

ADRENAL DISORDERS

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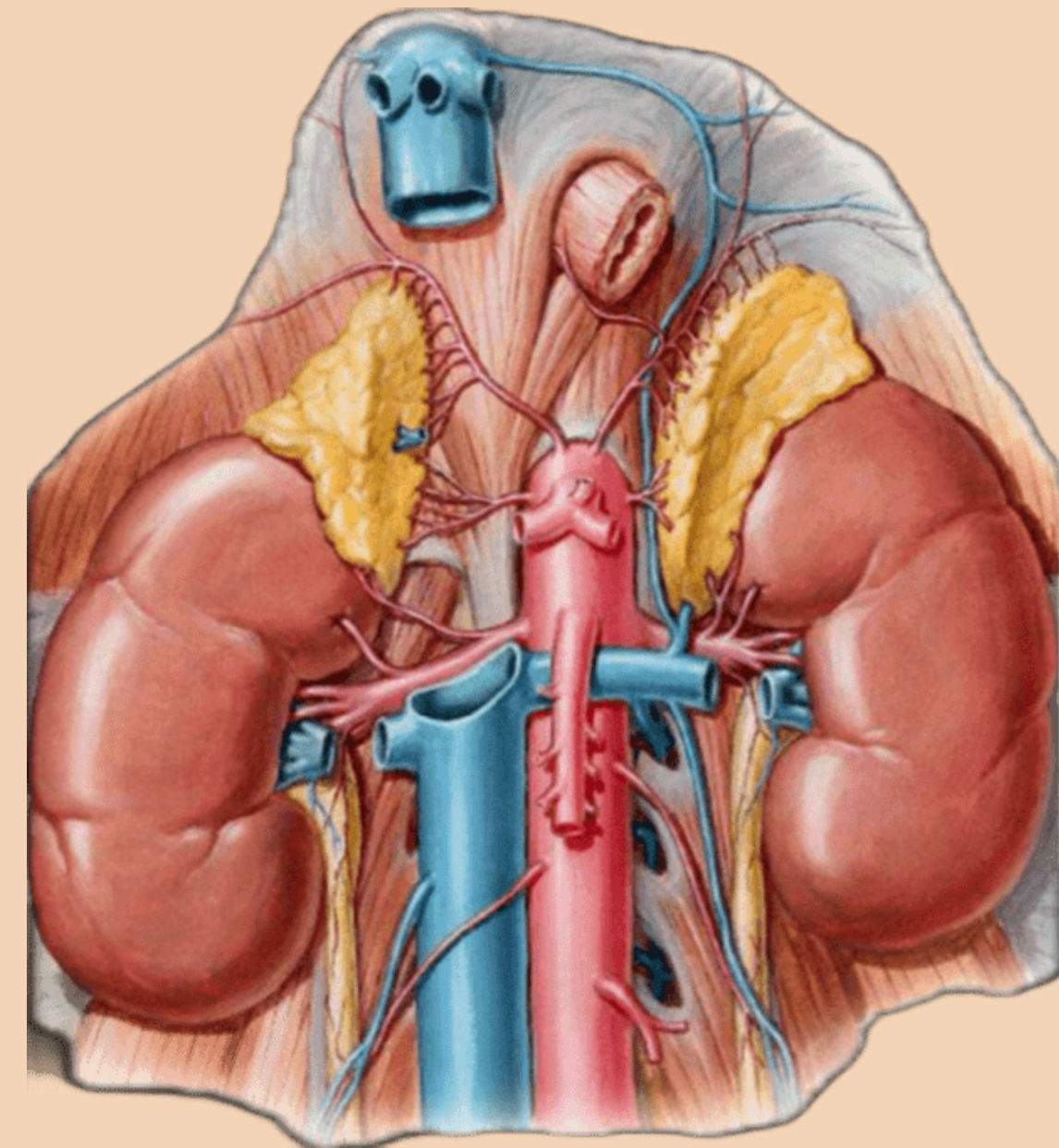
INTRODUCTION TO THE ADRENAL (SUPRARENAL) GLANDS

The adrenal glands (also called **suprarenal glands**) are small, paired endocrine organs located on the superior poles of the kidneys.

They are retroperitoneal, yellowish in color, and each weighs approximately 4-6 grams in adults. Despite their small size, they play a critical role in **regulating homeostasis**, the stress response, metabolism, electrolyte balance, and blood pressure.

Anatomical Structure

Each adrenal gland is enclosed in a connective tissue capsule and surrounded by perirenal fat and the renal fascia, separating them from the kidneys.

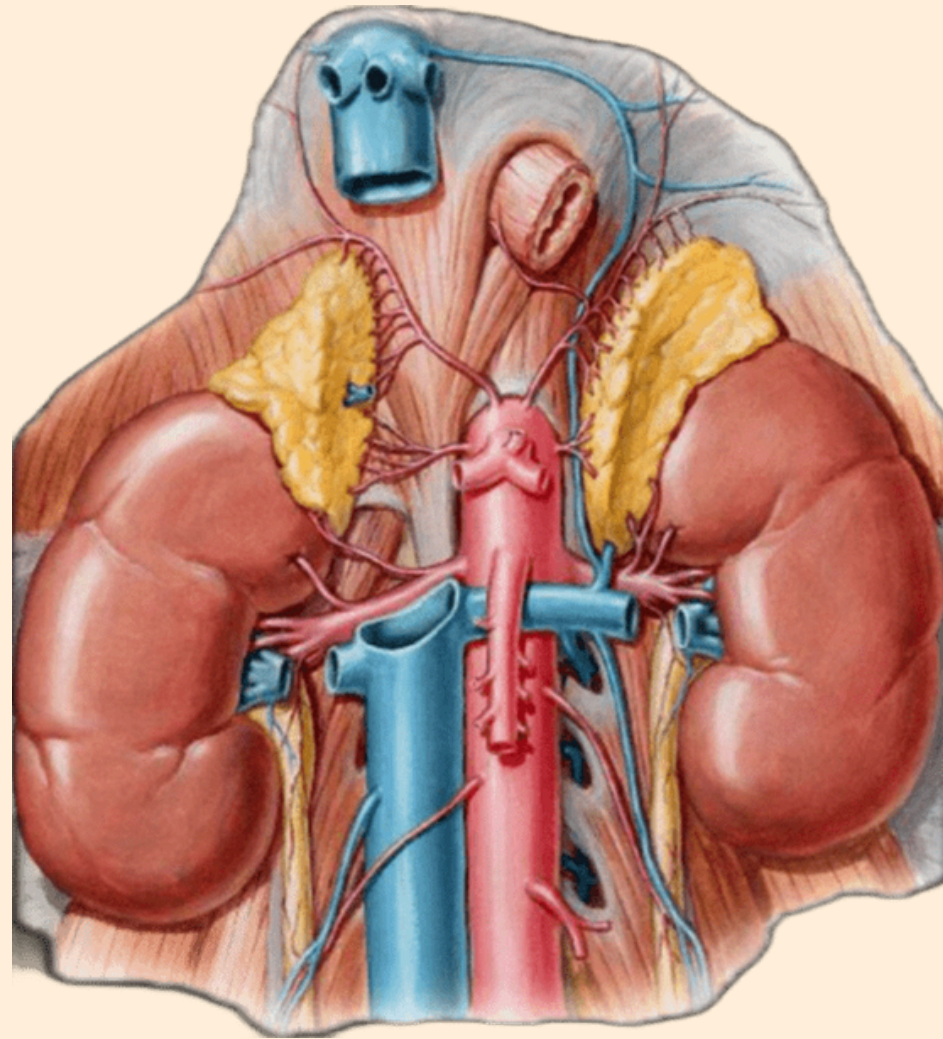


INTRODUCTION CONT...

Right Suprarenal Gland

triangular in shape, and caps the upper pole of the right kidney

it lies between the diaphragm posteromedially and the inferior vena cava anteromedially



Left Suprarenal Gland

Semilunar in shape & extends further inferiorly on the medial margin of the left kidney than does the right kidney

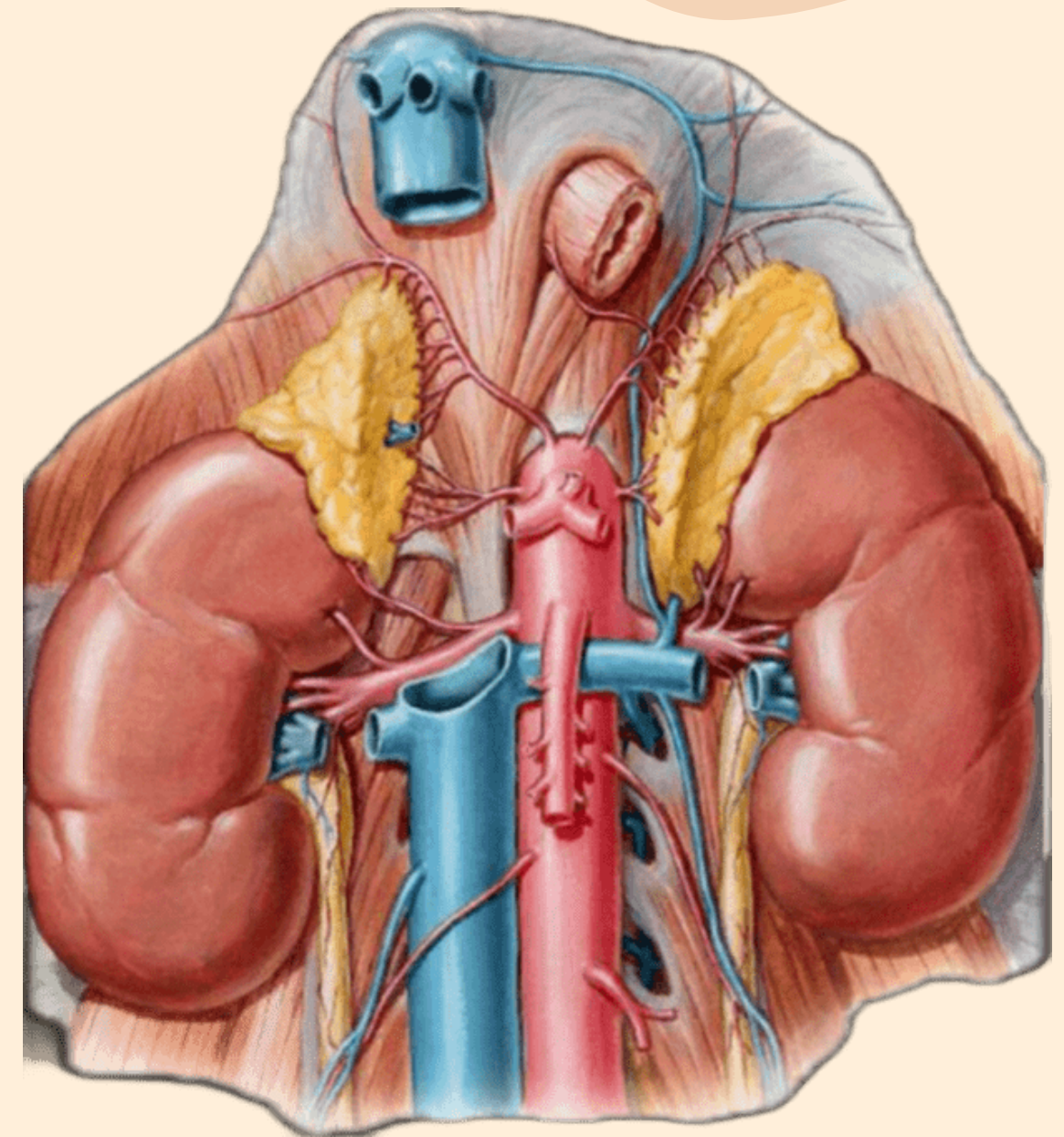
related anteriorly to the stomach and pancreas and posteriorly to the diaphragm

INTRODUCTION CONT...

