

Pressure Matters: Blood Pressure Management in Stroke



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Objectives

- Review cerebral autoregulation in acute stroke
- Discuss physiologic rationale behind BP targets
- Apply AHA/ASA BP recommendations across stroke subtypes
- Review antihypertensive selection in neurocritical care
- Discuss thrombectomy-era BP management controversies
- Apply evidence-based management to complex cases



Why Blood Pressure Matters in Stroke

Blood pressure management directly impacts:

- Cerebral perfusion
- Infarct expansion
- Hematoma expansion
- Edema formation
- Hemorrhagic transformation
- Reperfusion injury

Stroke BP management is not “one-size-fits-all.” Targets vary based on:

- Stroke subtype
- Reperfusion status
- ICP concerns
- Chronic hypertension history
- Vessel recanalization

Cerebral Autoregulation



Normally the brain self-regulates blood flow.



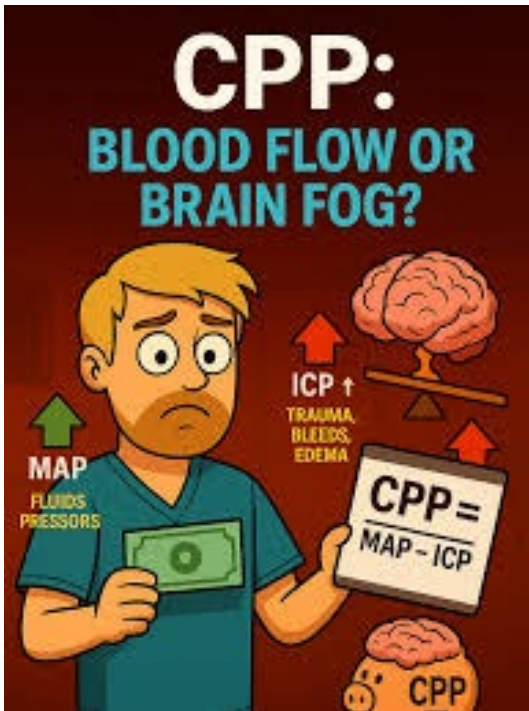
In stroke:

Autoregulation impaired
Brain becomes pressure
dependent



This is why aggressive lowering can be
dangerous.

Understanding CPP



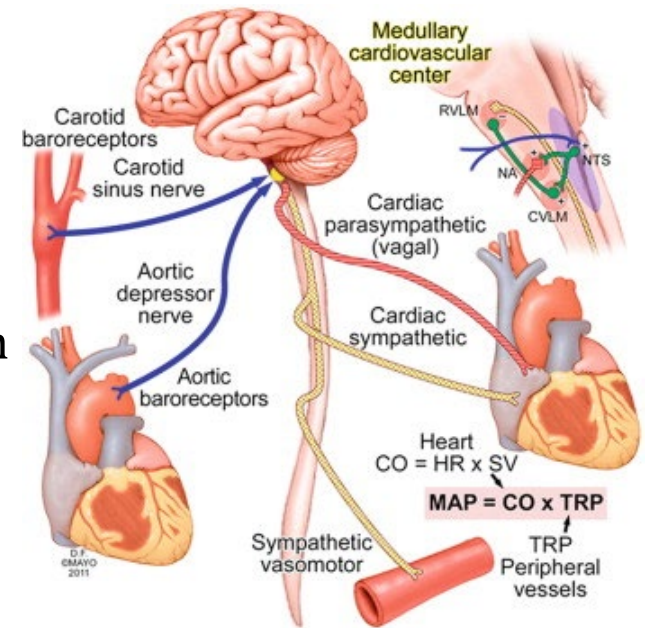
Cerebral Perfusion Pressure

- Formula:
 - $CPP = MAP - ICP$
 - Rising ICP lowers CPP
 - Lower systemic BP lowers CPP
- **Why this matters:** Excessive BP reduction may critically reduce penumbral perfusion.

MAP in Stroke Care

Mean Arterial Pressure

- Define:
 - This is the average pressure in a patient's arteries during one cardiac cycle, generally considered a better indicator of organ perfusion than systolic blood pressure.
- $MAP = \{SBP + 2(DBP)\} / \{3\}$
- MAP often better reflects organ perfusion than systolic BP alone.
- Particularly relevant in:
 - Large vessel occlusion
 - Critical carotid disease
 - Elevated ICP
 - Sedated/intubated thrombectomy patients



Acute Ischemic Stroke (No Thrombolytics)

- **Permissive Hypertension**
- Per AHA/ASA:
- If NOT receiving TNK/tPA or EVT:
- Treat only if:
 - **>220/120** Lower by ~15% in first 24 hours.
- Why?
Preserve collateral flow to ischemic penumbra.

Ischemic Stroke Receiving TNK/tPA

- Before thrombolytic:
Must be $<185/110$
- After thrombolytic:
Maintain $<180/105$ for 24 hrs
- Reason: Reduce risk of symptomatic intracranial hemorrhage.



