

HyperTensia 2025



Pathophysiology of Hypertension

Hypertension (HTN) is a chronic medical condition characterized by persistently elevated blood pressure (BP \geq 140/90 mmHg). Its pathophysiology involves complex interactions between the nervous system, renal system, vascular endothelium, and hormonal mechanisms.

- 1. Key Mechanisms in Hypertension Development
- A. Increased Sympathetic Nervous System (SNS) Activity
- ✓ Stress, obesity, and genetics can cause hyperactivation of the SNS, leading to:
 - Vasoconstriction (narrowing of blood vessels) → Increased peripheral resistance
 - Increased heart rate & cardiac output
- Overstimulation of $\beta1$ -receptors in the heart \rightarrow Increased BP Example: Chronic stress \rightarrow Excess norepinephrine \rightarrow Persistent BP elevation
- B. Renin-Angiotensin-Aldosterone System (RAAS) Overactivation
- ✓ RAAS plays a crucial role in blood pressure regulation through fluid balance & vasoconstriction.
- ✓ Overactivation leads to:
 - \uparrow Renin release (kidney) \rightarrow \uparrow Angiotensin II \rightarrow Vasoconstriction
 - ↑ Aldosterone → Sodium & water retention → Increased blood volume

Example: Kidney ischemia \rightarrow Renin release \rightarrow Angiotensin II surge \rightarrow Chronic hypertension

- C. Endothelial Dysfunction & Reduced Nitric Oxide (NO) Production
- ✓ Endothelium-derived NO is a natural vasodilator.
- ✓ In HTN, there is \downarrow NO & \uparrow endothelin-1 (vasoconstrictor), leading to:
 - Arterial stiffness

Hypertension

- Increased vascular resistance
- Chronic inflammation → Atherosclerosis → Worsening hypertension
 Example: Diabetes & smoking → Oxidative stress → Endothelial damage →



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D. Sodium Retention & Fluid Overload

- ✓ Excess dietary salt intake leads to:
 - Water retention → Increased blood volume → Increased cardiac output
 - Reduced sodium excretion (in salt-sensitive individuals)

Example: High-sodium diet → Increased plasma volume → Sustained BP elevation

E. Hormonal & Metabolic Factors

- ✓ Insulin Resistance & Obesity
 - Hyperinsulinemia → Activates SNS & RAAS → Increases BP
 ✓ Leptin & Adipokines (From Fat Cells)
 - Leptin 个 SNS activity & BP
- Adiponectin ↓ in obesity → Less NO production → Vasoconstriction
 Example: Obesity → ↑ Leptin, ↓ Adiponectin → Vasoconstriction →
 Hypertension

F. Genetic & Environmental Influences

- √ Family history of hypertension increases risk.
- ✓ Epigenetic changes due to lifestyle (smoking, alcohol, stress, diet) contribute to vascular dysfunction.

2. Long-Term Effects of Hypertension

✓ Heart: Left Ventricular Hypertrophy (LVH), Heart Failure, Coronary Artery Disease

✓ Brain: Stroke, Cognitive Decline

√ Kidneys: Chronic Kidney Disease (CKD)

✓ Eyes: Hypertensive Retinopathy

✓ Vessels: Aneurysms, Peripheral Artery Disease

Conclusion

✓ Hypertension is a multi-factorial disease involving SNS overactivity, RAAS dysfunction, endothelial damage, and sodium imbalance.



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- ✓ Uncontrolled HTN leads to organ damage, making early intervention crucial.
- ✓ Treatment involves lifestyle changes + antihypertensive medications targeting specific mechanisms.