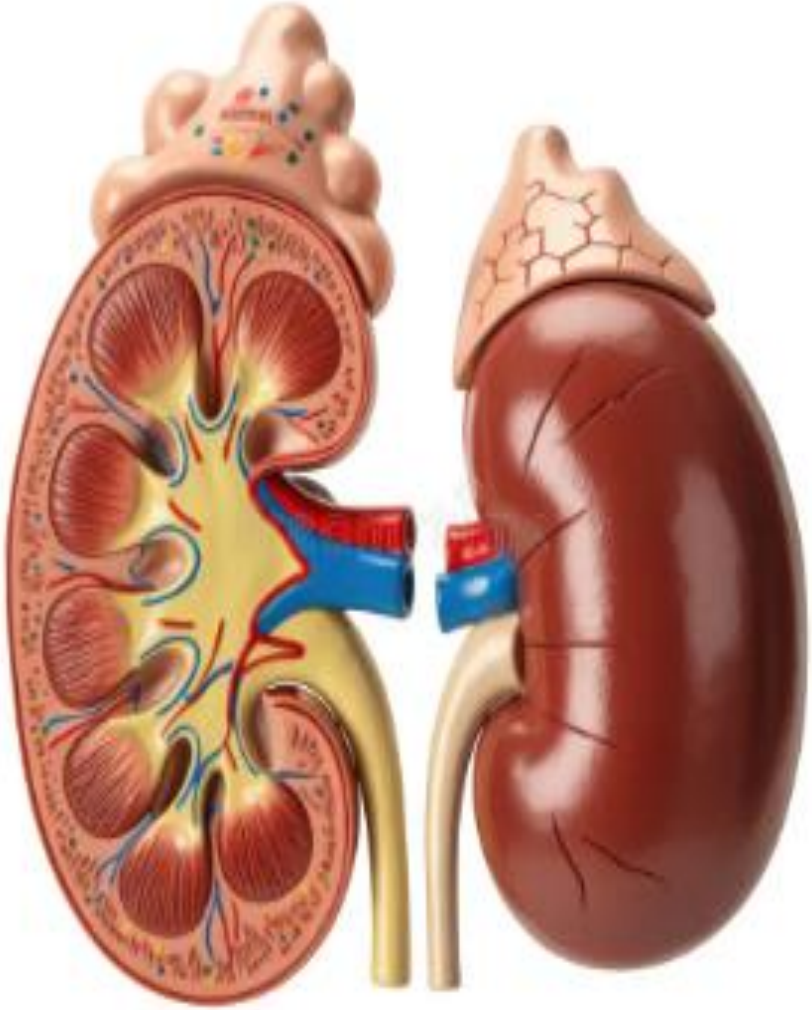
Adrenal gland

Advanced Medicine 2

4th Stage – Anesthesia Techniques

Lec: 2

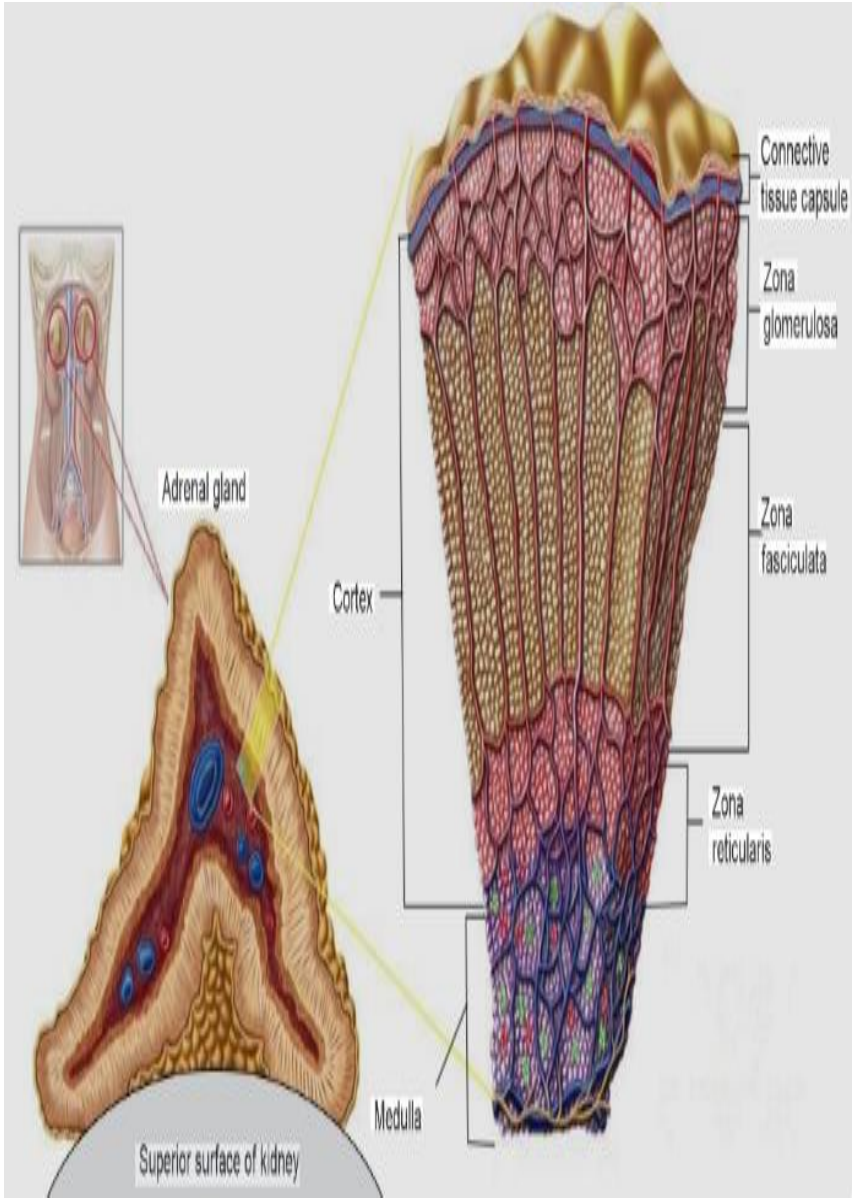
2nd Course



Dr. Bashar Hadi Alaaraji

Adrenal Gland

- The adrenal glands (suprarenal glands) are paired endocrine organs situated atop the kidneys. Each gland functions as two distinct endocrine organs in one, comprising an outer cortex [Zona Glomerulosa (outer); Zona Fasciculata (middle); Zona Reticularis (inner)] and an inner Medulla.



Functions by Zones

A. Zona Glomerulosa: Salt & Pressure

- Product: Aldosterone (mineralocorticoid)
- Stimuli: ↑K^+, Angiotensin II, ↓Na^+/volume **(NOT ACTH!)**
- Mechanism: Mineralocorticoid receptors → ENaC channels → Na^+ reabsorption, K^+ excretion

B. Zona Fasciculata: Stress & Metabolism

- Product: Cortisol (glucocorticoid)
- Regulation: Hypothalamic-pituitary-adrenal (HPA) axis
  - ✓ Stress → CRH → ACTH → Cortisol → negative feedback
  - ✓ Actions:
    1. Metabolic: Gluconeogenesis, lipolysis, proteolysis (↑blood sugar)
    2. Anti-inflammatory: Inhibits phospholipase A_2, ↓COX-2, ↓cytokines (IL-1, IL-6, TNF-α)
    3. Immunosuppressive: Lymphocyte apoptosis, thymic involution
    4. Permissive: Potentiates catecholamines on vessels

C. Zona Reticularis: Secondary Sexual Characteristics

- Product: DHEA, androstenedione (weak androgens)
- Role: Adrenarche (pubic/axillary hair in puberty), estrogen precursor in postmenopausal women
- Clinical: Excess → hirsutism, virilization

D.Medulla: Fight or Flight

- Cells: Chromaffin (modified postganglionic sympathetic neurons)
- Innervation: Preganglionic sympathetic fibers (unusual—usually postganglionic innervate effectors)
- Products: 80% Epinephrine, 20% Norepinephrine, trace dopamine
- Regulation: Direct neural stimulation (NOT hormonal)

Major Clinical Disorders: The "Big Four"

