Adrenal disorders

Simen Hagtvedt
5th grade MD

studyaid

Adrenal disorders

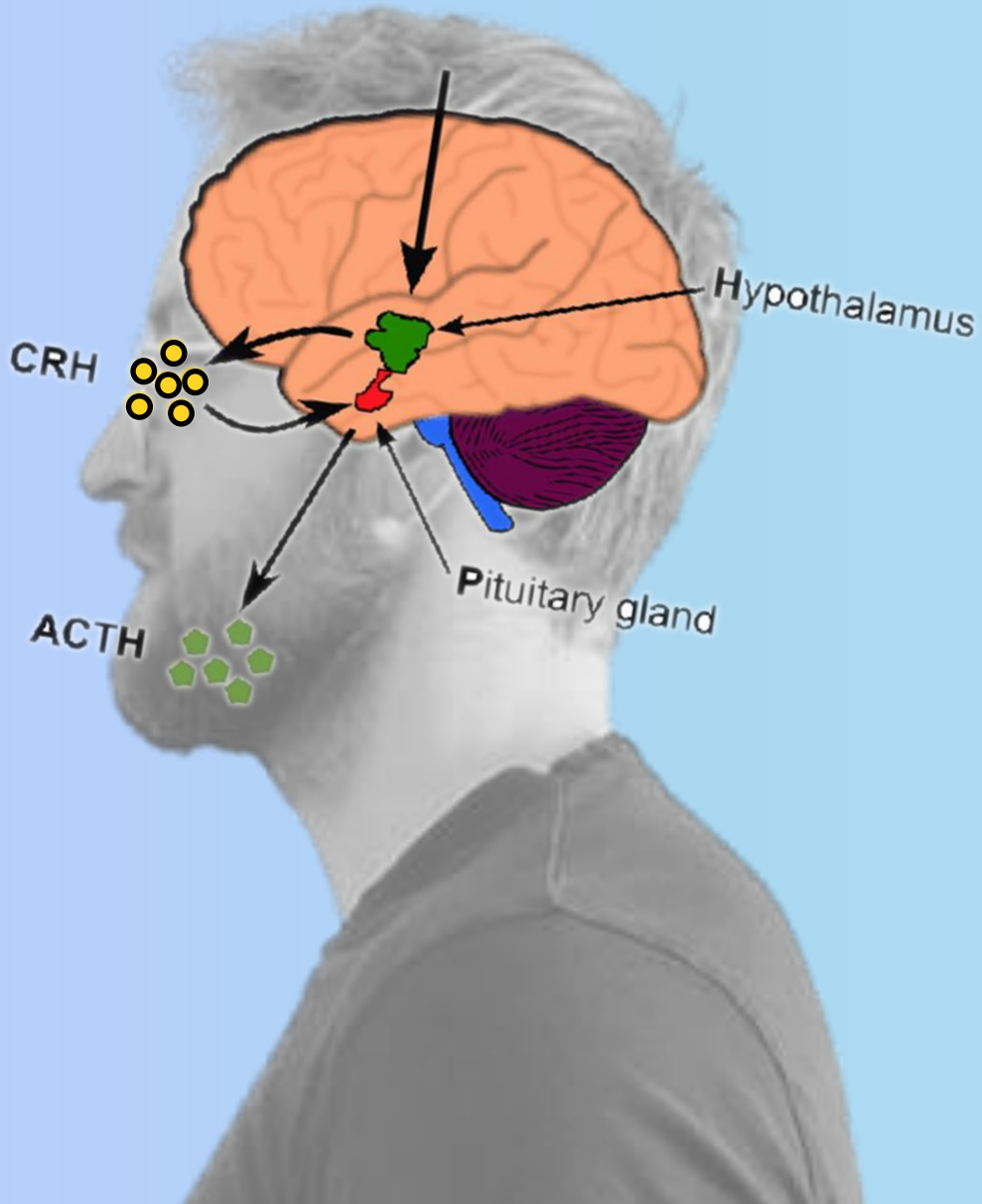
- Adrenal hypercorticolism/Cushing
- Adrenocortical insufficiency
- Hyperaldosteronism
- Pheochromocytoma

studyaid

Adrenal hypercorticolism/Cushing

studyaid

Pituitary adrenal axis

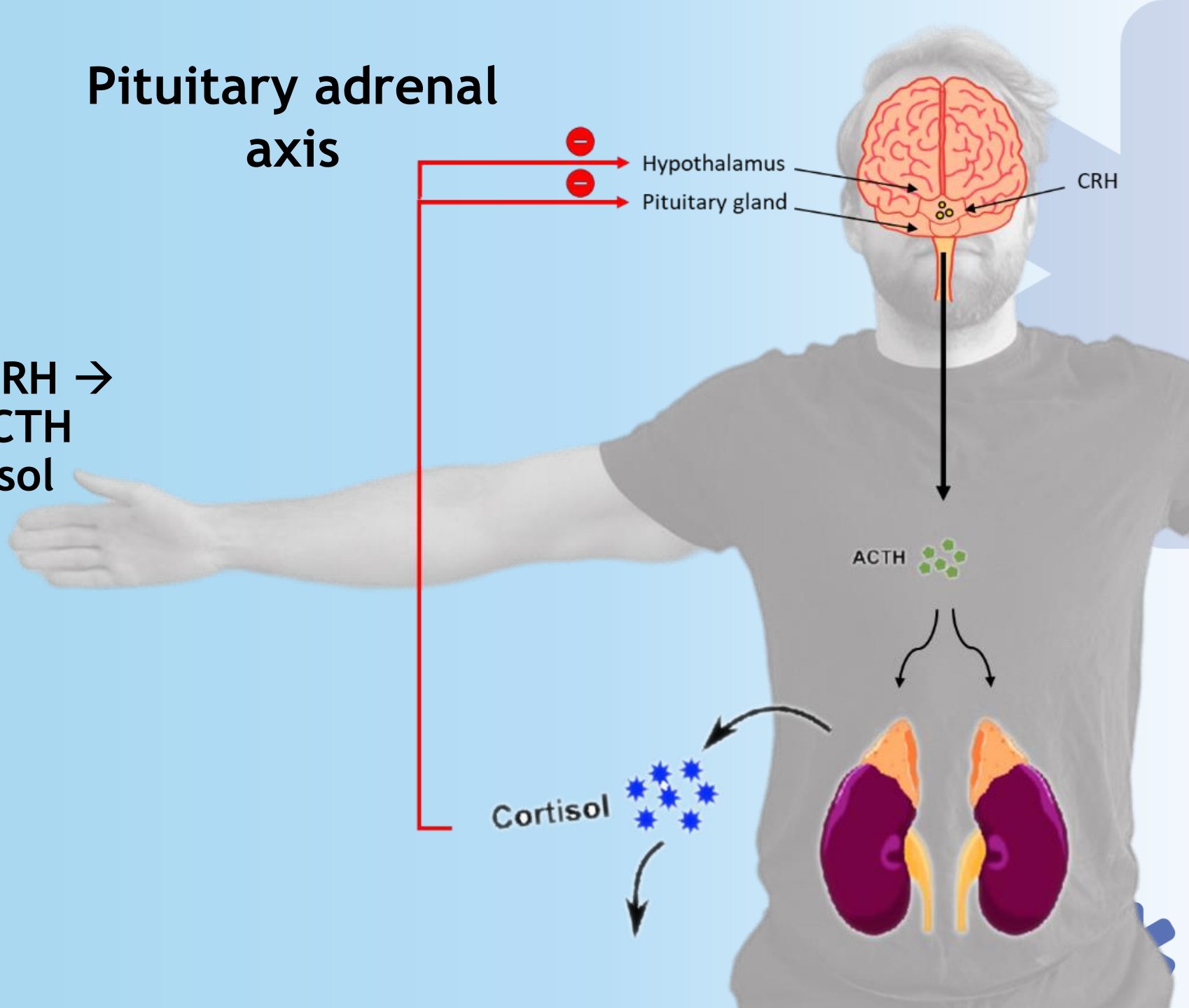


- Controls the secretion of cortisol
- Hypothalamus secretes **CRH** → ant. pituitary gland → **ACTH**

studyaid

Pituitary adrenal axis

- Controls the secretion of cortisol
- Hypothalamus secretes **CRH** → ant. pituitary gland → **ACTH** → adrenal gland → **cortisol**
- **Cortisol** causes a neg. feedback loop