Relations Between the Two Glands:

Between the right and left adrenal glands lie the following major structures

1. The crura of the diaphragm
2. The abdominal aorta
3. The coeliac trunk and associated plexus
4. The inferior vena cava (IVC)



INTRODUCTION CONT...

blood supply

- Arterial Supply
- Superior suprarenal arteries (from the inferior phrenic)
- Middle suprarenal arteries (from the aorta)
- Inferior suprarenal arteries (from the renal arteries)

Venous Drainage

- Right adrenal vein drains directly into the IV
- Left adrenal vein drains into the left renal vein (often after joining the inferior phrenic vein)
- Lymphatic Drainage: Para-aortic (lumbar) lymph nodes

Innervation

Cortex: No direct innervation (hormonal control)

Medulla: Innervated by preganglionic sympathetic fibers (T10-L1)

HISTOLOGICAL AND FUNCTIONAL ORGANIZATION

The adrenal gland is divided into two major regions

2-Cortex (origin: intermediate mesoderm)

Responsible for the synthesis of **steroid hormones**. It is organized into three concentric zones (from outer to inner):

1.Zona glomerulosa

Secretes **mineralocorticoids** (mainly aldosterone)

Regulated by the renin-angiotensin-aldosterone system (RAAS)

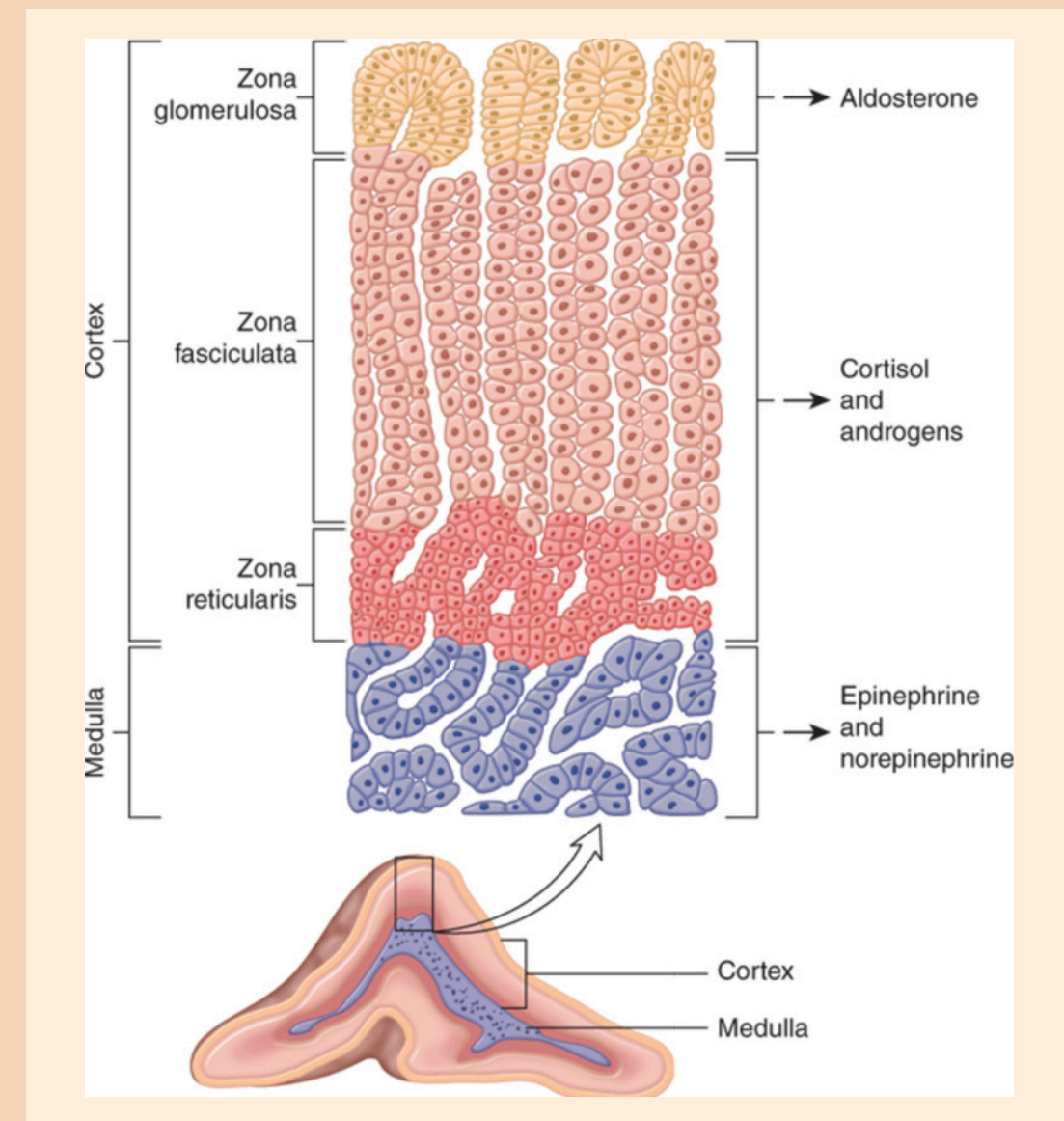
.Controls sodium and potassium balance and blood pressure

2.Zona fasciculata

Secretes **glucocorticoids** (mainly cortisol)

Regulated by the hypothalamic-pituitary-adrenal (HPA) axis

.Controls metabolism, immune suppression, and stress response.



HISTOLOGICAL AND FUNCTIONAL ORGANIZATION

3. Zona reticularis

Secretes androgens (e.g., DHEA)

Minor contribution to sex hormone production (more significant in females)

2-Medulla (origin: neural crest cells - ectodermal)

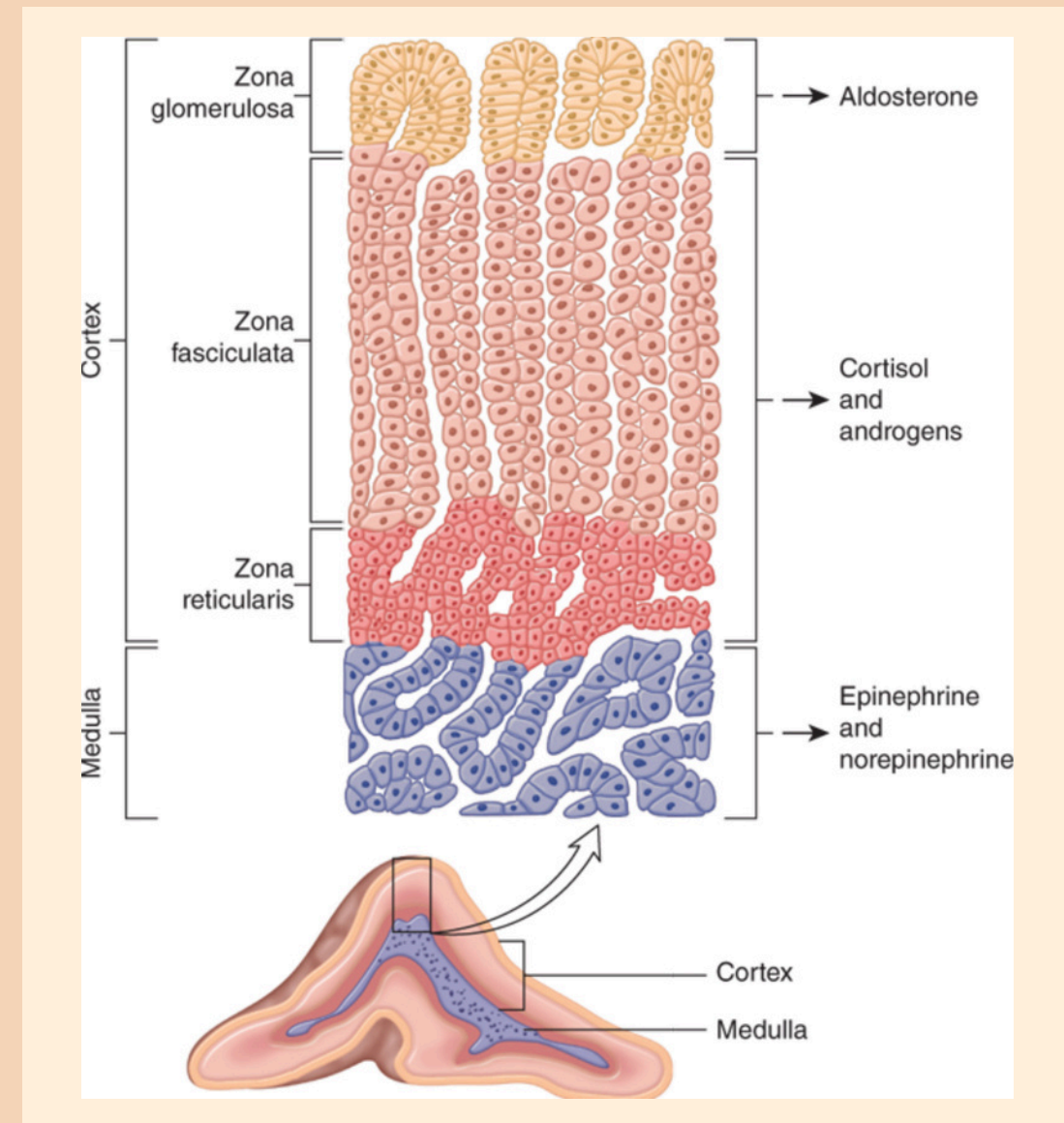
Contains **chromaffin cells**, which are modified postganglionic sympathetic neurons.