1. **Cushing's Syndrome**

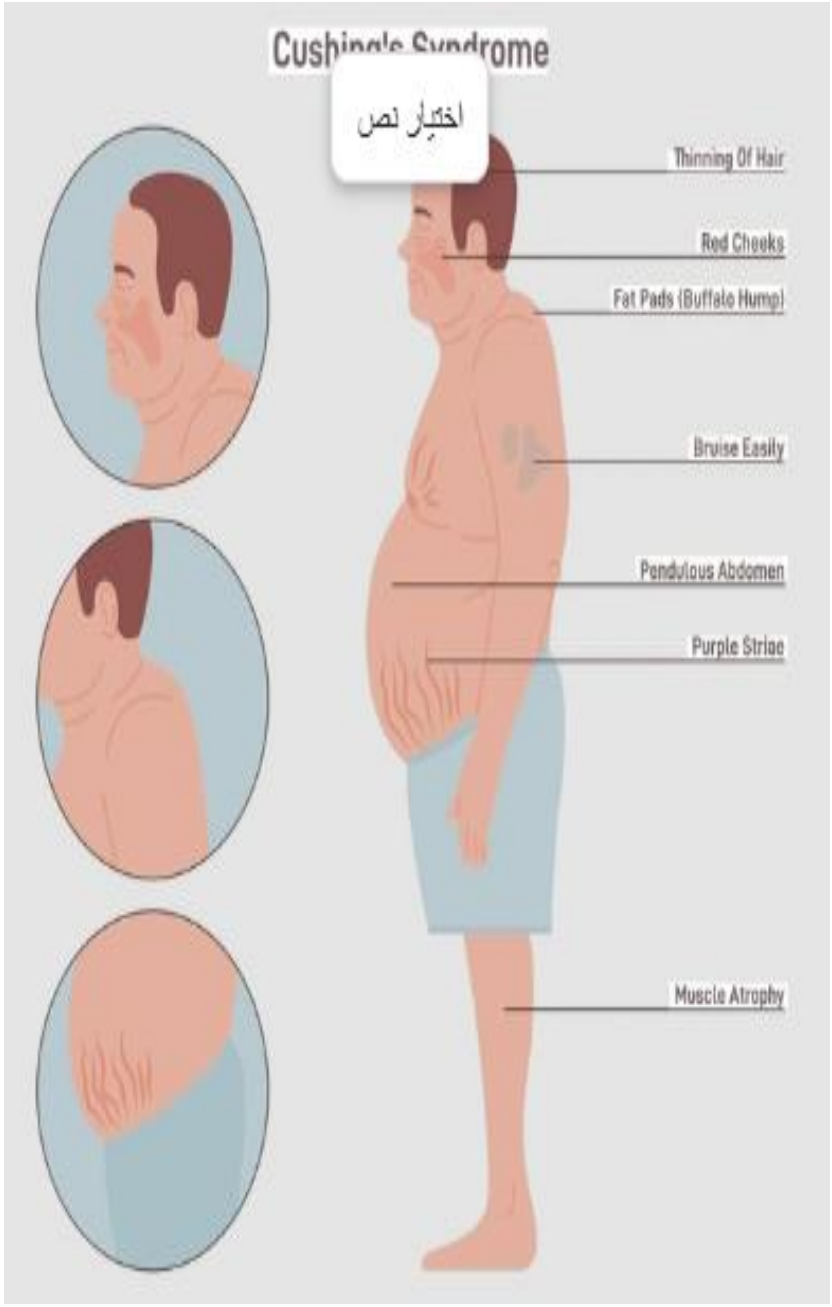
   (Hypercortisolism)

   Causes: **4 Cs**

   1. Corticosteroid drugs (most common)
   2. Cushing's disease (pituitary ACTH adenoma)
   3. Cortical adenoma/carcinoma
   4. Carcinoid/ectopic ACTH (e.g., small cell lung cancer)

Classic Features:

1. Central obesity, moon facies, buffalo hump
2. Proximal muscle weakness (difficulty rising from chair)
3. Purple striae (>1 cm wide), easy bruising
4. Hypertension, glucose intolerance
5. Women: Hirsutism, oligomenorrhea



Diagnosis:

1. Screen: Late-night salivary cortisol **OR** 1 mg overnight dexamethasone suppression test
2. Confirm: Failure to suppress cortisol <1.8 µg/dL → Cushing's confirmed
3. Localize: Measure ACTH
   - ✓ ACTH <10 pg/mL → adrenal source
   - ✓ ACTH >20 pg/mL → pituitary vs ectopic (high-dose dexamethasone test, imaging)

2.Addison Disease (Primary Adrenal Insufficiency)