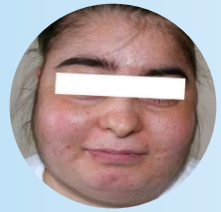


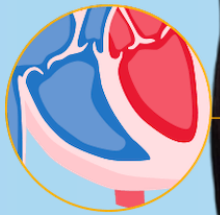
Major effect of excess cortisol	How?
Immunosuppresion	↓T cells and B cells in plasma, ↓ histamine, ↓ eosinophils and blocks neutrophils
Hyperglycemia	↑ Liver production of glucose, cells become more resistant to insulin → ↑ risk of DM
Hypertension	Important in maintaining normal blood pressure
Fat deposition	Destruction of fat cells → ↑ total cholesterol and triglycerides + stimulates fat deposition in face, around trunk and upper back
Muscle, bone and skin changes	Muscle atrophy → thin arms and legs Skin stretches → rapid fat deposition + thin skin Bad for bones → inhibits osteoblasts → hypercalemia
Reproductive effect	↓ LH and FSH **Woman** = irregular menses or absence of menstruation **Men** = low testosterone and low sperm count

studyaid

Thin extremities

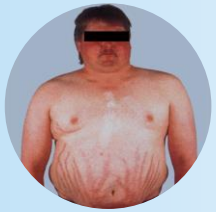
Typical “moon face”



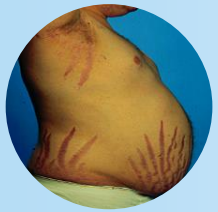


Hypertension

Central obesity



Abdominal striae



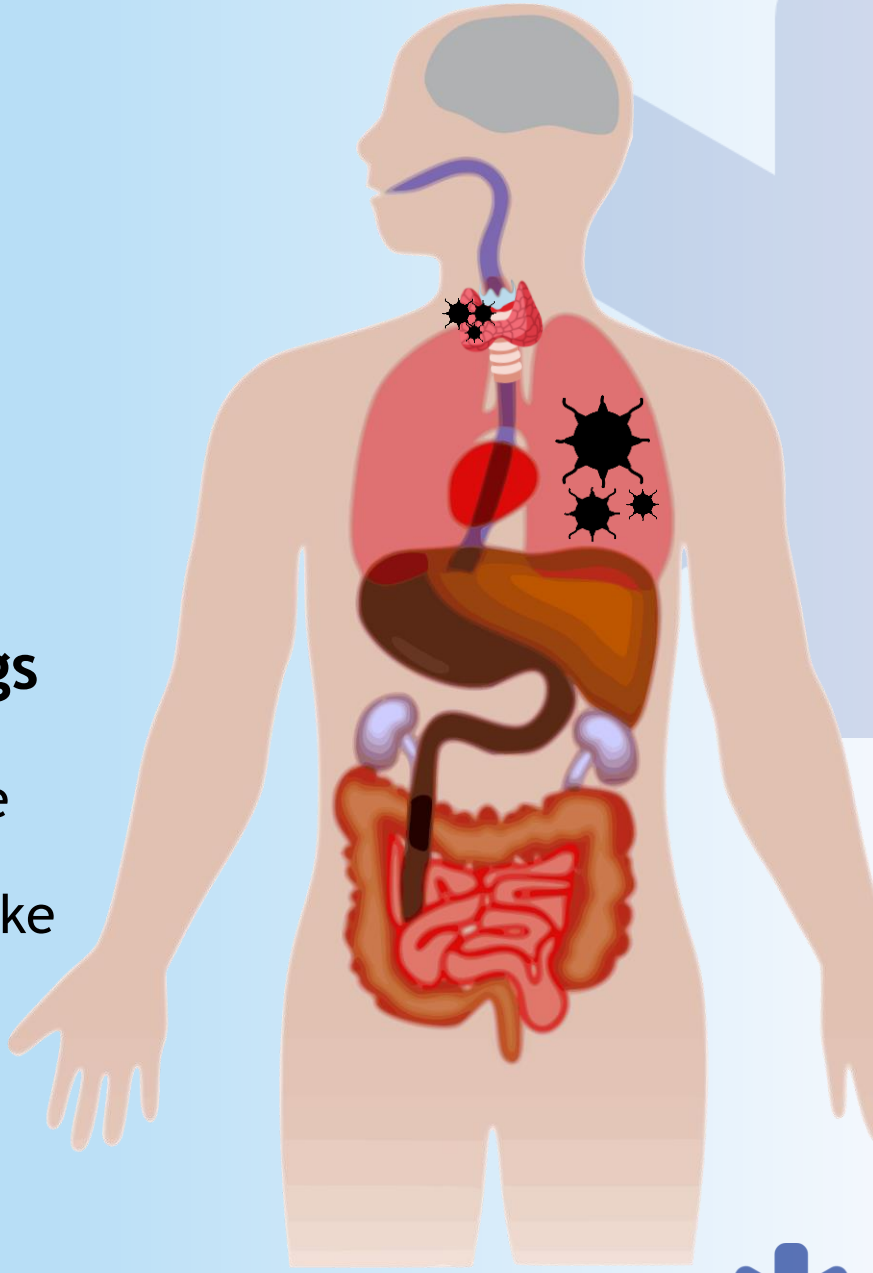
Menstrual irregularities

Hyperglycemia

studyaid

Causes of Adrenal hypercorticolism/Cushing

- Exogenous glucocorticoids – most common cause
  - Adrenal gland working fine - we just give the patient to much oral steroids
  - Used for its immunosuppressive effect
- Overproduction of ATCH by pituitary gland (**Cushings disease**)
  - **Cushing syndrome** = excess cortisol from outside or inside the body.
  - **Cushings disease** = pituitary tumor causes the body to make too much cortisol.
- Overproduction of ACTH by ectopic tumor
  - Small cell lung cancer (most common)
- Overproduction of cortisol by adrenal adenoma