They secrete: catecholamines in response to stress

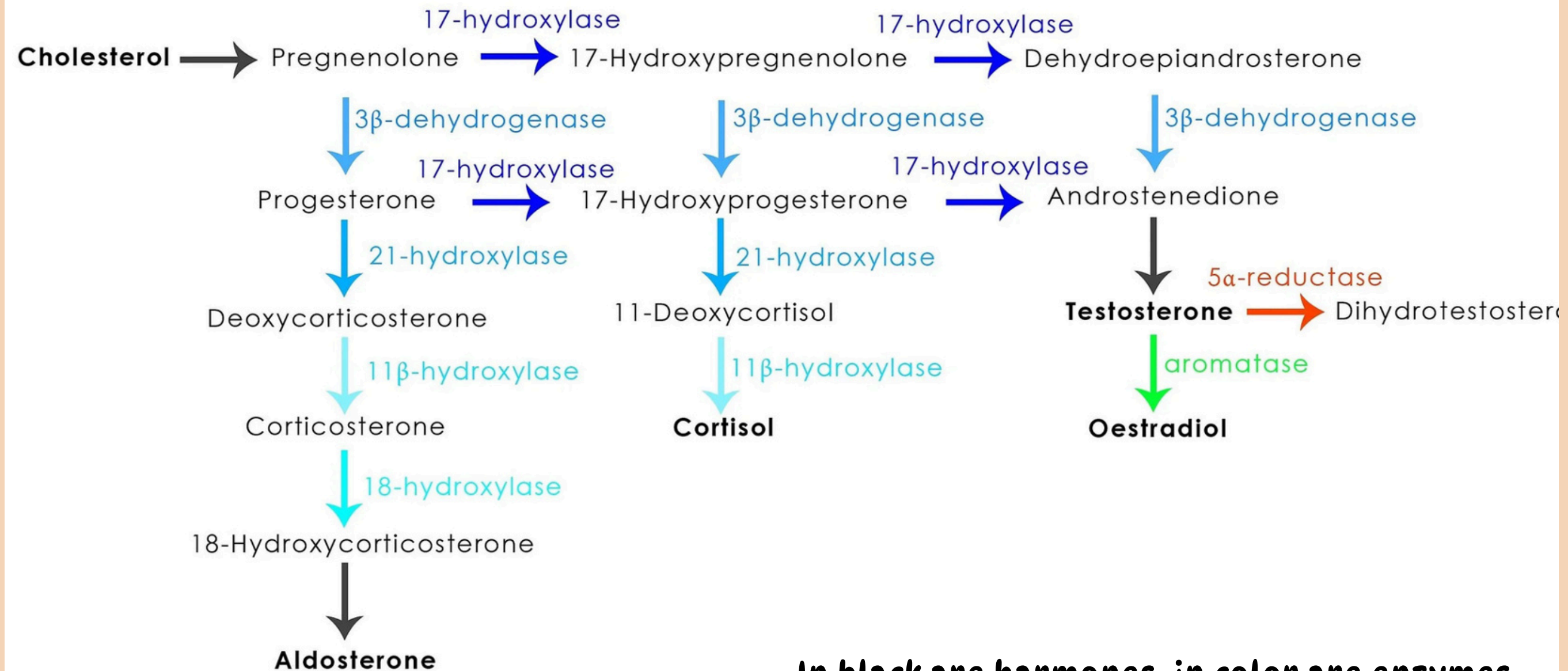
Epinephrine (adrenaline) - ~80%

Norepinephrine (noradrenaline) - ~20%

These hormones mediate the **"fight-or-flight"** response: **increasing** heart rate, blood pressure, blood glucose, and diverting blood to muscles



SYNTHESIS OF STEROID HORMONES

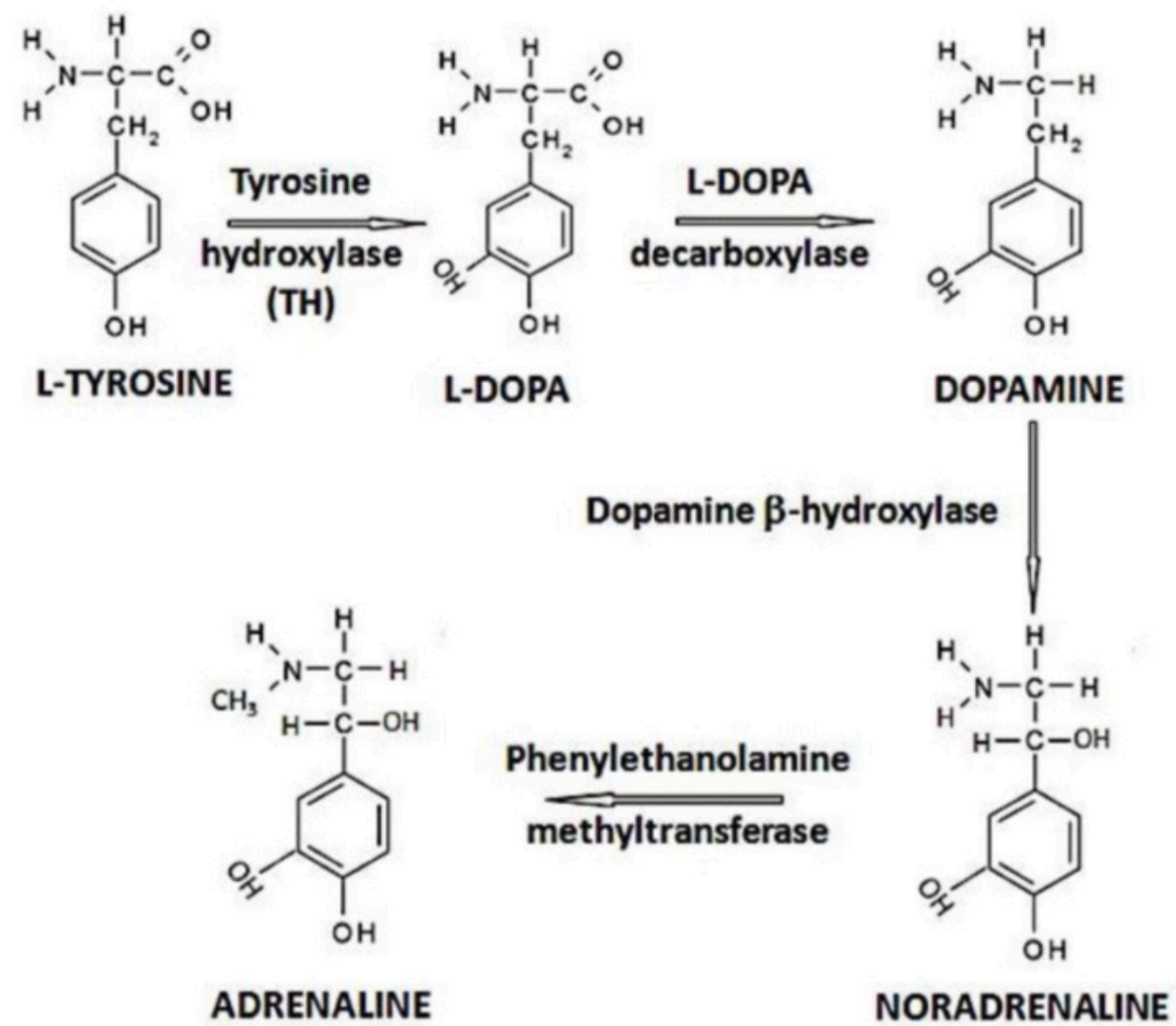


In black are hormones, in color are enzymes

CATECHOLAMINE SYNTHESIS:

Catecholamines Synthesis :

Figure 1



INTRODUCTION CONT...

Hormonal Regulation Overview

Cortisol → Controlled by ACTH from the anterior pituitary

Aldosterone → Controlled mainly by angiotensin II and serum potassium levels

Catecholamines → Released via sympathetic stimulation, mainly through acetylcholine

CLINICAL SIGNIFICANCE & INTRODUCTION TO ADRENAL DISORDERS

Because adrenal hormones influence essential processes like stress response, fluid/electrolyte balance, and metabolism, disorders of the adrenal gland can have severe systemic consequences. These disorders are typically classified as:

Hormone excess syndromes (Hyperfunction)

Cushing's syndrome excess cortisol

Primary hyperaldosteronism (Conn's syndrome) - excess aldosterone

Pheochromocytoma excess catecholamines.

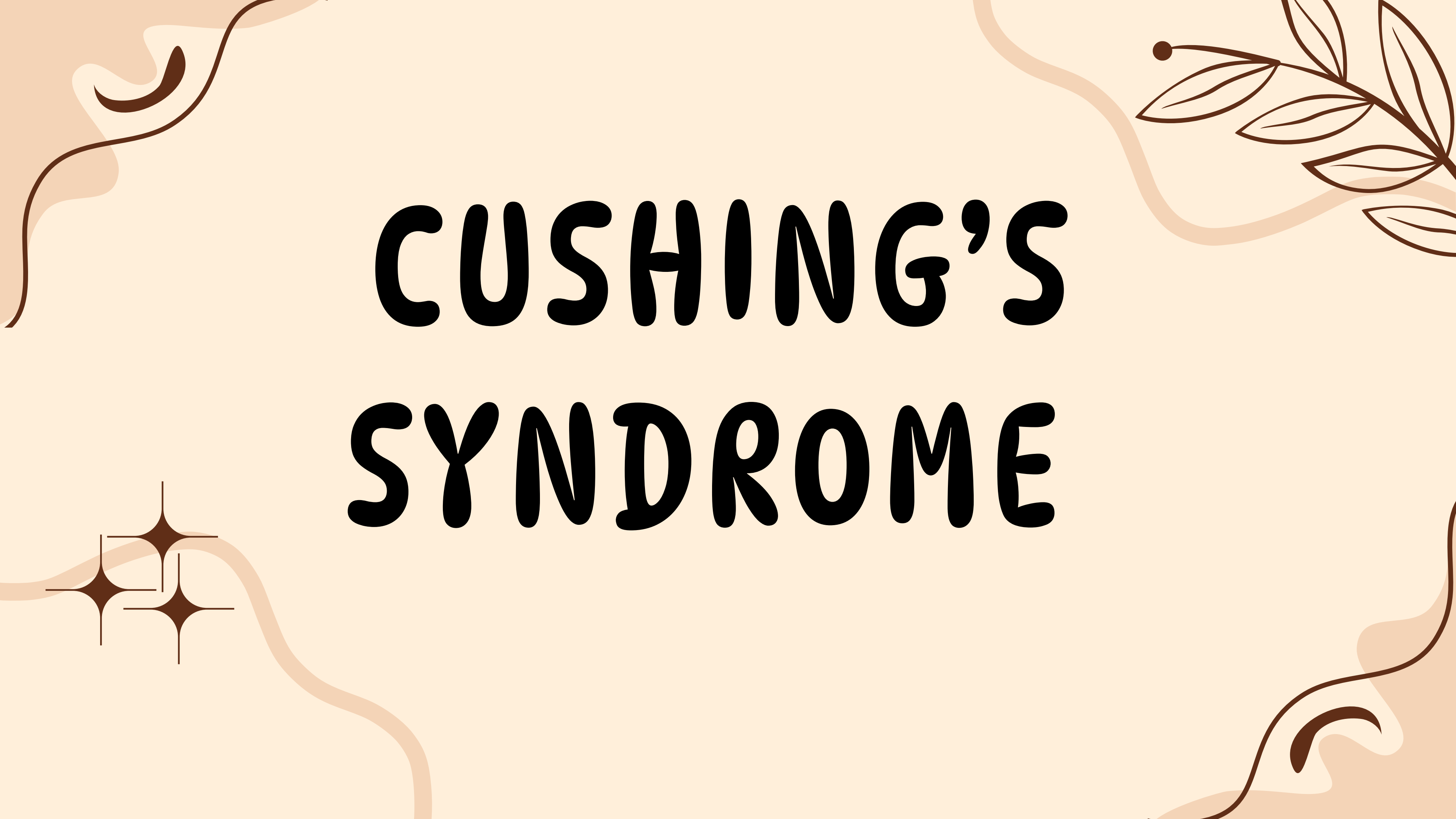
Hormone deficiency syndromes (Hypofunction)

Addison's disease - primary adrenal insufficiency

Secondary adrenal insufficiency - due to pituitary ACTH deficiency

Congenital disorders

Congenital adrenal hyperplasia (CAH) - enzyme defects affecting cortisol/aldosterone synthesis



CUSHING'S SYNDROME

DEFINITION

Cushing's Syndrome is a clinical condition caused by prolonged exposure of body tissues to **high levels of cortisol** (hypercortisolism), regardless of the cause.