- Def: is the failure of the adrenal cortex to produce adequate cortisol and aldosterone, usually due to autoimmune destruction of the gland.
- Causes:
  - Autoimmune (80% in developed countries) – often with other autoimmune diseases (thyroiditis, T1DM)
  - TB (worldwide leading cause)
  - Hemorrhage (Waterhouse-Friderichsen syndrome in meningococcemia)

Clinical Presentation:

Acute (Addisonian Crisis)	Chronic
Hypotension (refractory to fluids)	Fatigue, weight loss
Nausea/vomiting, abdominal pain	Hyperpigmentation (↑ ACTH/MSH)
Hyponatremia, hyperkalemia	Salt craving
Hypoglycemia	Postural dizziness

ADDISON'S DISEASE

KEY FEATURES

Mnemonic: "ADDISON"

A – Appetite loss & weight loss

D – Darkened skin

D – Dizziness

I – Irritability & fatigue

S – Salt craving / hyponatremia

O – Orthostatic hypotension

N – Nausea, vomiting, abdominal pain

Diagnosis:

- **Gold standard**: ACTH stimulation (cosyntropin) test
  - ✓ Give 250 μg cosyntropin IV → measure cortisol at 30/60 min
  - ✓ Peak <18 μg/dL = adrenal insufficiency
- **Confirmatory**: ↑ ACTH (>100 pg/mL) in primary Adrenal Insufficiency

Treatment:

- Glucocorticoid:

Hydrocortisone 15–25 mg/day (2/3 AM, 1/3 PM)

- Mineralocorticoid:

Fludrocortisone 0.1 mg/day (primary AI only)

- Stress dosing: Double/triple dose for fever/surgery; IM hydrocortisone 100 mg for crisis

3. Primary Hyperaldosteronism (Conn's Syndrome)

- Def: is a disorder of autonomous excess aldosterone secretion from the adrenal zona glomerulosa, independent of the renin-angiotensin system (RAS). Most common curable cause of secondary hypertension (5–10% of hypertensives)
- Etiology:
  - Aldosterone-producing adenoma.
  - Bilateral adrenal hyperplasia.

Primary Hyp[illegible]losteronism

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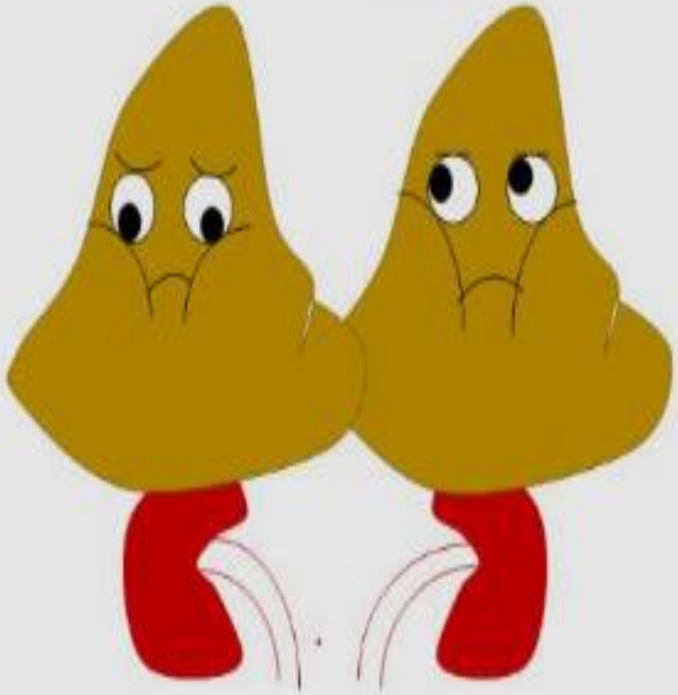
Adrenal Tumor
(Conn's Syndrome)