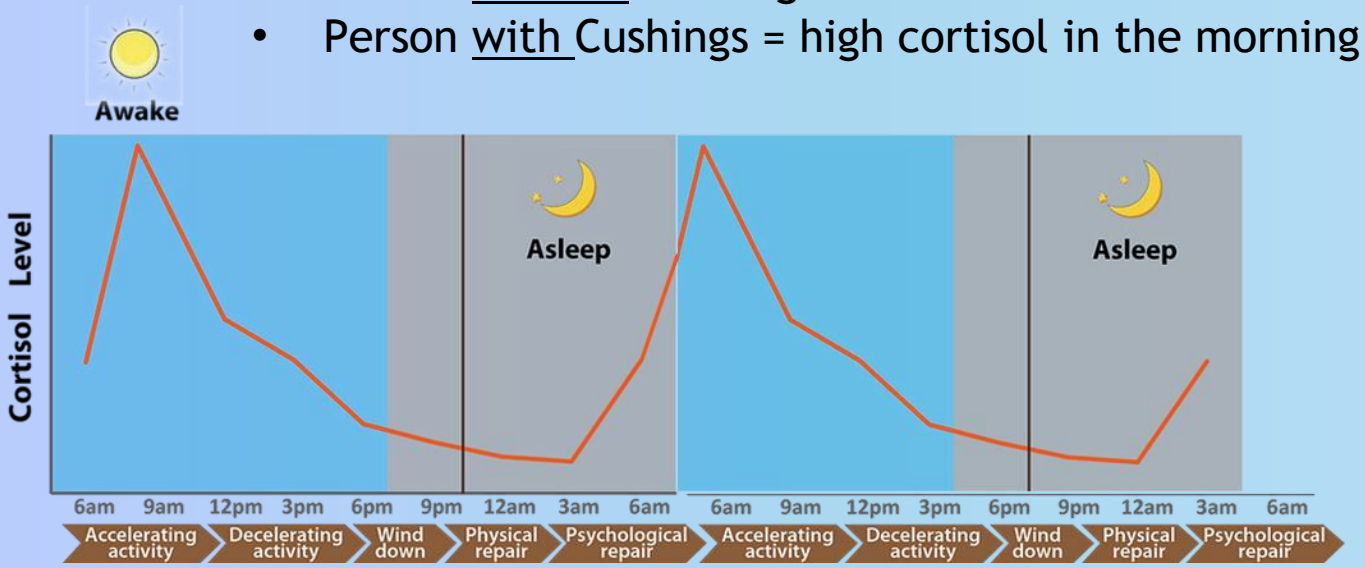


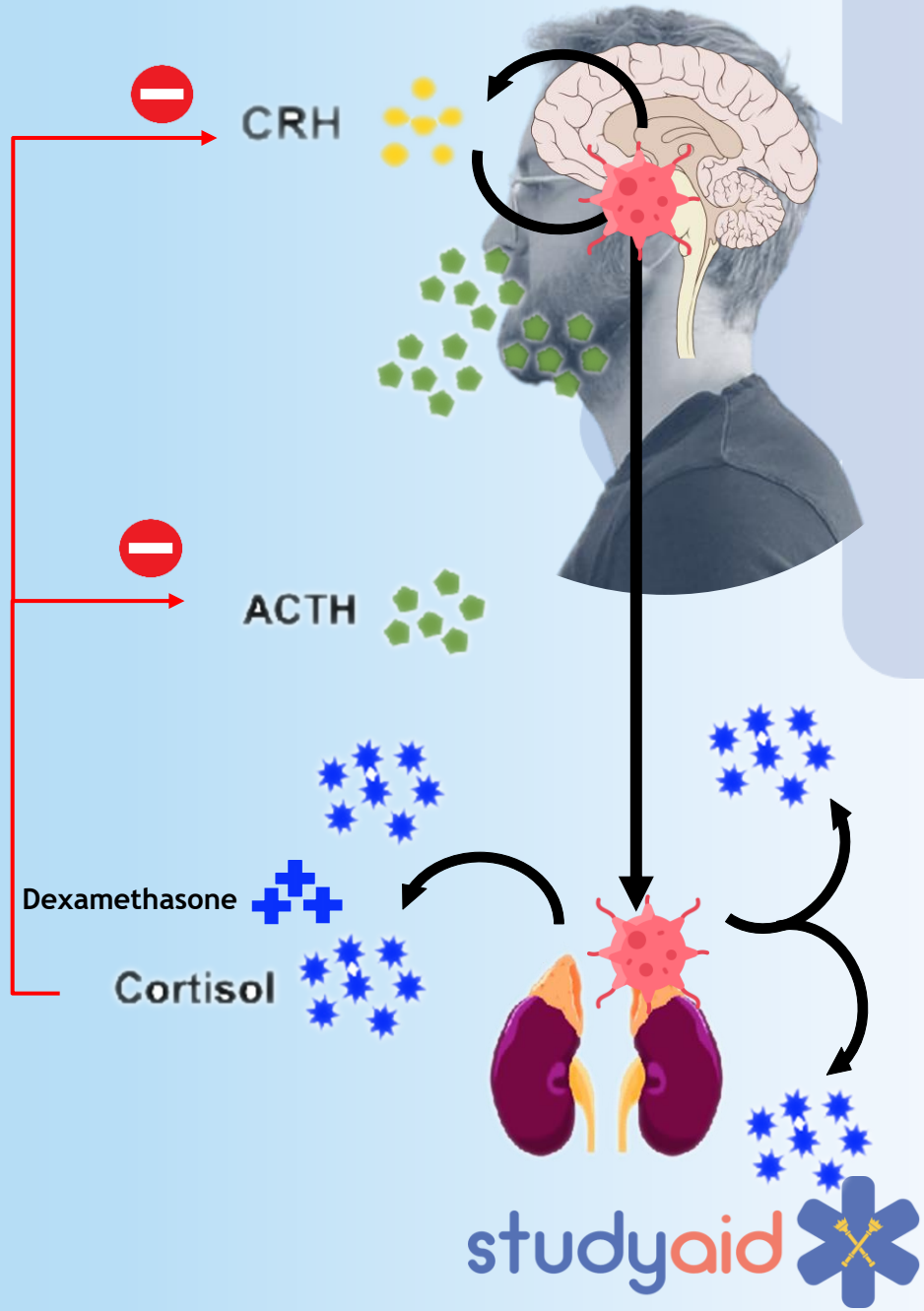
studyaid

Workup for Cushings

1st – check for elevation of cortisol

- 24 hour urinary cortisol excretion test
- Late-night serum cortisol
  - Cortisol goes up and down during the day
- Low dose dexamethasone suppression test
  - Dexamethasone = «synthetic cortisol»
  - Give a **low** dose of dexamethasone (1mg) at bed time
  - Person without Cushings = low cortisol in the morning
  - Person with Cushings = high cortisol in the morning



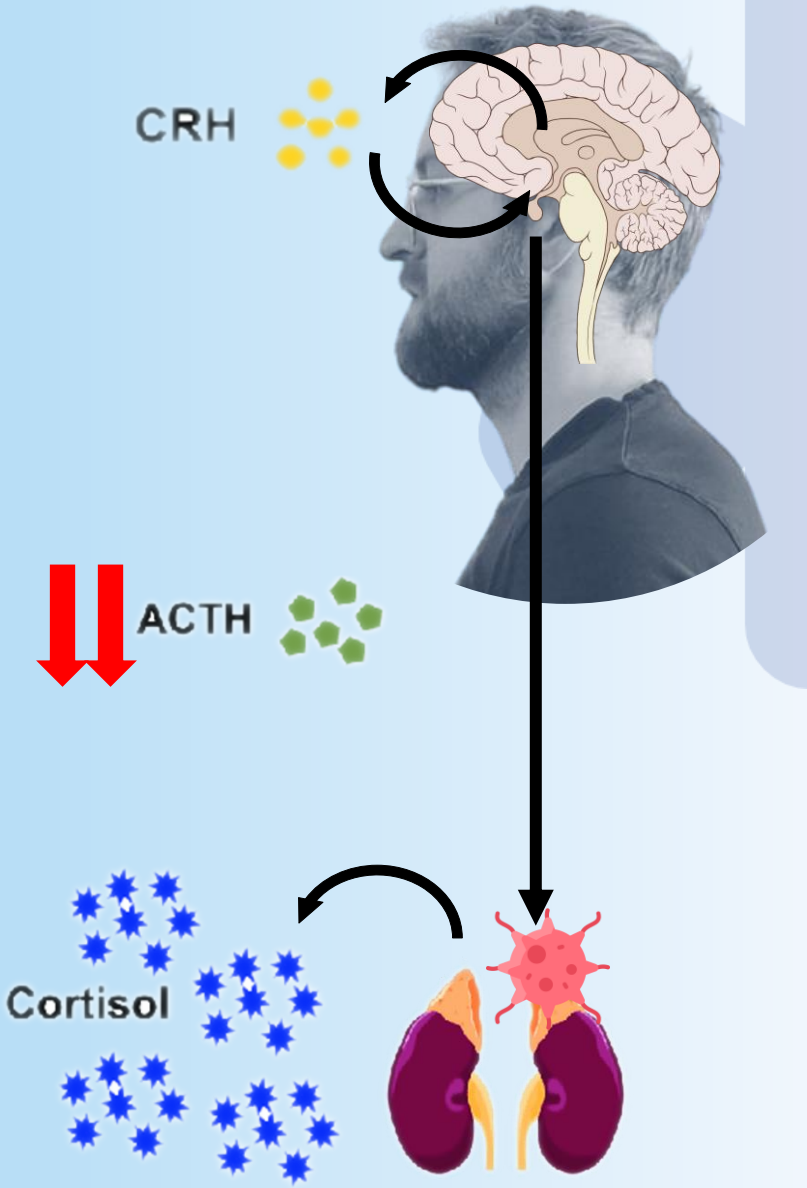


studyaid

Workup for Cushings

2nd

- Measure plasma ACTH – to find out if it`s:
  - **ACTH independent** = low plasma ACTH → seen in adrenal adenoma
  - **ACTH dependent** = normal/high plasma ACTH → seen in Cushings disease and ectopic adenoma