It may **result** from:

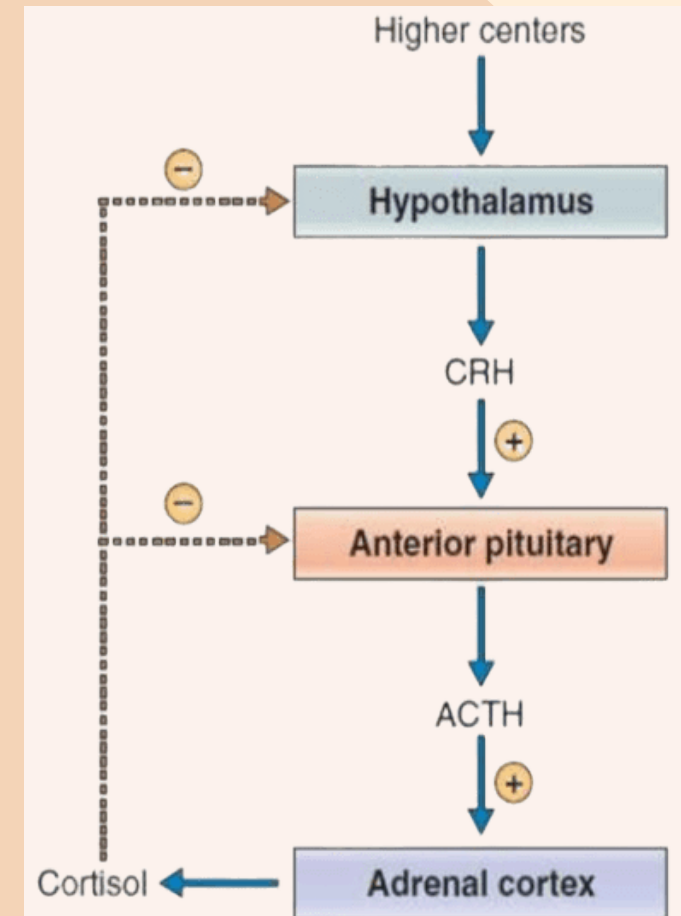
- **Endogenous** overproduction of cortisol (by pituitary or adrenal disorders), or
- **Exogenous** administration of corticosteroid medications.

In this syndrome, the normal circadian rhythm and stress-related regulation of cortisol secretion are lost, leading to persistently elevated cortisol and characteristic physical & metabolic changes.

PATHOPHYSIOLOGY OF CUSHING'S SYNDROME

Normal Physiology

- Hypothalamus → CRH → stimulates Anterior pituitary → ACTH → stimulates Adrenal cortex (zona fasciculata) → secretes Cortisol.
- Cortisol provides negative feedback on hypothalamus & pituitary.
- Secretion follows a circadian rhythm and increases during stress.



In Cushing's Syndrome

- Loss of feedback control within the HPA axis.
 - Causes persistent hypercortisolism, leading to widespread metabolic, immune, and structural changes.

CAUSES (CLASSIFICATION) OF CUSHING'S SYNDROME

1. Exogenous (iatrogenic) - **Most Common**

- Prolonged corticosteroid therapy (e.g., prednisone, methylprednisolone).
- Exogenous steroids mimic cortisol → suppress CRH & ACTH → adrenal atrophy,
but serum cortisol stays high due to the medication.

💡 Seen in patients treated for autoimmune or inflammatory diseases

CAUSES (CLASSIFICATION) OF CUSHING'S SYNDROME

2. Endogenous Causes

A. ACTH-Dependent

1. Cushing's Disease: Pituitary microadenoma (<10 mm) → secreted excess ACTH → ↑ Cortisol from both glands.

Most common endogenous cause.

2. Ectopic ACTH Production(paraneoplastic cause): Non-pituitary tumors (e.g., Small cell lung cancer, Carcinoid, Medullary thyroid carcinoma)result in ↑ ACTH → ↑ Cortisol.

B. ACTH-Independent

1. Adrenal Adenoma: Benign tumor of the adrenal cortex → autonomous cortisol secretion.
2. Adrenal Carcinoma: Malignant tumor → excessive cortisol + possible androgen excess
3. Bilateral Adrenal Hyperplasia: Both glands enlarged and overactive → uncontrolled cortisol overproduction

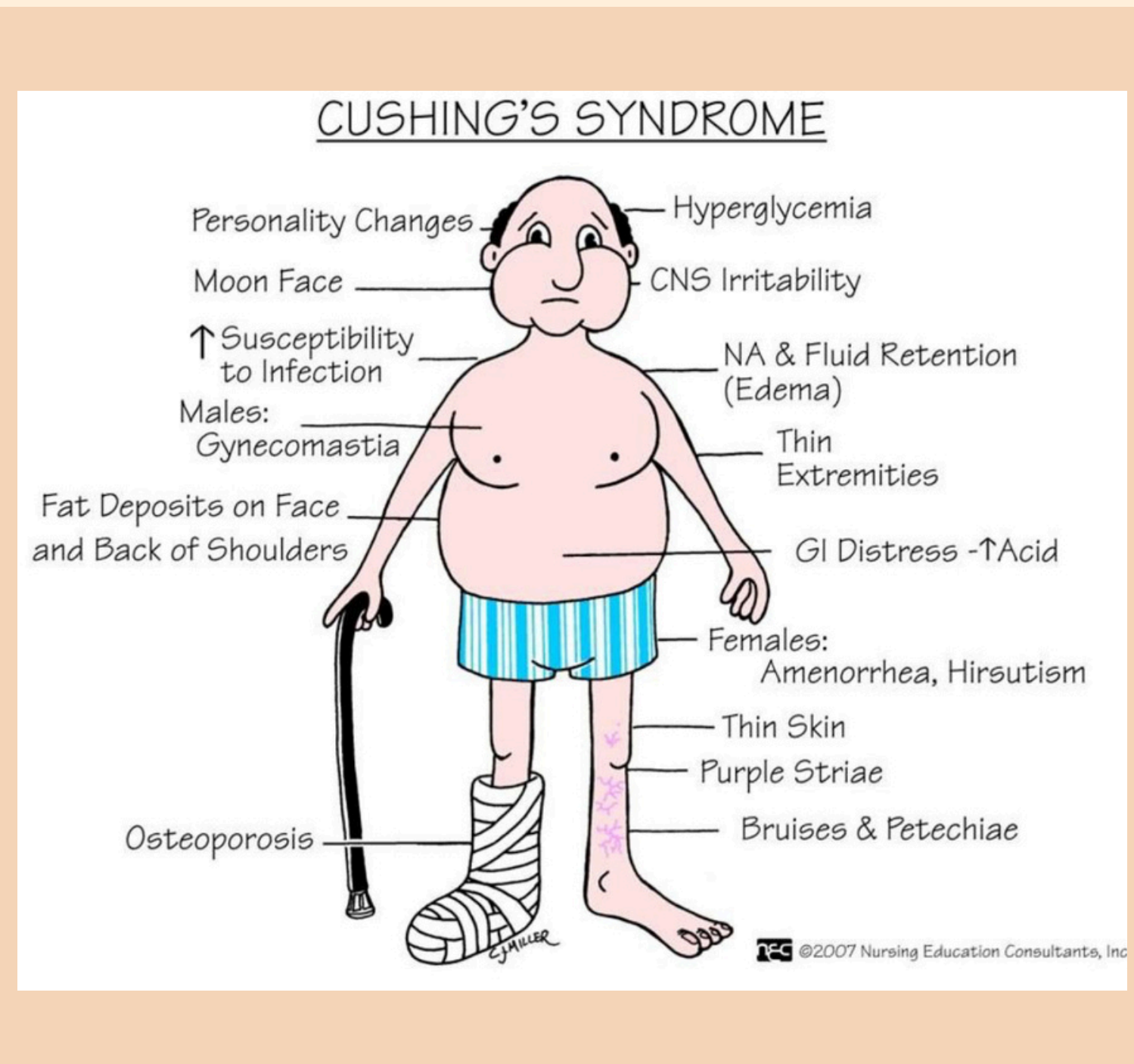
COMMON CLINICAL FEATURES:

1. Weight gain
2. Hypertension
3. Hyperglycemia
4. Round face

5. Menstrual irregularities
6. Thin skin
7. Bruising and striae
8. Delayed wound healing

Special Clinical features:

1. Skin hyperpigmentation
2. Androgen excess



COMPLICATIONS

1. Hypertension

Cortisol increases α_1 receptor activity and causes sodium and water retention, leading to elevated blood pressure due to increased vascular resistance and blood volume.

2. Diabetes Mellitus

Cortisol stimulates gluconeogenesis and glycogenolysis, which raise blood glucose levels and cause insulin resistance, resulting in hyperglycemia.

3. Osteoporosis

Cortisol enhances osteoclast activity and protein breakdown, reducing bone mass and predisposing to fractures.

(BMD < -2.5 on DEXA scan)

COMPLICATIONS CONT...

4. Hypogonadism

High cortisol suppresses GnRH, leading to reduced FSH and LH levels, decreased sex hormones, and manifestations such as low libido, menstrual irregularities or amenorrhea, and infertility.

5. Increased Risk of Infection

Cortisol decreases lymphocytes and eosinophils, causing immunosuppression and higher risk of infections like candidiasis, cellulitis, MRSA, and PJP.

6. Encephalopathy

Excess cortisol increases sympathetic activity, which may cause psychosis and agitation.

DIAGNOSIS STEPS

1

- Confirm Hypercortisolism (Screening tests)
- 24-hour urinary cortisol: increased
- Low-dose dexamethasone test: cortisol not suppressed
- Late-night salivary cortisol: increased
-  If 2 of 3 positive → Cushing's confirmed

2

- Determine the source (ACTH level)
- Low ACTH → Adrenal Cushing's (Cushing's syndrome)
- High ACTH → Pituitary (Cushing's disease) or Ectopic ACTH

4

- Imaging
- Low ACTH → Adrenal CT/MRI → Adenoma
- High ACTH + positive tests → Pituitary MRI → Microadenoma
- High ACTH + negative tests → Whole-body CT → Ectopic tumor (e.g. lung cancer)

3

- Differentiate Disease vs Ectopic ACTH
- CRH stimulation test:
- Increased ACTH → Cushing's disease
- No change → Ectopic ACTH
- High-dose dexamethasone test:
- Decreased cortisol → Cushing's disease
- No change → Ectopic ACTH

TREATMENT

Medical Management:

- Gradual steroid taper: Used when the cause is chronic exogenous corticosteroid therapy, to allow recovery of the hypothalamic-pituitary-adrenal (HPA) axis and prevent adrenal crisis.
- Ketoconazole: Inhibits cortisol synthesis. It can be used as preoperative preparation or as long-term therapy for adrenal or pituitary tumors when surgery is not possible.