B/L Adrenal Hyperplasia

CYP11B1-B2
CLCN2 mutation
KCNJ5 mutations
CACNA1H mutation
CACNA1D mutations

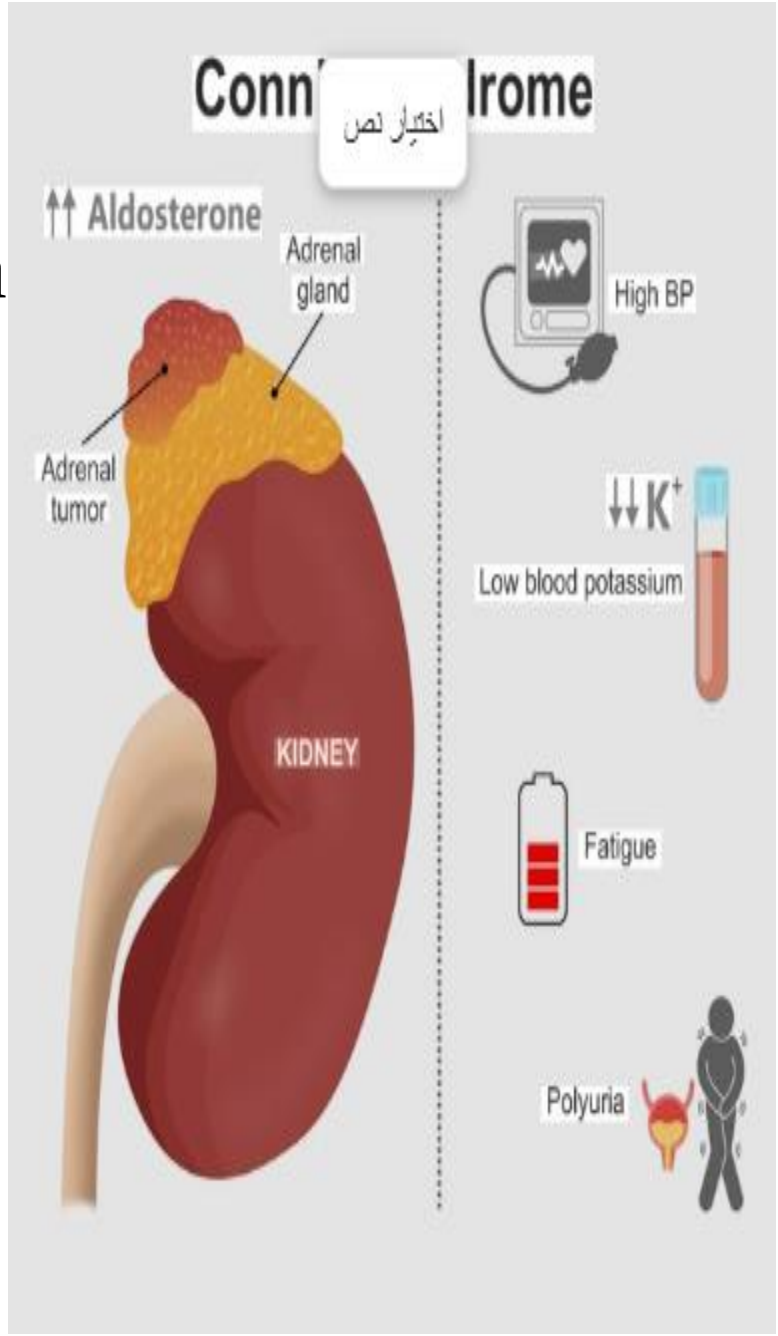
Familial Hyperaldosteronism I-IV
PASNA

Hypertension ↓K^+ ↓Renin

By @drM_sudha

Clinical features:

1. Resistant hypertension (≥3 drugs)
2. Hypokalemia (only in 30–50% – absence does NOT rule it out)
3. Metabolic alkalosis
4. Absence of edema (unlike heart failure/renal disease)



- **Diagnosis**:

1. Screening (in resistant hypertension or hypokalemia).
2. Confirmatory testing (saline suppression test, oral salt loading).
3. Subtype differentiation:
   - ✓ CT/MRI adrenals
   - ✓ Adrenal venous sampling (AVS) - gold standard for lateralization if surgery planned