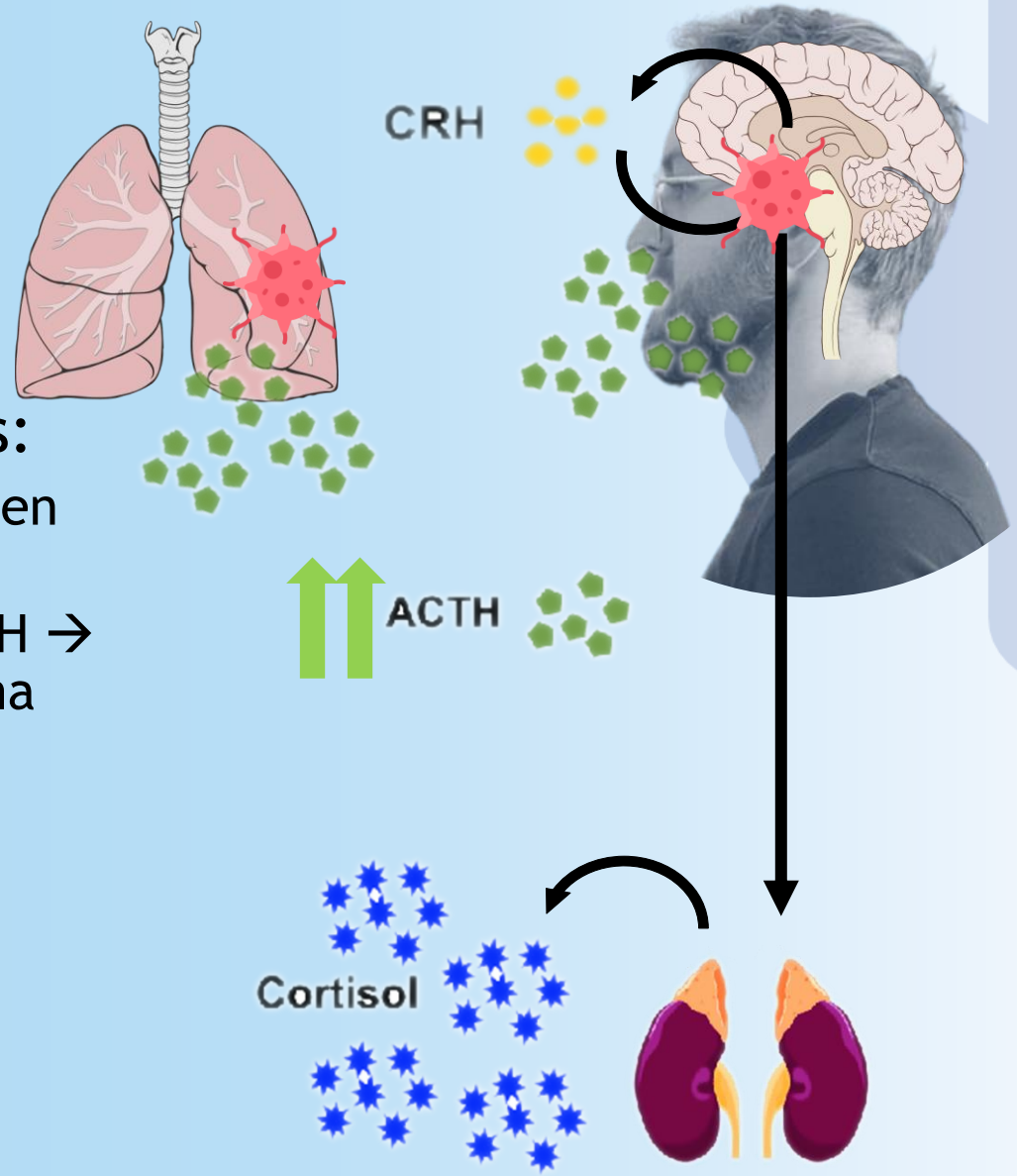


studyaid

Workup for Cushings

2nd

- Measure plasma ACTH – to find out if it`s:
  - **ACTH independent** = low plasma ACTH → seen in adrenal adenoma
  - **ACTH dependent** = normal/high plasma ACTH → seen in Cushings disease and ectopic adenoma