Why 185/110 → 180/105?

This reflects:

- Post-thrombolytic endothelial fragility
- Increased hemorrhagic transformation risk
- Reperfusion-associated vascular injury

Even modest elevations may increase bleeding risk after thrombolysis.



Mechanical Thrombectomy

BP in the EVT Era

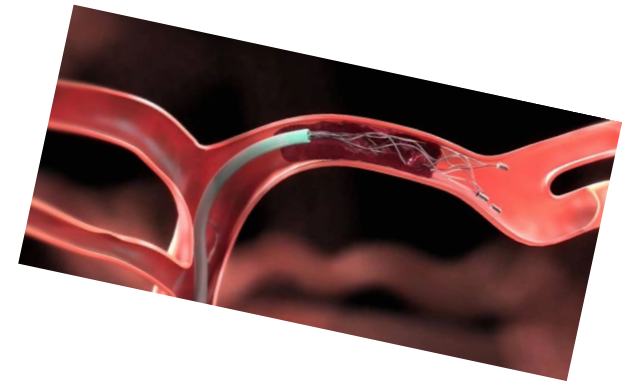
Pre-procedure:

- Avoid hypotension
- Preserve collateral circulation

Post-thrombectomy:

Common targets:

- $<180/105$
- Some centers: SBP <160
- Select cases: SBP <140 after TICI 2B/3 reperfusion



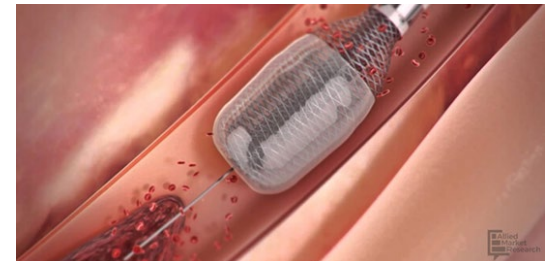
Thrombectomy Controversies

Emerging evidence suggests:

- Higher BP after successful reperfusion may worsen edema and hemorrhagic conversion
- Excessively low BP may worsen infarct growth if reperfusion incomplete

BP management increasingly individualized based on:

- TICI score
- Core infarct size
- Edema risk
- Chronic hypertension



Intracerebral Hemorrhage

AHA Recommendations

If presenting SBP:

150–220

Acute lowering to:

~140 mmHg is safe and reasonable

Goals:

- Prevent hematoma expansion
- Reduce secondary injury
- Avoid cerebral hypoperfusion



ICH Pathophysiology

Elevated BP may contribute to:

- Ongoing vascular disruption
- Hematoma expansion
- Elevated ICP
- Perihematomal edema

Most hematoma expansion occurs early:

First 6 hours are critical.

Subarachnoid Hemorrhage

Before aneurysm securement:

SBP <140

Common target:

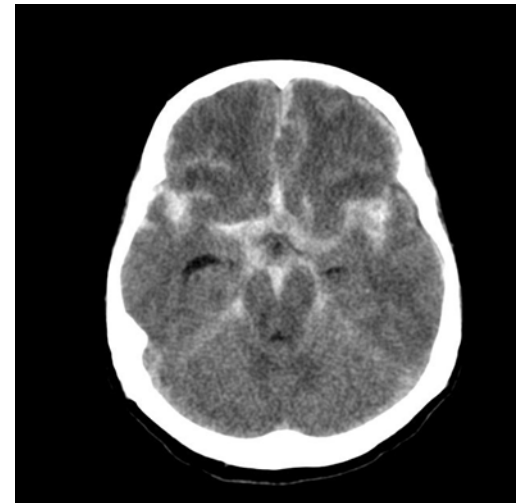
SBP <160

Goal:

- Reduce rebleeding risk
- Preserve cerebral perfusion

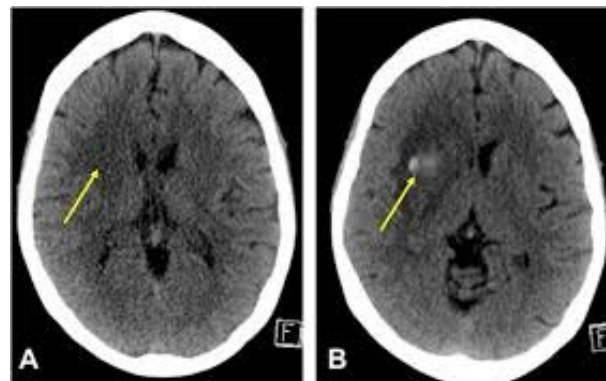
After aneurysm treatment:

- BP goals individualized
- Vasospasm management may require permissive hypertension



Hemorrhagic Transformation

- Acute ischemic stroke can convert to hemorrhage.
- Risk increases with:
 - Severe hypertension
 - Large infarcts
 - Thrombolytics
 - Reperfusion after thrombectomy
- **This is why post-TNK BP control matters.**