- **Treatment:**

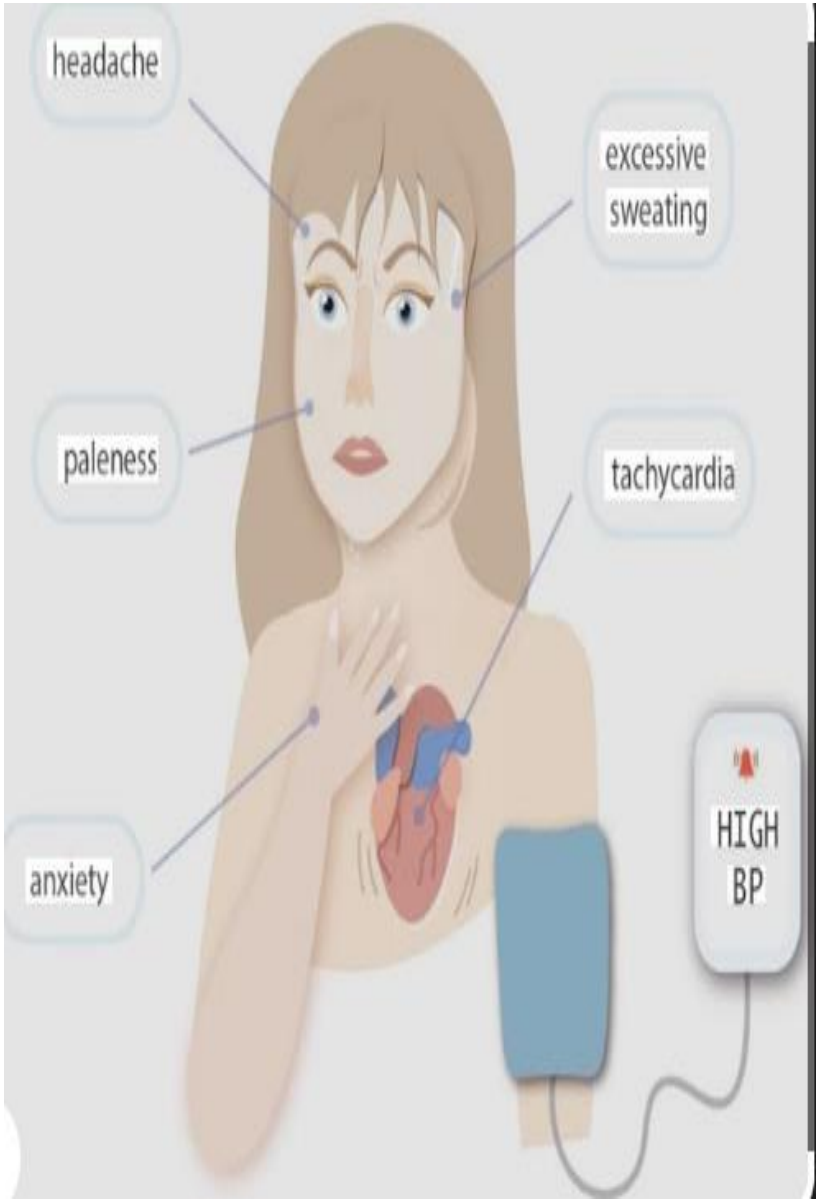
- ✓ Unilateral adenoma → laparoscopic adrenalectomy
- ✓ Bilateralhyperplasia → spironolactone (mineralocorticoid receptor antagonist)

4. PHEOCHROMOCYTOMA

- Def: is a catecholamine-secreting tumor arising from chromaffin cells of the adrenal medulla (or extra-adrenal paraganglia). It produces the classic triad of episodic hypertension, headache, and sweating.

Classic Triad (present in <25% of patients):

1. Episodic headache
2. Diaphoresis (profuse sweating)
3. Palpitations/tachycardia



- Clinical Presentation:
  - ✓ Paroxysmal or sustained hypertension
  - ✓ Pallor (not flushing!)
  - ✓ Anxiety/panic attacks
  - ✓ Weight loss despite normal appetite
- Diagnosis:
  - ✓ 1st line: Plasma-free metanephrines OR 24-hr urine fractionated metanephrines
  - ✓ Imaging: CT/MRI abdomen → functional imaging if metastatic disease suspected.

- Treatment

Preoperative Preparation (Crucial)

- Goal:
- Block catecholamine effects to prevent intraoperative cardiovascular collapse

1. α-blockade first (10-14 days pre-op):
   - Target: Orthostatic hypotension, nasal stuffiness, mild tachycardia (HR 90-100 bpm)
2. β-blockade second (only after full α-blockade):
   - Never give beta-blocker alone (unopposed α-stimulation → hypertensive crisis)
3. Volume expansion:
   - High salt diet + IV fluids pre-op (catecholamine-induced vasoconstriction causes chronic volume depletion)

Thank you