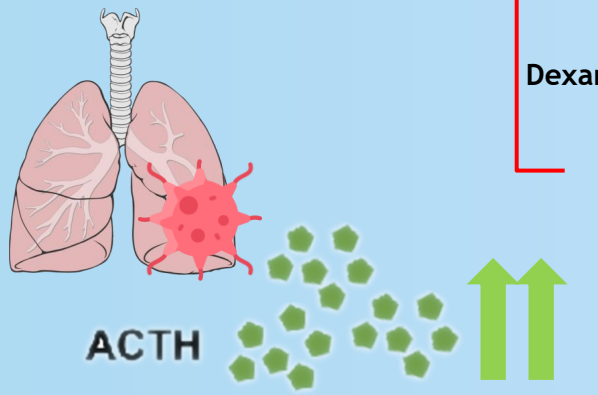


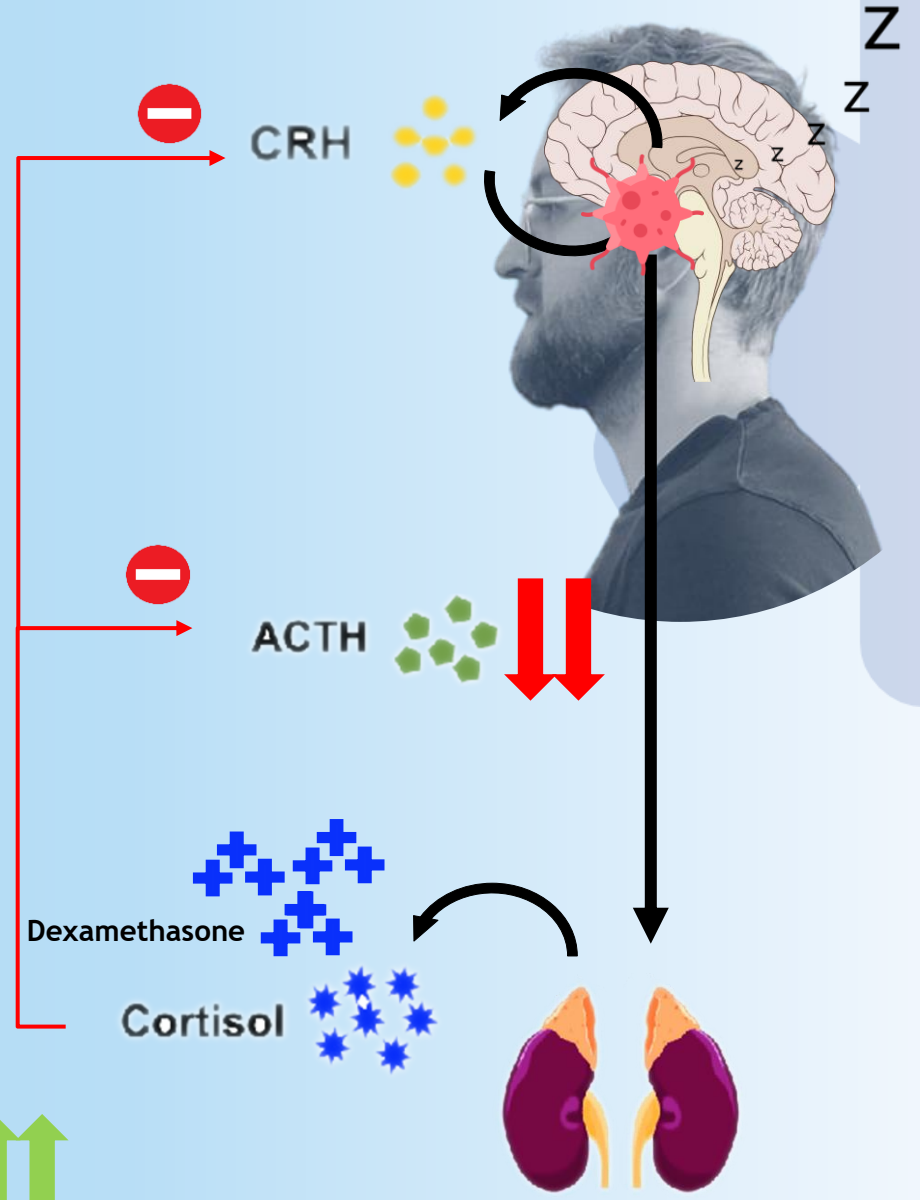
studyaid

Workup for Cushings

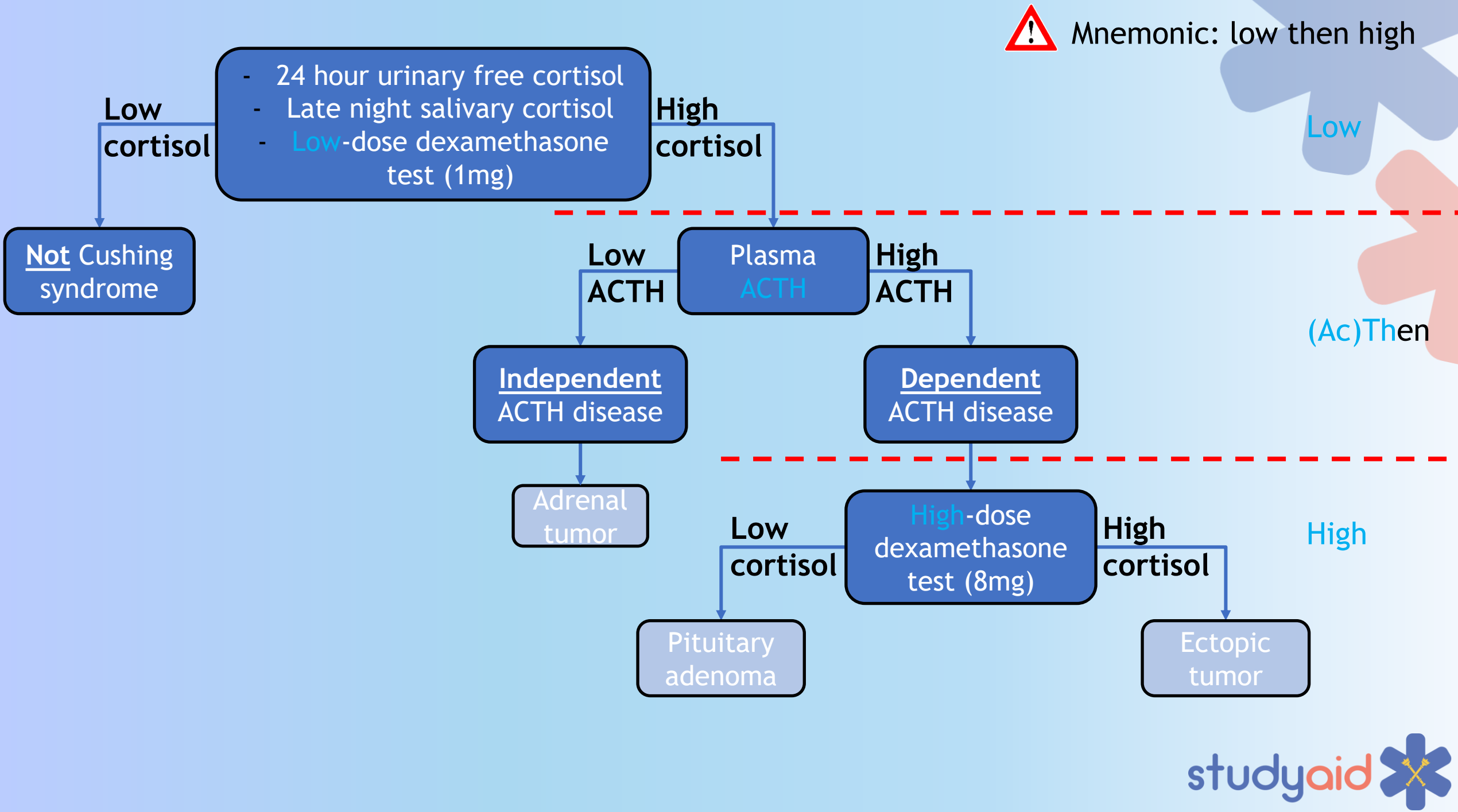
3rd

- High dose dexamethasone test
  - Give a **high** dose of dexamethasone (8mg) at bed time
  - Low cortisol = pituitary adenoma
  - High cortisol = ectopic





studyaid

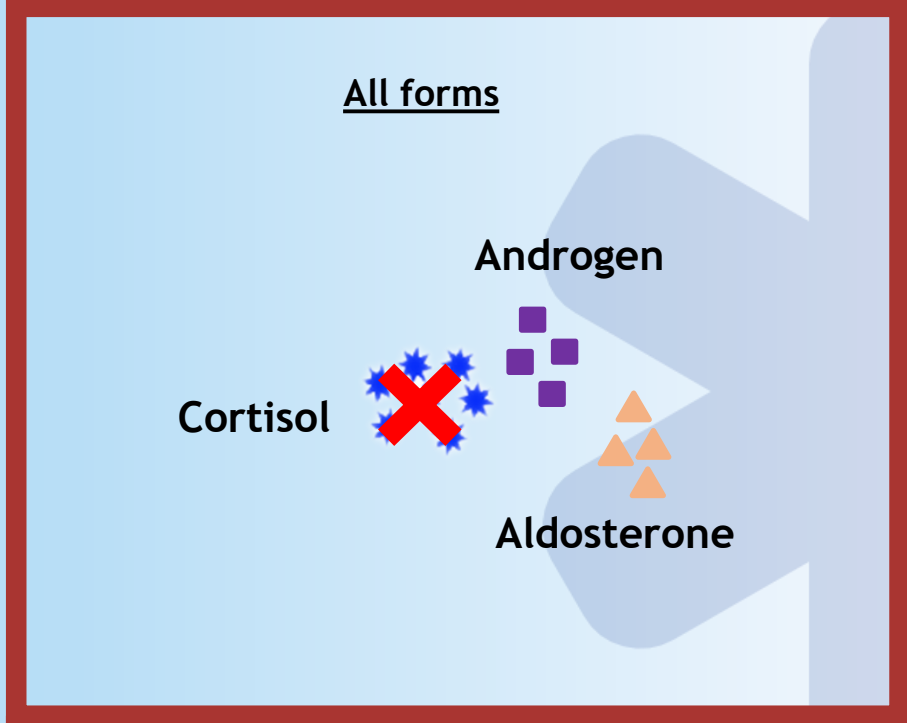

Mnemonic: low then high
- 24 hour urinary free cortisol
- Late night salivary cortisol
- Low-dose dexamethasone test (1mg)
Low cortisol
High cortisol
Not Cushing syndrome
Plasma ACTH
Low ACTH
High ACTH
Independent ACTH disease
Dependent ACTH disease
Adrenal tumor
High-dose dexamethasone test (8mg)
Low cortisol
High cortisol
Pituitary adenoma
Ectopic tumor
Low
(Ac)Then
High
studyaid