Surgical Management:

- Adrenalectomy: Indicated for adrenal adenoma or carcinoma producing excess cortisol.
- Transsphenoidal resection: Indicated for pituitary microadenoma (Cushing's disease).

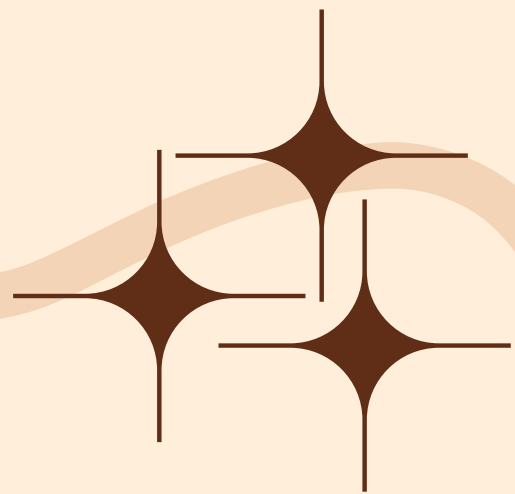
TREATMENT

Monitoring and Follow-up:

- Regular measurement of ACTH and cortisol levels to assess treatment response.
- Monitor for signs of adrenal insufficiency or adrenal crisis after surgery or during tapering.
- Check liver function tests (LFTs) during ketoconazole therapy due to risk of hepatotoxicity.
- Watch for hypopituitarism following pituitary surgery and replace deficient hormones as needed.



HYPERALDOSTERONSIM



OVERVIEW

Mineralocorticoids Overview (aldosterone)

Aldosterone enters the cell and binds to its receptor in the cytoplasm.

The hormone-receptor complex moves to the nucleus and activates genes by binding to mineralocorticoid response elements (MREs).

This leads to the production of proteins like ENaC and Na⁺/K⁺ ATPase, which **increase Na⁺ reabsorption and K⁺ excretion** in the kidney's collecting ducts

Triggers for aldosterone secretion:

- Renin-angiotensin system
- Hyperkalemia
- ACTH (minor trigger)

OVERVIEW

Effects of Aldosterone:

↑ Number of Na^+/K^+ ATPase pumps.

↑ Synthesis of epithelial Na^+ channels (ENaC) → more Na^+ enters the cell → more Na^+ pumped into the blood.

↑ K^+ conductance → K^+ moves from the cell to the lumen.

As more Na^+ is being reabsorbed from the lumen into the blood, lumen **negativity increases**, which acts as a driving force for K^+ to leave the cell into the lumen.

Also, because the negativity in the lumen increases, **H^+ ATPase** in the α -intercalated cell **increases** in activity, leading to H^+ excretion. BUT this creates more negativity in the cell itself, so **HCO_3^-** must leave the cell into the blood to compensate for the increased negativity, so the activity of $\text{HCO}_3^-/\text{Cl}^-$ increases to keep the overall balance of charges stable.

Net results:

Na^+ and water retention → ↑ extracellular volume (ECV)

K^+ excretion

H^+ secretion → metabolic alkalosis

OVERVIEW

Renin

is released by the healthy kidneys from the **juxtaglomerular apparatus** in response to at least three independent factors:

- Decreased** blood volume, as measured by the juxtaglomerular cells. These are specialized vascular smooth muscle cells in the afferent arteriole.
- Elevated** levels of filtered sodium, as measured by the efferent macula densa cells in the DCT.
- Sympathetic nervous system** stimulation (β_1 receptors).

Function of Renin: It converts angiotensinogen to angiotensin I, which is then converted to angiotensin II by ACE- mainly in the lungs.

Angiotensin II has pressor effects and **stimulates** aldosterone release from the zona glomerulosa in the adrenal glands.

DEFINITION

Hyperaldosteronism

Hyperaldosteronism is a syndrome associated with **hypersecretion** of the major adrenal **mineralocorticoid, aldosterone**, from the adrenal glands, leading to sodium and water retention and potassium loss.

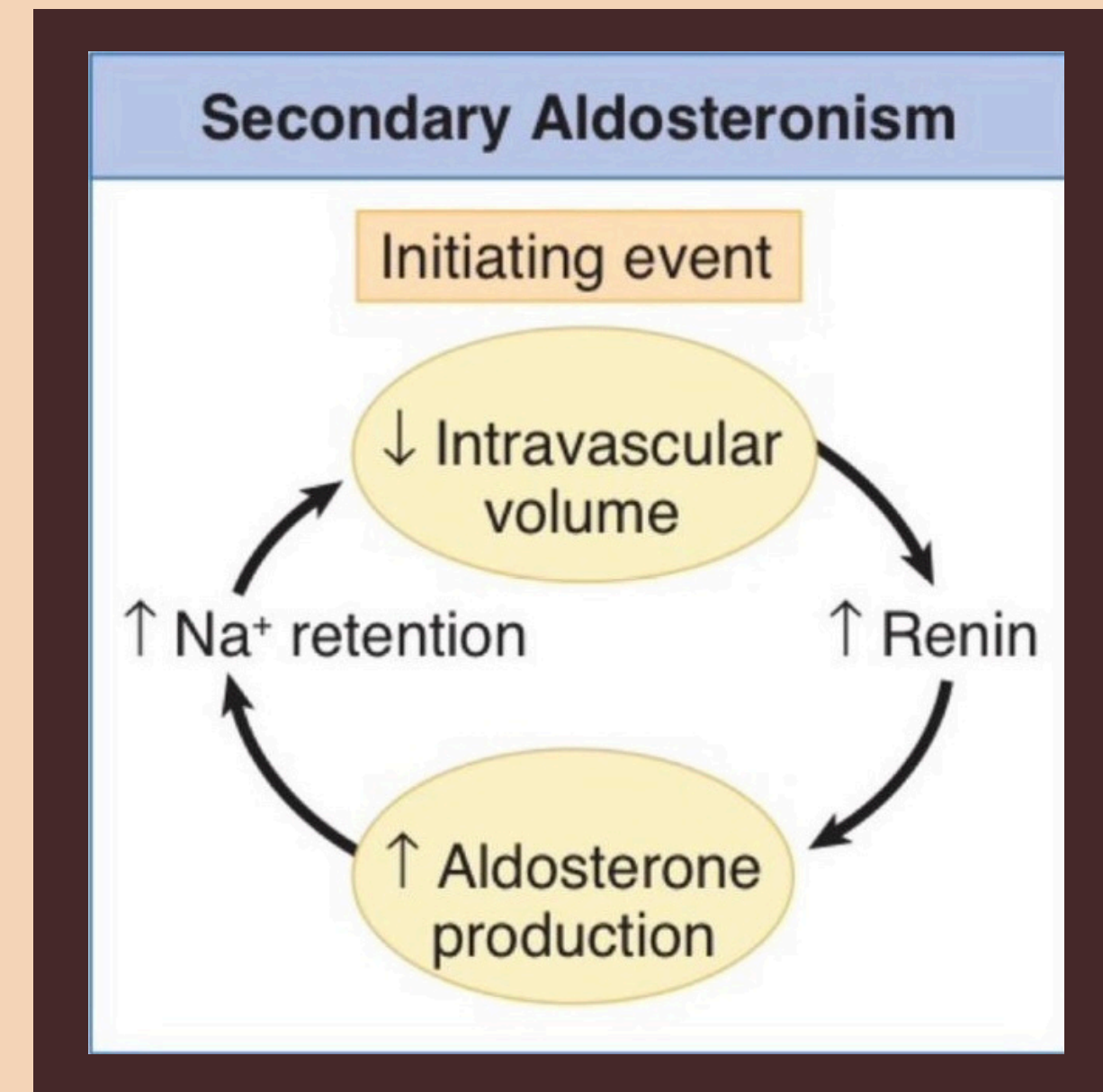
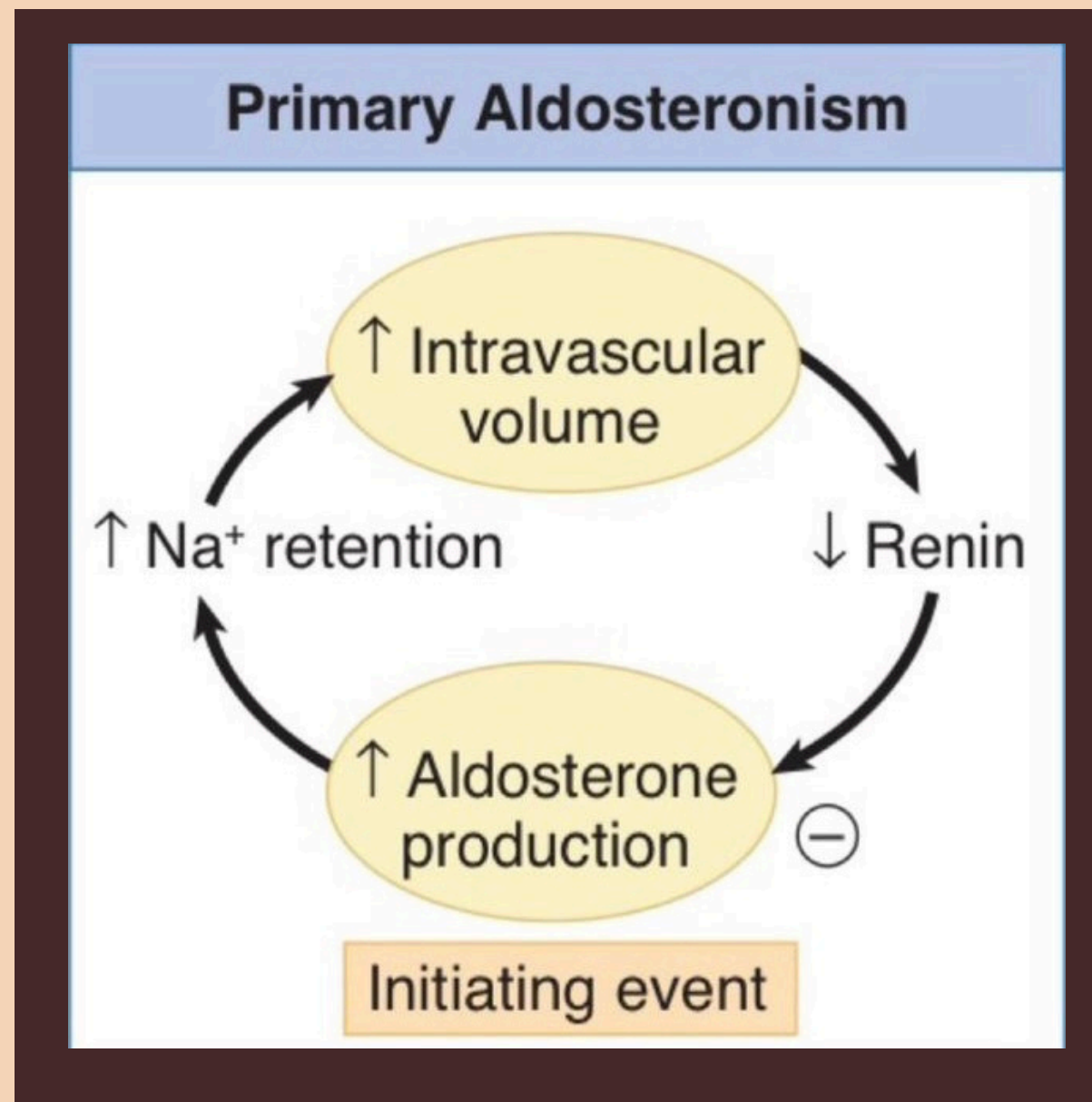
Hyperaldosteronism can be divided into the following:

Primary (Conn's syndrome): caused by a problem within the **adrenal gland itself**, such as an adenoma or hyperplasia.

