2 min, repeat every 20–30 min as needed. Initial 10 mg via slow IV infusion (maximum initial dose 20 mg); repeat every 4–6 h as needed. Unpredictable response and long duration of action go against its use as a first-line agent.
- Labetalol Dosage: Begin with 20 mg IV over 2 min, followed by doses of 40 mg and then 80 mg at 10-min intervals if needed, up to a total of 220 mg. Initial 0.3–1.0-mg/kg dose (maximum 20 mg), slow IV injection every 10 min or 0.4–1.0-mg/kg/h IV infusion up to 3 mg/kg/h. Adjust rate up to a total cumulative dose of 300 mg. This dose can be repeated every 4–6 h.
- Agents to Avoid: ACE inhibitors and ARBs are contraindicated due to teratogenic risks and adverse effects on fetal renal development.

58.11.4 Renal Protection

In patients with renal compromise:

- Preferred Agents: Labetalol and nicardipine are safer options due to minimal nephrotoxicity.
- Agents to Avoid: Nitroprusside should be avoided because its metabolites (thiocyanate and cyanide) can accumulate in renal failure, leading to toxicity.

58.11.5 Alternative Medications for Special Cases

Clevidipine: An ultra-short-acting dihydropyridine calcium channel blocker suitable for rapid titration and blood pressure control, especially when beta-blockers are contraindicated.

- Dosage and Titration: Start at 1–2 mg/hour IV infusion, doubling the dose at short intervals (90 s) initially, then increasing by less than double every 5–10 min as needed. Maximum recommended dose is 32 mg/hour.

Esmolol: A short-acting beta-blocker beneficial in situations requiring quick onset and offset of action, such as in perioperative hypertension or tachyarrhythmias.

- Dosage and Titration: Begin with a 500 mcg/kg IV bolus over 1 min, followed by an infusion of 50 mcg/kg/min. Adjust infusion rate by 25–50 mcg/kg/min every 5–10 min as needed, up to a maximum of 300 mcg/kg/min.

58.11.6 Patient-Specific Considerations

- Chronic Obstructive Pulmonary Disease (COPD): Beta-blockers may exacerbate bronchospasm; thus, agents like nicardipine or clevidipine are preferred.
- Chronic Kidney Disease (CKD): Drugs with minimal renal metabolism and excretion are favored to prevent accumulation and toxicity.
- Heart Failure with Reduced Ejection Fraction (HFrEF): Avoid negative inotropic agents; consider ACE inhibitors or angiotensin receptor blockers (ARBs).
- Heart Failure with Preserved Ejection Fraction (HFpEF): Focus on controlling blood pressure and relieving symptoms without compromising diastolic filling.
- Pregnancy: Use labetalol or hydralazine for blood pressure control; avoid ACE inhibitors and ARBs due to teratogenic risks.

Tables summarizing medications, dosages, and special considerations for various organ systems can provide quick reference for clinicians (Tables 58.1 and 58.2).

Table 58.1 Medications for rapid blood pressure control in organ crises

Organ system	Preferred medications	Dosage guidelines
Brain (e.g., intracerebral hemorrhage)	Labetalol, nicardipine	Labetalol: 20 mg IV over 2 min; repeat every 10 min or infusion 1–8 mg/min. Nicardipine: Start at 5 mg/h, increase by 2.5 mg/h every 5–15 min
Heart (ACS, pulmonary edema)	Nitroglycerin, labetalol	Nitroglycerin: Start at 5mcg/min, increase by 5 mcg/min every 3–5 min. Labetalol: 20 mg IV, may repeat every 10 min based on response
Arteries (aortic dissection)	Esmolol, labetalol	Esmolol: 500 mcg/kg bolus, then 50 mcg/kg/min infusion, titrate as needed. Labetalol: Similar dosages as in brain crises. Beta blockade should precede vasodilator use if any needed (nicardipine/nitroprusside)
Kidneys (AKI)	Labetalol, nicardipine	As per brain crisis dosages; avoid agents with nephrotoxic potential
Retina (hypertensive retinopathy)	Nicardipine, labetalol	Follow brain crisis dosing to prevent worsening retinopathy
Pregnancy (severe preeclampsia, eclampsia)	Labetalol, hydralazine, magnesium sulfate	Labetalol: 20 mg IV over 2 min; repeat as needed. Hydralazine: 5–10 mg IV over 2 min; may repeat every 20–30 min. Magnesium sulfate: Loading dose of 4–6 g IV over 15–20 min, maintenance 1–2 g/h