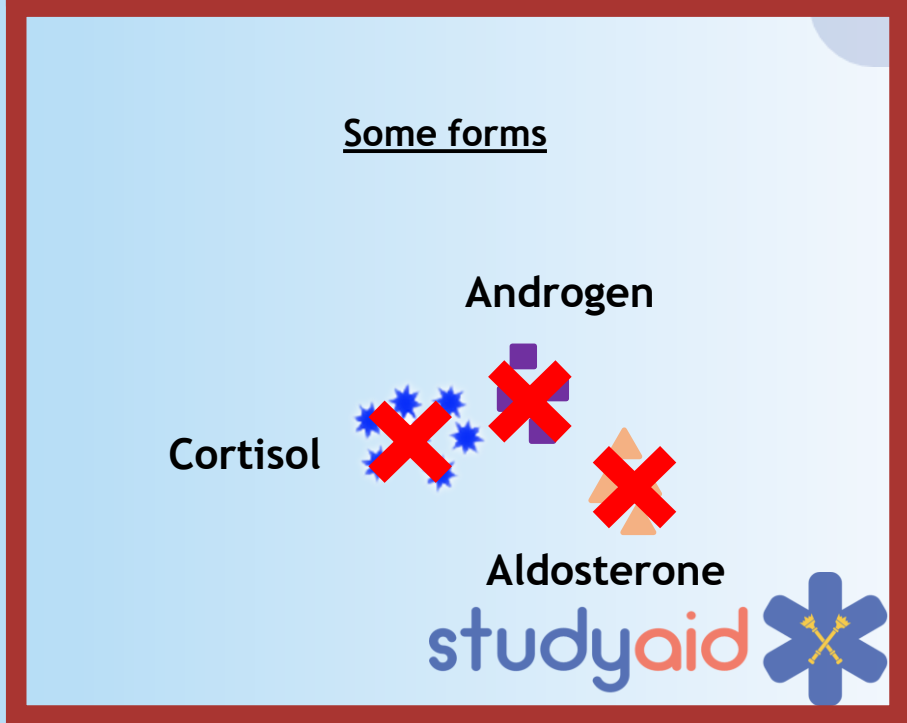
Adrenocortical insufficiency

Adrenal insufficiency

- Loss of one or more adrenal hormones
- All forms of adrenal insufficiency → loss of production of **glucocorticoids (cortisol)** some forms also involves loss of mineralcorticoids (aldosterone) and some androgen (DHEA)
- Can be seperated into:
  - Primary
  - Secondary
  - Tertiary

} Central adrenal insufficiency

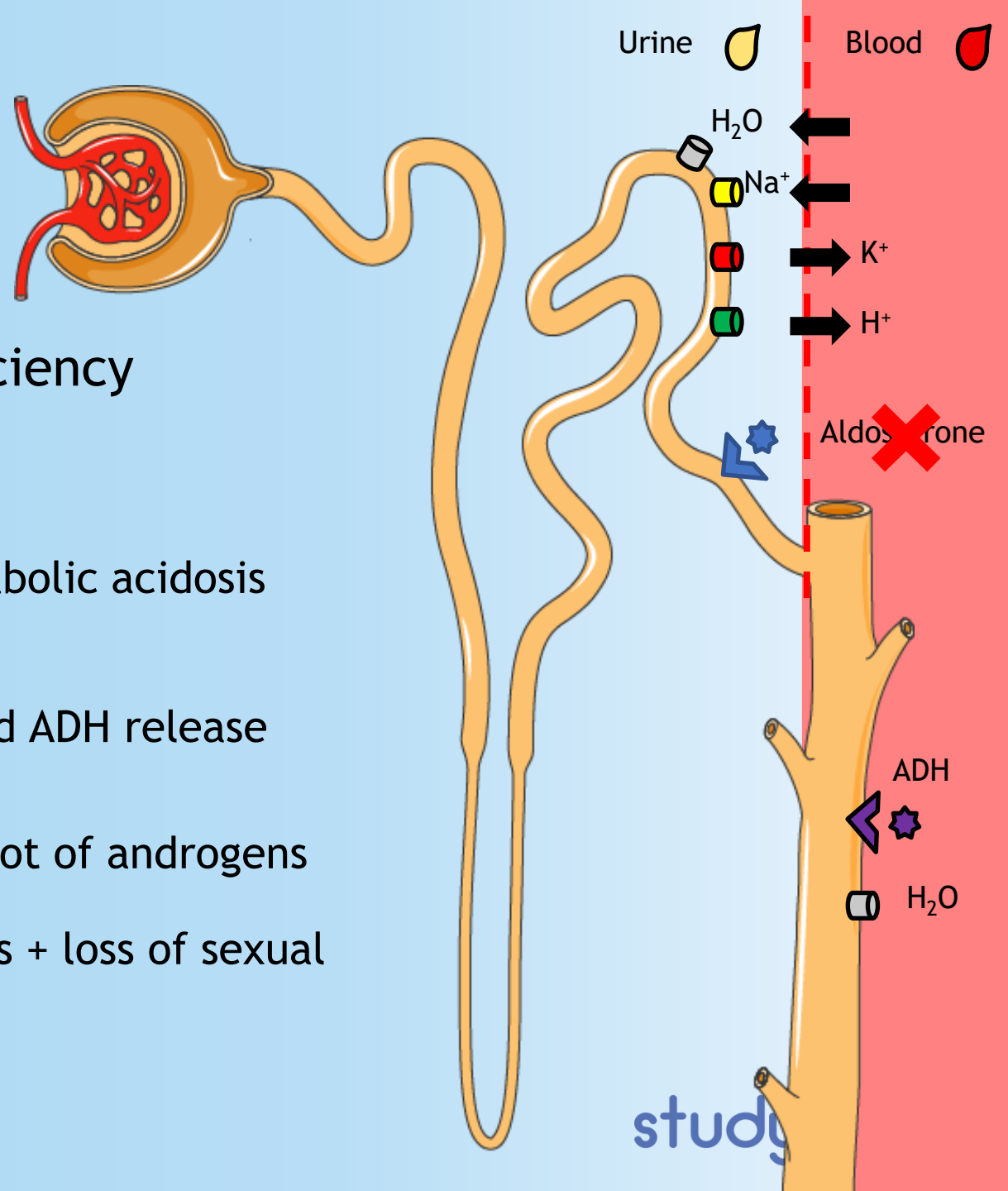




Glucocorticoid deficiency	How?
Hypotension	Cortisol maintains blood pressure → therefore ↓cortisol = hypotension
Hypoglycemia	Cortisol normally provide glucose to the body → therefore ↓cortisol = hypoglycemia
N/V + abdominal pain	
Adrenal crisis	Acute onset of life-threatening shock due to the lack of cortisol.