Antihypertensive Selection

Ideal neurocritical care agent:

- Rapid onset
- Easily titratable
- Predictable response
- Minimal ICP effect
- Limited cerebral vasodilation



Medications Commonly Used

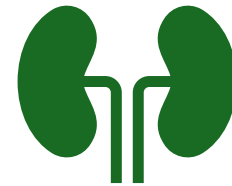


IV Push

Labetalol

Hydralazine (less ideal)

Metoprolol (less common)



Continuous Infusions

Nicardipine

Clevidipine (Cleviprex)

Esmolol

Nicardipine (Cardene)

Nicardipine (Cardene)



Start:



5 mg/hr IV drip



Increase by 2.5 mg/hr q5 min



Max usually 15 mg/hr



Pros:

Smooth titration
Reliable onset
Favorable neuro profile



Watch:

Reflex tachycardia
Phlebitis
Large-volume infusions

Clevidipine



Ultra-short acting calcium channel blocker



Start: 1–2 mg/hr



Very rapid on/off



Great for:

Very rapid onset/offset

Tight BP control

Excellent for procedural settings



Avoid:

Soy/egg allergy

Lipid restrictions

Cost

Labetalol



Dose:



10–20 mg IV over 1–2 min
May repeat or double q10 min



Pros:

Fast onset
Easy push option



Avoid:

Bradycardia
CHF
Asthma/COPD severe bronchospasm

Common Clinical Pitfalls

- Overcorrecting ischemic stroke BP
- Delayed treatment before thrombolytic administration
- Failure to reassess neuro exam after BP reduction
- Hypotension during thrombectomy
- Restarting antihypertensives prematurely
- Treating numbers instead of physiology

Case Study 1

72 y/o LVO

PMH of HTN, A-fib, Tobacco user

BP 210/104

LKW 7 hours prior to arrival
Not a TNK candidate

Awaiting thrombectomy

Question:

Would you aggressively lower BP?

Discussion:

- Avoid hypotension
- Preserve collateral circulation
- Individualized reduction strategy



Case Study 2

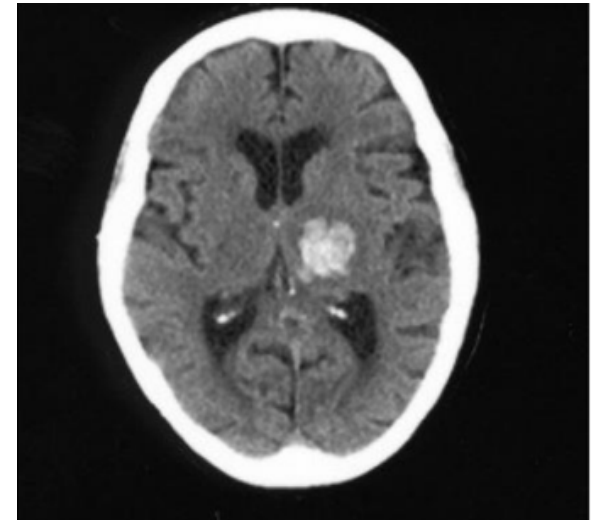
68 y/o basal ganglia ICH with past medical history of HTN and Tobacco use

BP 238/122

Question:
Best next step?

Management considerations:

- Rapid titratable infusion
- Goal ~140
- Repeat imaging
- ICP monitoring considerations



Case Study #3

65 y/o female with PMH of HTN and HLD

LKW is 1 hour prior to arrival

Neuro deems a TNK candidate

BP 196/112

What do you want to do?

Action:

Lower to <185/110 before thrombolytic.

Use labetalol, cleviprex, or nicardipine.





Key takeaways

- BP management in stroke is physiology-driven
- Ischemic stroke often requires permissive hypertension
- Hemorrhagic stroke benefits from controlled reduction
- Post-thrombectomy management remains evolving
- Avoid extremes
- Reassess neurologic status continuously

Stroke BP Management

Precision Over Protocol

The goal is not simply lowering pressure.

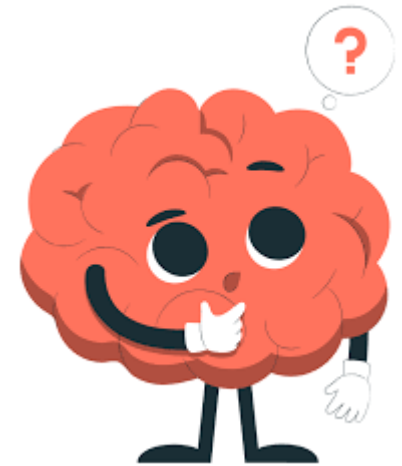
The goal is:

- preserving perfusion,
- minimizing secondary injury,
- and optimizing neurologic outcome.

Questions?

Remember:

“Time is Brain... but Perfusion is Everything.”



References

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