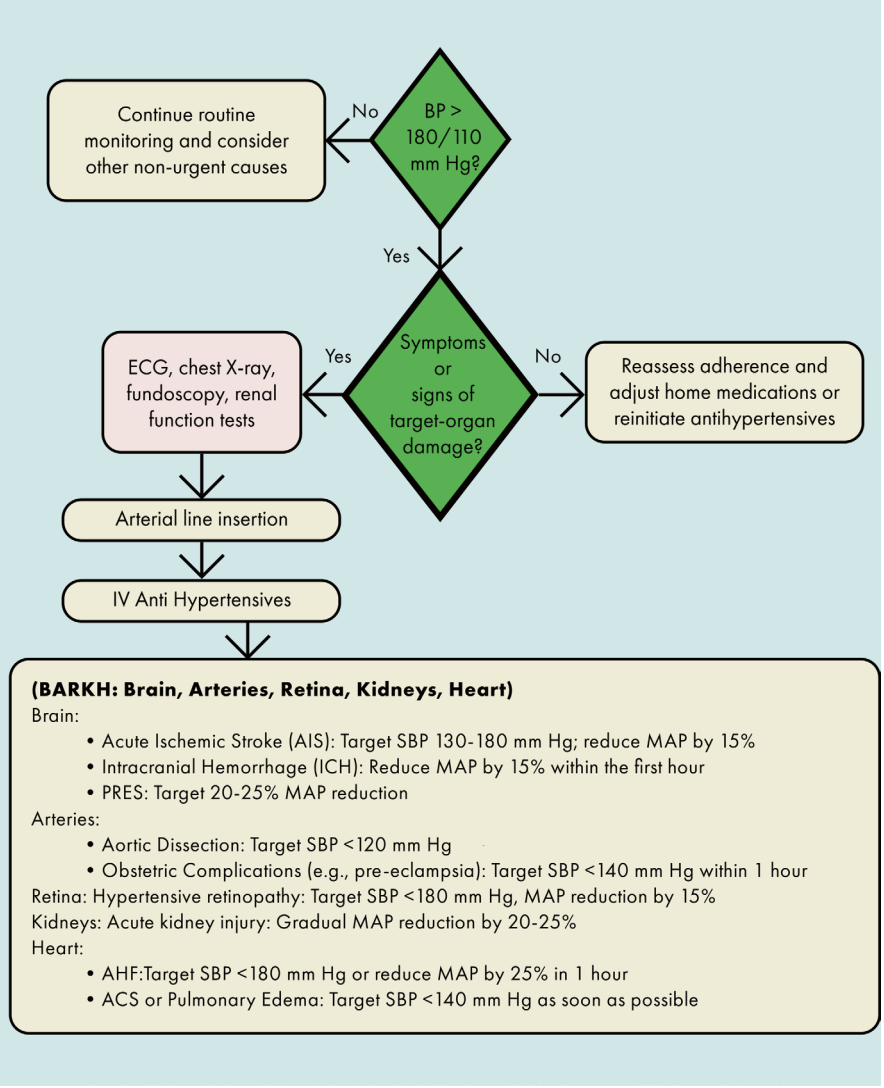
Table 58.2 Special considerations in comorbid conditions

Condition	Preferred agents	Agents to avoid
COPD	Nicardipine, clevidipine	Beta-blockers due to risk of bronchospasm
Chronic kidney disease (CKD)	Labetalol, nicardipine	Nitroprusside (risk of thiocyanate/cyanide toxicity)
Heart failure with reduced ejection fraction (HFrEF)	ACE inhibitors, ARBs	Calcium channel blockers due to negative inotropy
Heart failure with preserved ejection fraction (HFpEF)	Diuretics, ACE inhibitors	Avoid negative inotropic agents like beta-blockers
Pregnancy	Labetalol, hydralazine	ACE inhibitors, ARBs, due to fetal toxicity

58.12 Conclusion

Managing hypertension in the ICU requires a comprehensive and systematic approach that prioritizes patient safety and optimal outcomes. By focusing on accurate assessment, individualized treatment strategies, and careful monitoring, healthcare providers can effectively address hypertensive crises. Incorporating organ-specific management plans, providing detailed titration guidance, and ensuring proper transition to post-acute care are essential components of high-quality care. Including practical case scenarios enhances understanding and application of these principles. Adherence to the latest clinical guidelines and evidence-based practices will improve patient outcomes and reduce the risk of long-term complications associated with hypertension. By integrating these refinements, the chapter provides ICU clinicians with a precise, actionable guide for managing hypertensive crises, balancing rapid intervention with careful titration to avoid complications.

Algorithm 58.1: Approach to acute severe hypertension in the ICU



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