studyaid

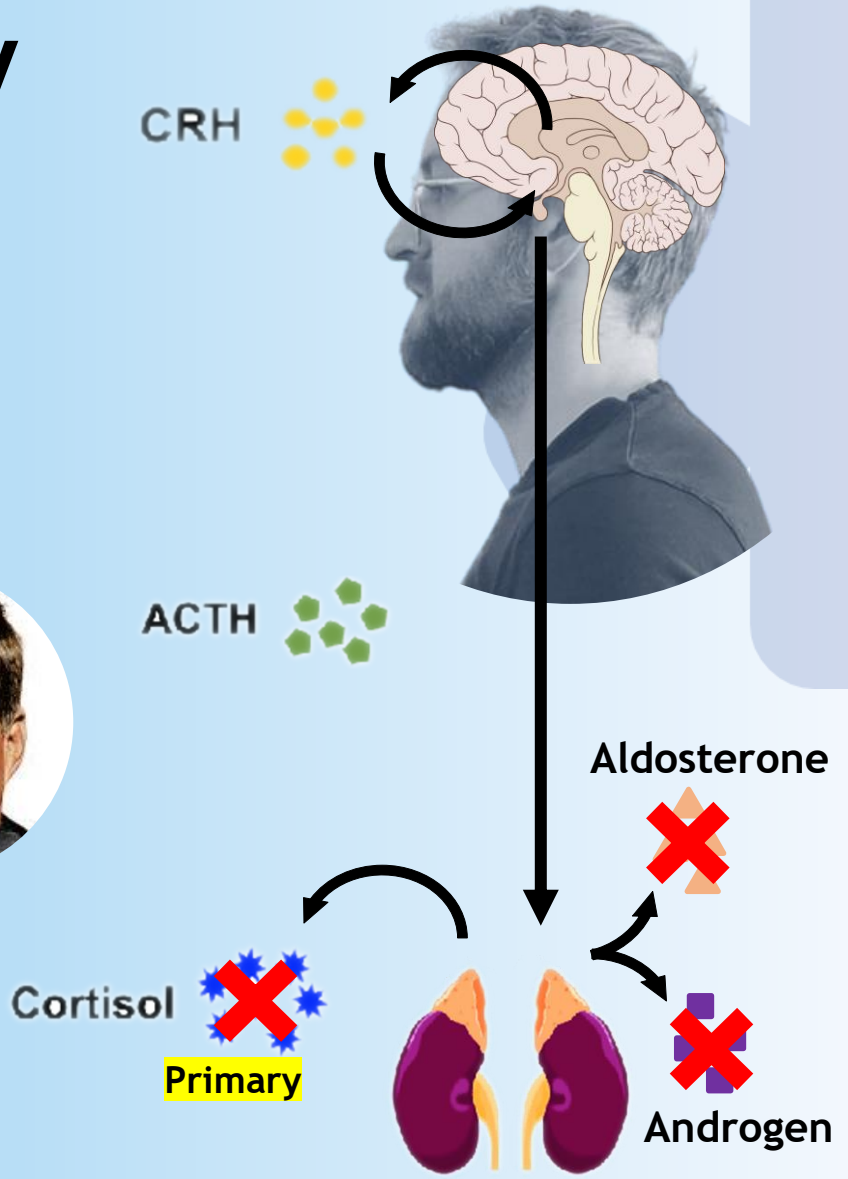
Aldosterone and androgen deficiency



- Mineralcorticoid (aldosterone) deficiency
  - Aldosterone normally:
    - **increase** Na^+
    - **decrease** K^+ and H^+
  - ↓aldosterone = hyperkalemia and metabolic acidosis (because of ↑ H^+)
  - Salt craving – due to the loss of Na
  - Increased free water – due to increased ADH release
- Androgen deficiency
  - Males usually no significant impact – alot of androgens produced by testes
  - Females decreased axillary, pubic hairs + loss of sexual interest

Primary adrenal insufficency (Addisons disease)

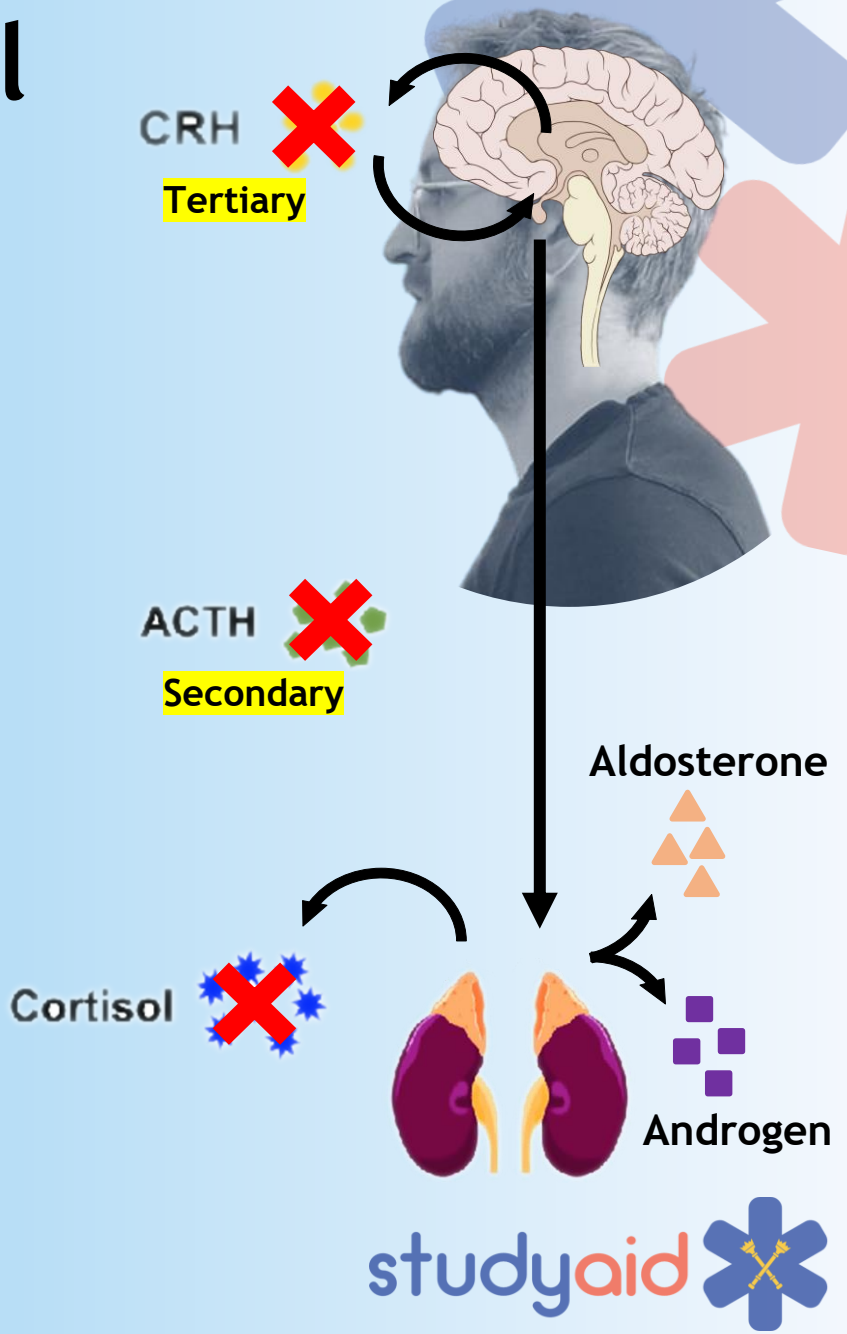
- Destruction of the entire adrenal gland → loss of **all** adrenal hormones
- Can be referred to as **Addisons disease**
- Symptoms
  - Salt craving
  - Hyponatremia
  - Hyperkalemia
  - **Hyperpigmentation** → ↑ACTH causes an ↑MSH, because they share the same precursor (POMC) → patient will present with a **sun tan** (John F. Kennedy)



studyaid

Secondary or tertiary adrenal insufficency

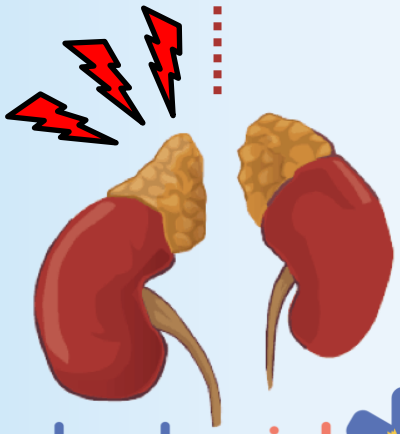
- Secondary:
  - Loss of ACTH from the pituitary gland
- Tertiary:
  - Loss of CRH from the hypothalamus
- Both of these conditions = **loss of cortisol** (glucocorticoid) and **sometimes** androgens
- Can be due to ACTH deficiency due to patient taking chronic glucocorticosteroid therapy
- Symptoms:
  - Hypotension → vasodilation (less prominent)
  - **NO** hyperpigmentation – due to **low** ACTH