Secondary: caused by **external factors** that increase aldosterone production, such as renal artery stenosis or kidney disease.

The **most common** cause of primary hyperaldosteronism is a **unilateral adrenal adenoma** (70%). Bilateral hyperplasia accounts for 25–30%. Excessive black licorice ingestion can mimic this effect. Licorice has aldosterone-like qualities

DEFINITION



CLINICAL FEATURES OF HYPERALDOSTERONISM

1

1. Hypertension (resistant hypertension) is the most common

2

2. Hypokalemia-related symptoms:

- Muscle weakness
- Muscle cramps
- Fatigue
- Paresthesia (tingling or numbness)
- Polyuria and polydipsia (due to renal potassium loss)

3

3. Metabolic alkalosis

4

4. Absence of peripheral edema (aldosterone escape)

5

5. Cardiovascular complications

- (if untreated):
- Left ventricular hypertrophy
- Arrhythmias

RISK FACTORS FOR HYPERALDOSTERONISM:

- **Family history**
- **Resistant hypertension**
- **Age >30**
- **Obesity**
- **Sleep apnea**

DIAGNOSIS

When to suspect it (indicating for testing):

1. Resistant or difficult to control hypertension
2. Unexplained hypokalemia
3. Hypertension at a young age
4. Family history of early onset hypertension or stroke

initial Screening

. Aldosterone to Renin Ratio (ARR)

-Measure Plasma Aldosterone Concentration (PAC) and Plasma Renin Activity (PRA)

-High ARR suggest primary hyperaldosteronism

-A positive screen is a PAC/PRA ratio $>20:1$ and a PAC > 15

. Some medication, such as diuretics or ACE inhibitors, must be stopped before testing because they affect the result

DIAGNOSIS CONT...

confirmatory test

If the **ARR is elevated**, do a confirmatory test to prove inappropriate aldosterone secretion:

1. saline infusion
2. Oral sodium loading test (best)
3. Fludrocortisone suppression test (best)
4. Captopril challenge test (best)

These tests check if aldosterone remains high even when it should be suppressed

localization(finding the cause):

Once the diagnosis is confirmed, identify where the excess aldosterone comes from:

1. CT scan or MRI of the adrenal gland
 - Detects adenoma or adrenal hyperplasia.
2. Adrenal Venous Sampling (AUS)
 - The most accurate method to distinguish unilateral vs. bilateral disease

TREATMENT

-Types and treatment approach:

A. Primary Hyperaldosteronism

1. Unilateral disease (usually aldosterone-producing adenoma).

Treatment: Surgical removal of the affected adrenal gland (**laparoscopic adrenalectomy**).

2. Bilateral adrenal hyperplasia

Treatment: medical therapy, **not surgery**

Use mineralcorticoid receptor antagonist:

1. Spironolactone (**most common**)

2. Eplerenone (fewer side effects, preferred in men due to less gynecomastia)

Use antihypertensive drug if needed

TREATMENT

B. Secondary Hyperaldosteronism

Treat the underlying cause, such as:

Renal artery stenosis, Angioplasty or stenting.

Congestive Heart Failure, Heart Failure management.

Cirrhosis or nephrotic syndrome, Manage fluid overload

ADRENAL INSUFFICIENCY

ADRENAL INSUFFICIENCY:

- Loss of adrenal function**

- Loss of one or more adrenal hormones**

- . **Glucocorticoids: cortisol**

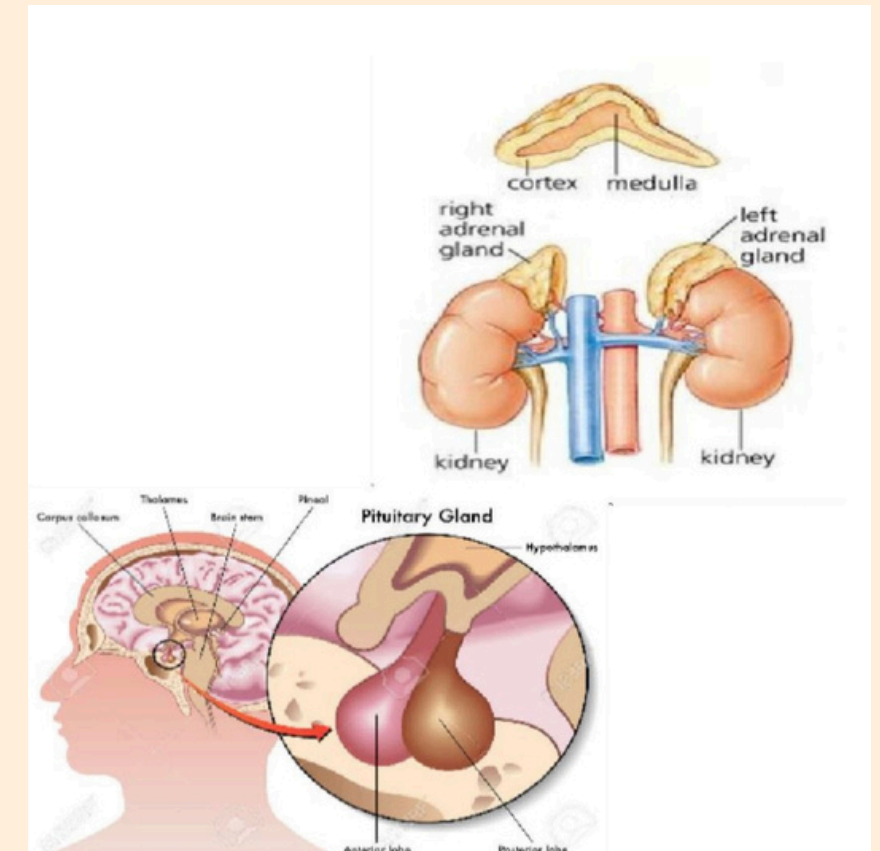
- . **Mineralocorticoids: aldosterone**

- . **Androgens: dehydroepiandrosterone (DHEA)**

TYPES

There are three major types of adrenal insufficiency

- **Primary adrenal insufficiency :**
is due to impairment of adrenal glands. (Addison's disease) **Loss of cortisol, aldosterone and androgens.**
- **Secondary adrenal insufficiency :**
is caused by impairment of pituitary glands **Loss of ACTH from pituitary (secondary) Loss of cortisol only.**
- **Tertiary adrenal insufficiency:**
is due to hypothalamic disease and decrease in corticotropin releasing hormone (CRH) **Loss of cortisol only.**



CLINICAL FEATURES

Glucocorticoid Deficiency Clinical Features

- Fatigue
- Weight loss
- Nausea + vomiting or abdominal pain
- Hypoglycemia
- Low blood pressure (may cause dizziness or fainting, especially when standing).
- Muscle and joint pain
- Hyponatremia (↑ ADH)

CLINICAL FEATURES

Mineralocorticoid Deficiency Clinical Features

- Low volume (salt & water loss, salt craving, possible shock)
- Hyponatremia (↑ ADH, water retention)
- Hyperkalemia
- Metabolic acidosis (↓ acid excretion)

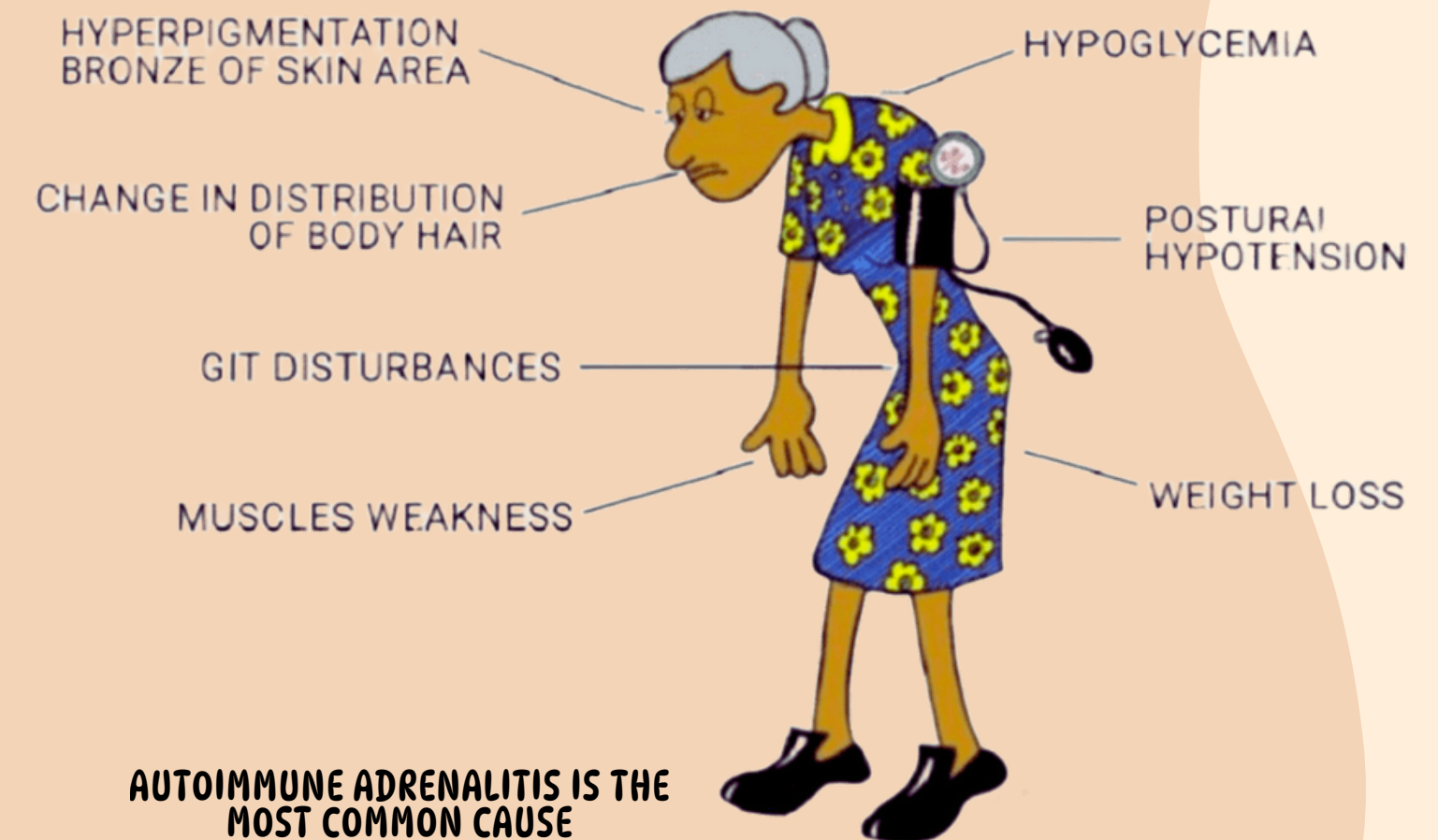
Androgen Deficiency Clinical Features

- No major effect in males
- ↓ Axillary & pubic hair in females

PRIMARY ADRENAL INSUFFICIENCY

Clinical feature:

- Fatigue
- Weight loss
- Nausea, vomiting and abdominal pain
- Muscle and joint pain
- Postural hypotension
- Salt craving
- Hyponatremia
- Hyperkalemia
- Eosinophilia



SKIN HYPER PIGMENTATION

- ACTH is high in primary adrenal insufficiency
- ↑ melanocyte stimulating hormone (MSH)
- Common precursor in pituitary with ACTH
- Proopiomelanocortin (POMC)
- ↑ ACTH → ↑ MSH → ↑ melanin synthesis
- Most obvious in sun exposed areas **Face, neck, backs of hands**
- May also occur on **mucous membranes**



PRIMARY ADRENAL INSUFFICIENCY - CAUSES:

- **Autoimmune** (most common in developed countries)
- **Infections** (TB most common worldwide, also fungal, CMV, etc.)
- **Iatrogenic** (e.g. bilateral adrenalectomy)
- **Metastasis** (from lung or breast cancer)

CENTRAL ADRENAL INSUFFICIENCY:

Central Adrenal Insufficiency:

- Weakness, fatigue, muscle/joint pain, weight loss
- Nausea, vomiting, abdominal pain
- Hypotension (less severe; ↓ vascular tone only, mineralocorticoids intact)
- Hyponatremia (less common; mineralocorticoids normal)
- No skin hyperpigmentation (↓ ACTH)

CENTRAL ADRENAL INSUFFICIENCY:

Causes:

Secondary:

- a. Long-term steroid use (most common): chronic suppression of CRH & ACTH → no cortisol response during stress.
- b. Hypopituitarism (rare): no ACTH → no glucocorticoids.

Tertiary:

Hypothalamic disease: no CRH → no ACTH → no glucocorticoids.

- **Diagnosis:**

CENTRAL ADRENAL INSUFFICIENCY:

Diagnosis:

Morning cortisol: normally high; low level suggests adrenal insufficiency.

Plasma ACTH:

Low cortisol + high ACTH → primary

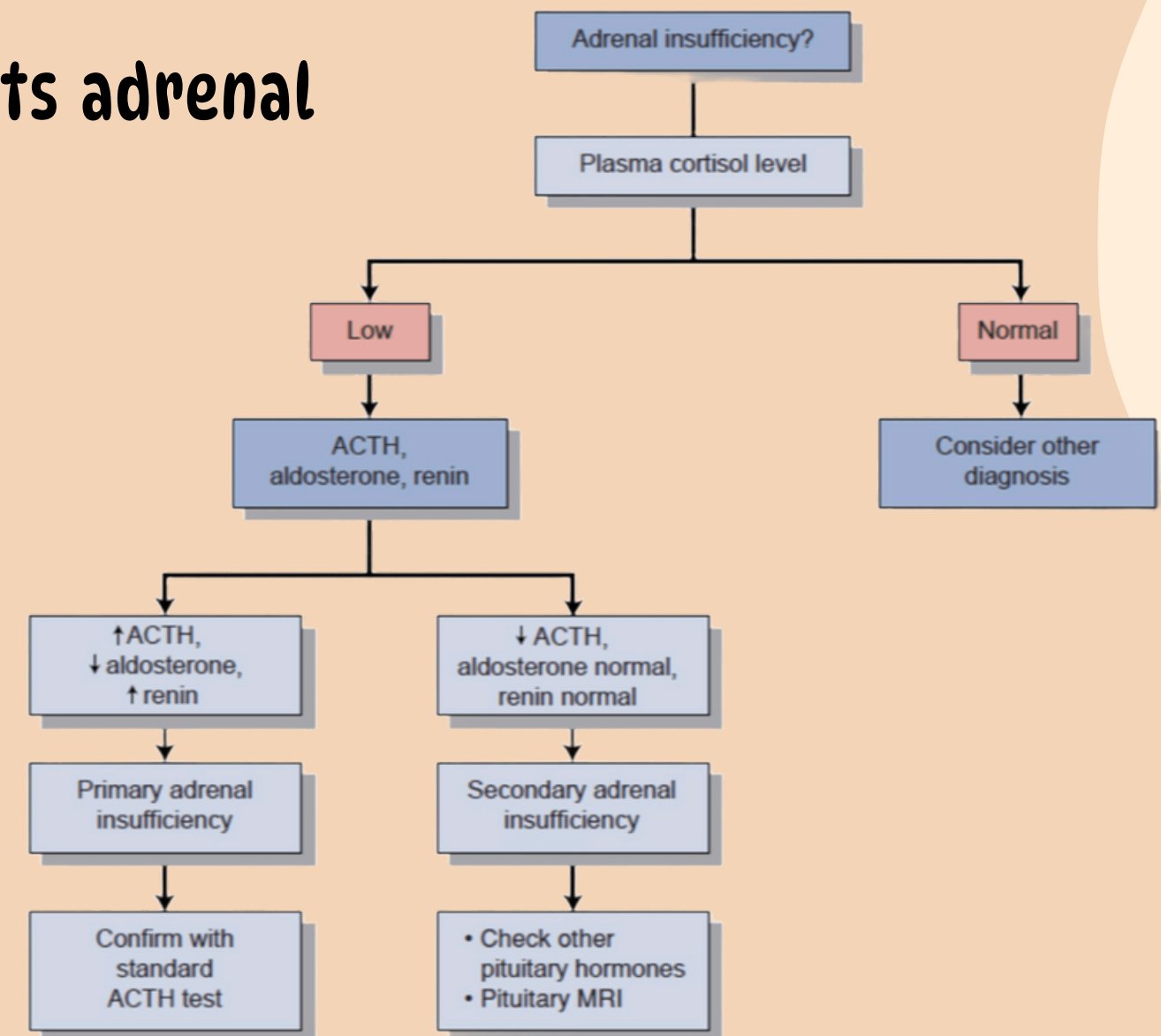
Low cortisol + low ACTH → central

Head MRI for pituitary/hypothalamic lesions.

CRH stimulation test:

No cortisol rise secondary (pituitary failure)

- Cortisol rise tertiary (hypothalamic failure)



CENTRAL ADRENAL INSUFFICIENCY:

Cosyntropin Stimulation Test:

Cosyntropin: synthetic ACTH

Purpose: definitive test for primary adrenal insufficiency

Method: IV cosyntropin, measure plasma cortisol at 30 & 60 min

Normal: cortisol rises $\geq 18-20 \mu\text{g/dL}$

Primary AI: insufficient cortisol rise

- **Secondary AI:** initial **poor** response (adrenals unaccustomed), but respond after 4-5 days of **repeated stimulation**

CENTRAL ADRENAL INSUFFICIENCY:

Determination of Cause:

History & exam: clues like TB, HIV, meningococemia

21-hydroxylase antibodies: autoimmune adrenalitis

Abdominal CT: infection, hemorrhage, malignancy

CT-guided FNA: confirm infection or malignancy

Treatment:

- Primary AI: daily oral glucocorticoid (hydrocortisone/prednisone) + fludrocortisone (mineralocorticoid)
- Secondary AI: daily glucocorticoid only; mineralocorticoid not needed

ADRENAL CRISIS

Adrenal Crisis:

Acute, severe adrenal insufficiency: hypotension, cardiovascular collapse, abdominal pain, acute renal failure, risk of death

Triggered by stress (trauma, infection, surgery)

Can be fatal if untreated

Treatment: IV hydrocortisone, IV fluids (NS + 5% dextrose),

identify & treat underlying cause

Adrenal Crisis Management

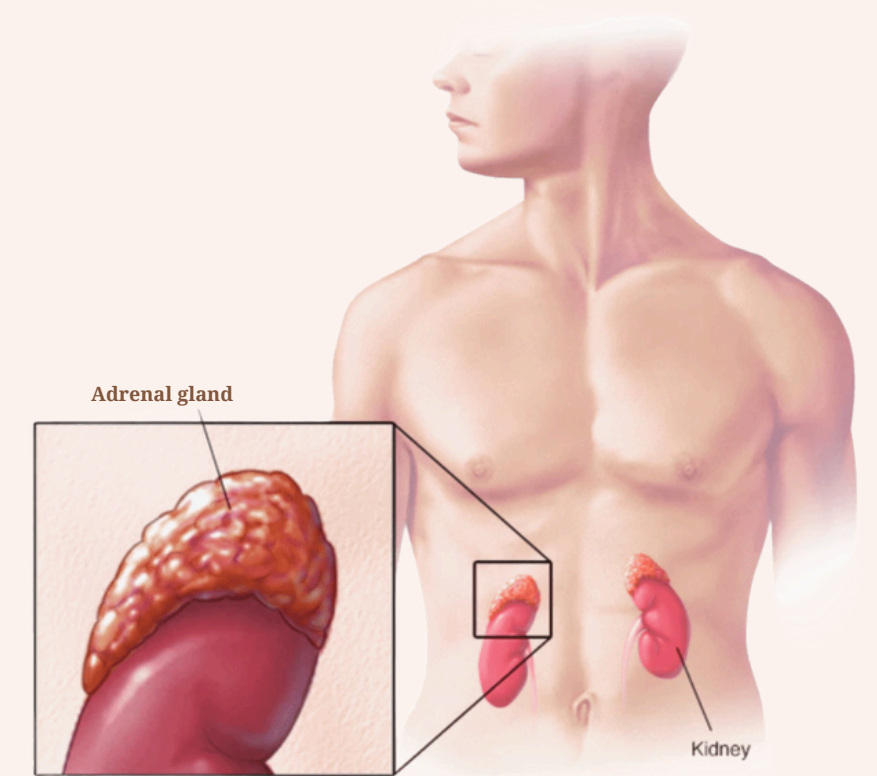
- . Immediate IV hydrocortisone
- . Rapid IV fluids (normal saline + 5% dextrose)
- . Correct electrolyte imbalances
- . Treat underlying cause/stress trigger
- . Monitor vital signs and urine output
- . Gradual transition to oral glucocorticoids once stable

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Congenital Adrenal Hyperplasia (CAH)

Definition:

- Congenital adrenal hyperplasia (CAH) refers to a group of autosomal recessive disorders.
- It results from enzyme deficiencies in the cortisol biosynthesis pathway in the adrenal cortex.
- The **most common** form is 21-hydroxylase deficiency (~90-95% of cases).



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Adrenal glands

Located on top of the kidneys, the adrenal glands make hormones that help regulate metabolism, the immune system, blood pressure and other important functions. Although small, these glands control much of what happens in the body.



Pathophysiology

Deficient cortisol synthesis leads to **increased ACTH** secretion via loss of negative feedback.

- This causes **adrenal hyperplasia** and **overproduction** of androgen precursors.
- Depending on the enzyme affected, there may be aldosterone deficiency or excess.

-The **brain** have a control on the secretion level of corticosteroid, If the level in blood is **low** a signal is sent to the adrenal gland to make more, If the level is **elevated** the inhibition occurs by **negative feedback mechanisms**.



Types of CAH

1. 21-hydroxylase **deficiency** (most common)
2. 11 β -hydroxylase **deficiency**
3. 17 α -hydroxylase **deficiency**
4. 3 β -hydroxysteroid dehydrogenase **deficiency**
5. 18-hydroxylase **deficiency** (rare)

Each enzyme defect affects cortisol, aldosterone, and sex steroid synthesis differently.



Clinical Features

- **Vary** depending on the enzyme defect and severity (classic vs non-classic forms).
 - **Classic 21-hydroxylase deficiency.**
 - Salt-wasting crisis (hyponatremia, hyperkalemia, dehydration)
 - Ambiguous genitalia in females
 - Precocious puberty in males
 - **Non-classic form:** mild androgen excess, hirsutism, menstrual irregularities.
- 
- 

Diagnosis

- Elevated 17-hydroxyprogesterone levels
➔ (especially after ACTH stimulation test).
- Electrolyte abnormalities (↓ Na⁺, K⁺).
- Genetic testing for **CYP21A2** mutations (21- hydroxylase deficiency).
- Newborn screening programs detect elevated 17-OHP on blood spots.



Treatment

- Lifelong glucocorticoid replacement (e.g., hydrocortisone, prednisone) to **suppress ACTH**.
 - Mineralocorticoid replacement (**fludrocortisone**) in salt-wasting forms.
 - Salt supplementation for **infants**.
 - Surgical correction of ambiguous genitalia if needed.
 - Stress-dose steroids during illness or surgery.
- 
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Prognosis

- With **early diagnosis** and appropriate hormone replacement, prognosis is **excellent**.
 - Lifelong **follow-up** required for dose adjustment and monitoring growth and fertility.
 - Genetic counseling is recommended for affected families.
- 
- 



Pheochromocytoma



Definition:

- Catecholamine**-secreting tumor from **chromaffin cells** of the adrenal medulla.
 - Usually benign but may have malignant potential.
 - May occur sporadically (idiopathic) or as part of genetic syndromes (MEN2A, MEN2B, VHL, NF type 1).
- 



Rule of 10s

- 10% are extra-adrenal (90% intra-adrenal).
- 10% are malignant.
- 10% occur in children.
- 10% are bilateral.
- 10% are familial.



Clinical features

Symptoms are usually paroxysmal (episodic attacks):

- Headache
- Palpitations
- Sweating
- Anxiety, tremor, weight loss
- Hypertension (persistent or during attacks)

(Mnemonic: 5 P's → Pressure, Pain, Palpitations, Perspiration, Pallor)



Pain

Classical Triad

Diaphoresis

Palpitations



Diagnosis

1. Clinical presentation:

- High blood pressure
- Headache
- Irregular heartbeat
- Sweating

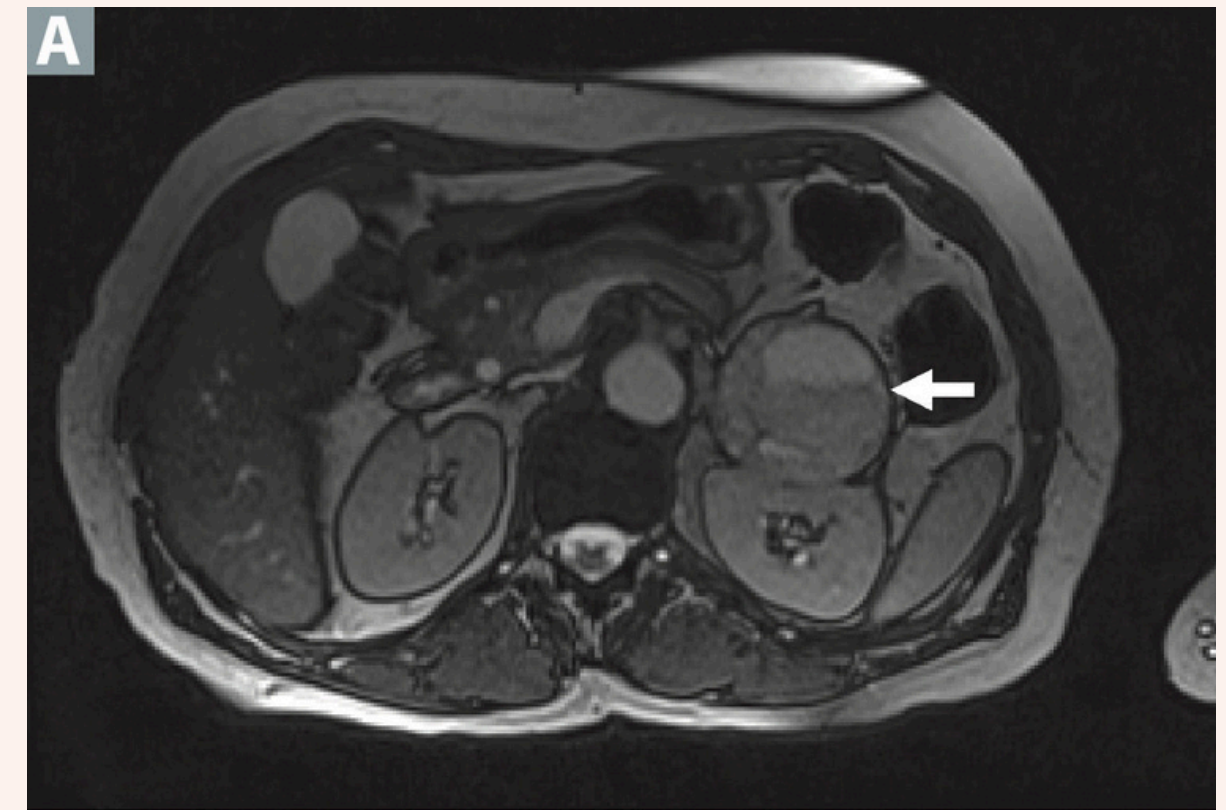
2. Laboratory tests (Urine or plasma):

- ↑ Catecholamines or their metabolites (metanephrines, VMA)
- 
- 

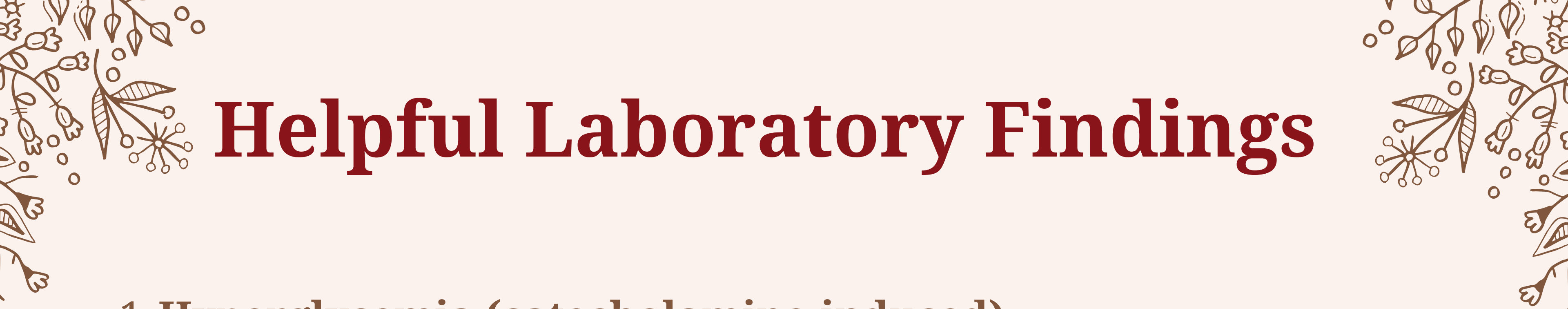
Diagnosis cont...

3. maging:

1. CT or MRI (after confirming biochemically).
2. MIBG scan to localize extra-adrenal or metastatic lesions.



MRI showing left suprarenal mass (pheochromocytoma)



Helpful Laboratory Findings

1. Hyperglycemia (catecholamine-induced).
 2. Polycythemia (if EPO secreted).
 3. Family history → MEN2A/B, VHL, NF1.
- 

Treatment

1. Control Blood Pressure First:

- **Alpha-blocker: Phenoxybenzamine (before surgery)**

2. Then Beta-blocker:

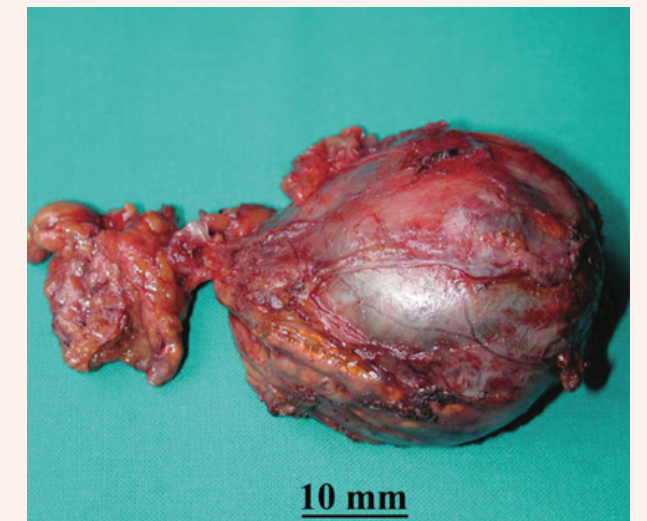
- **Added preoperatively to prevent hypertensive crisis**
-

3. Surgery:

- **Curative surgical resection after stabilization**

4. Post-op Care:

- **Monitor for hypotension → IV fluids or norepinephrine**
- **If bilateral adrenalectomy → glucocorticoid replacement**



B: Gross specimen of pheochromocytoma



Key Facts / Summary

- **Always** block alpha before beta to **avoid** hypertensive crisis.
 - Think of **pheochromocytoma** in patients with episodic headaches, sweating, and palpitations.
 - **Diagnosis:** ↑ catecholamines + imaging confirmation.
 - **Definitive cure:** Surgical removal.
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Thank you

اللهم علمنا ما ينفعنا، وانفعنا بما علمتنا، وزدنا
علمًا، ووفقنا لنكون سببًا في شفاء الناس ورحمة
بعبادك يا أرحم الراحمين