studyaid

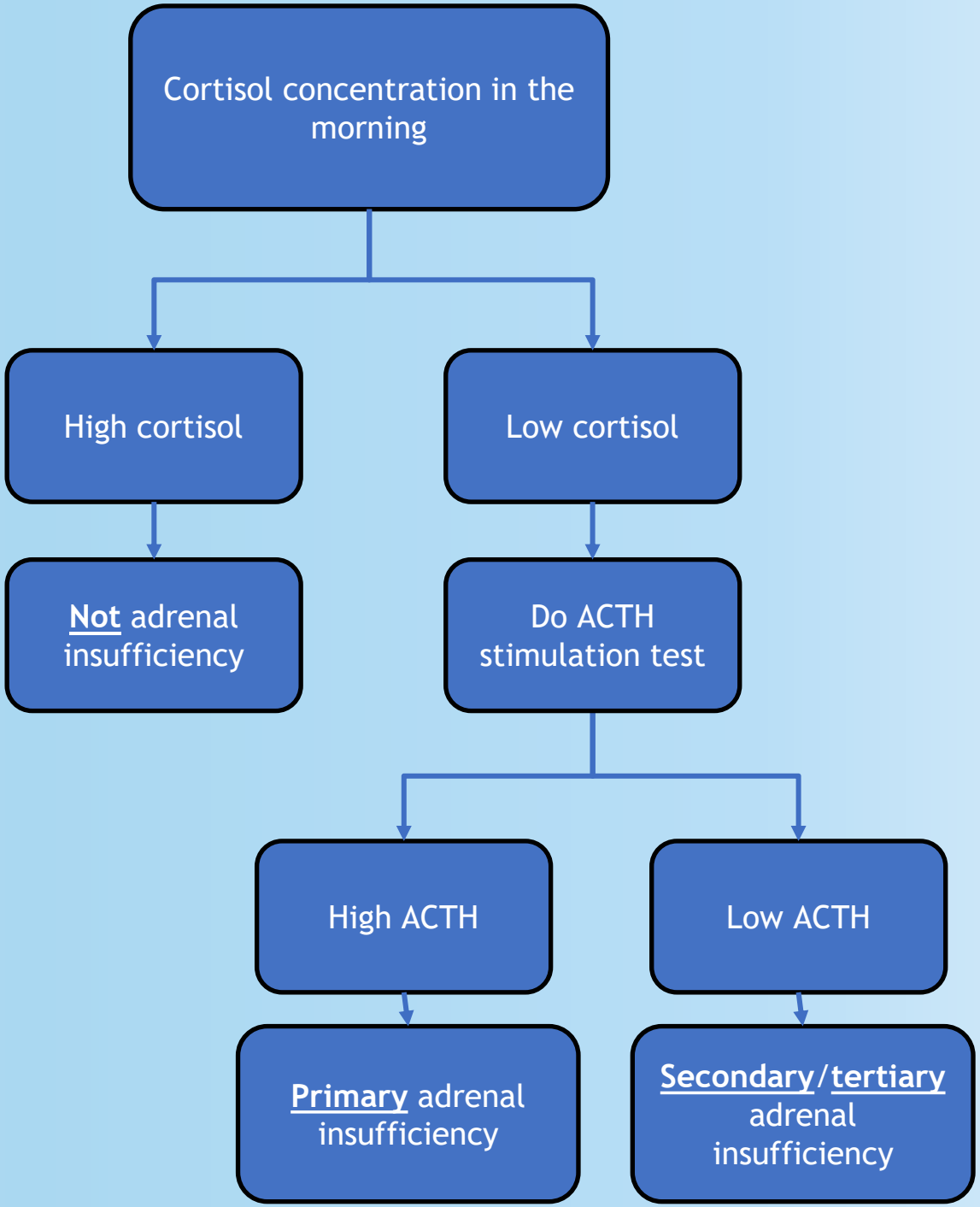
What is the cause of primary adrenal insufficency?

- Autoimmune adrenalitis
  - **Most common** → J.F.K
  - Autoimmune destruction of adrenal gland by antiadrenal antibodies (antibodies to the **21-hydroxylase enzyme**)
- Infectious adrenalitis
- Hemorrhagic infarction
  - Bleeding into the adrenal gland → underperfusion and death of adrenal gland
  - Usually caused by sepsis (called "Waterhouse-Friedrischen syndrome")
- Metastatic cancer
  - Rare – because over 90% of both adrenal glands must be destroyed



studyaid

Diagnosis

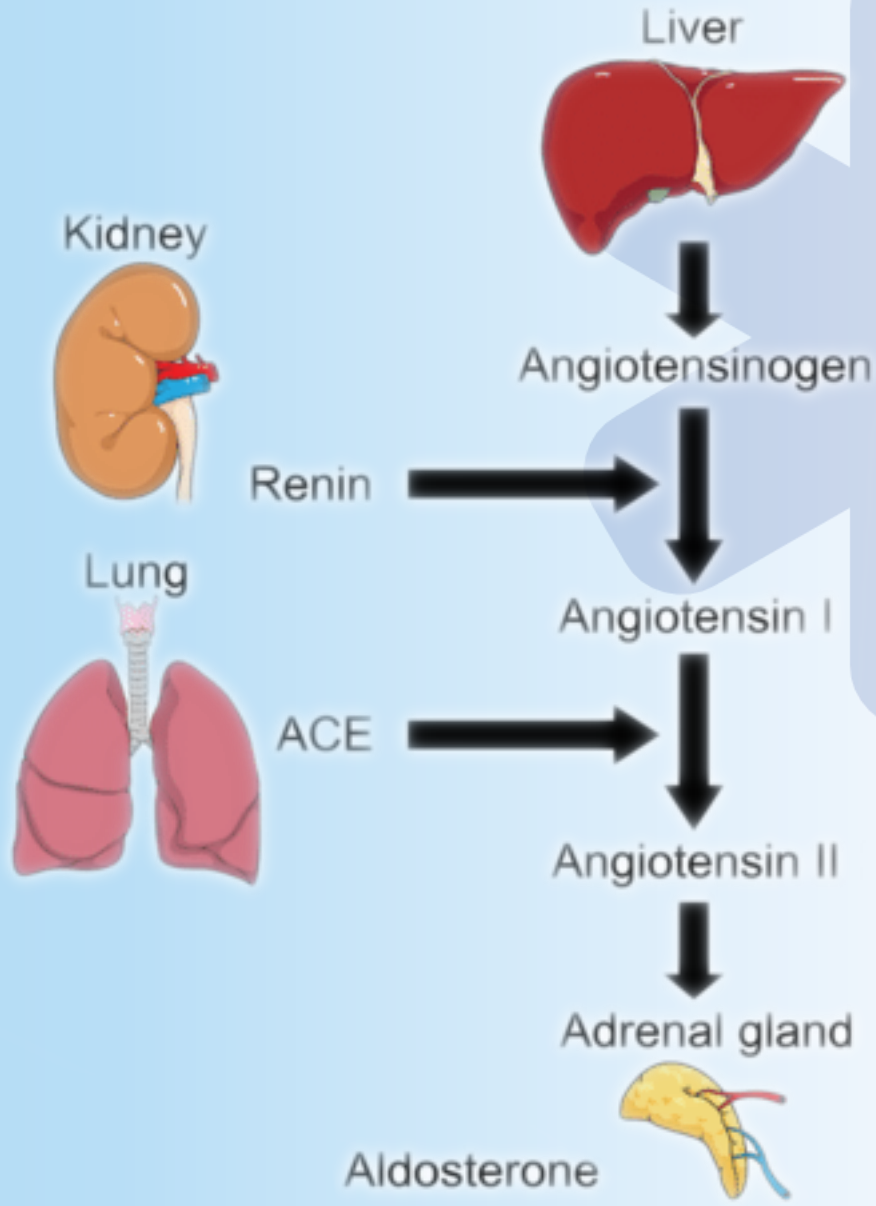


studyaid

Hyperaldosteronism

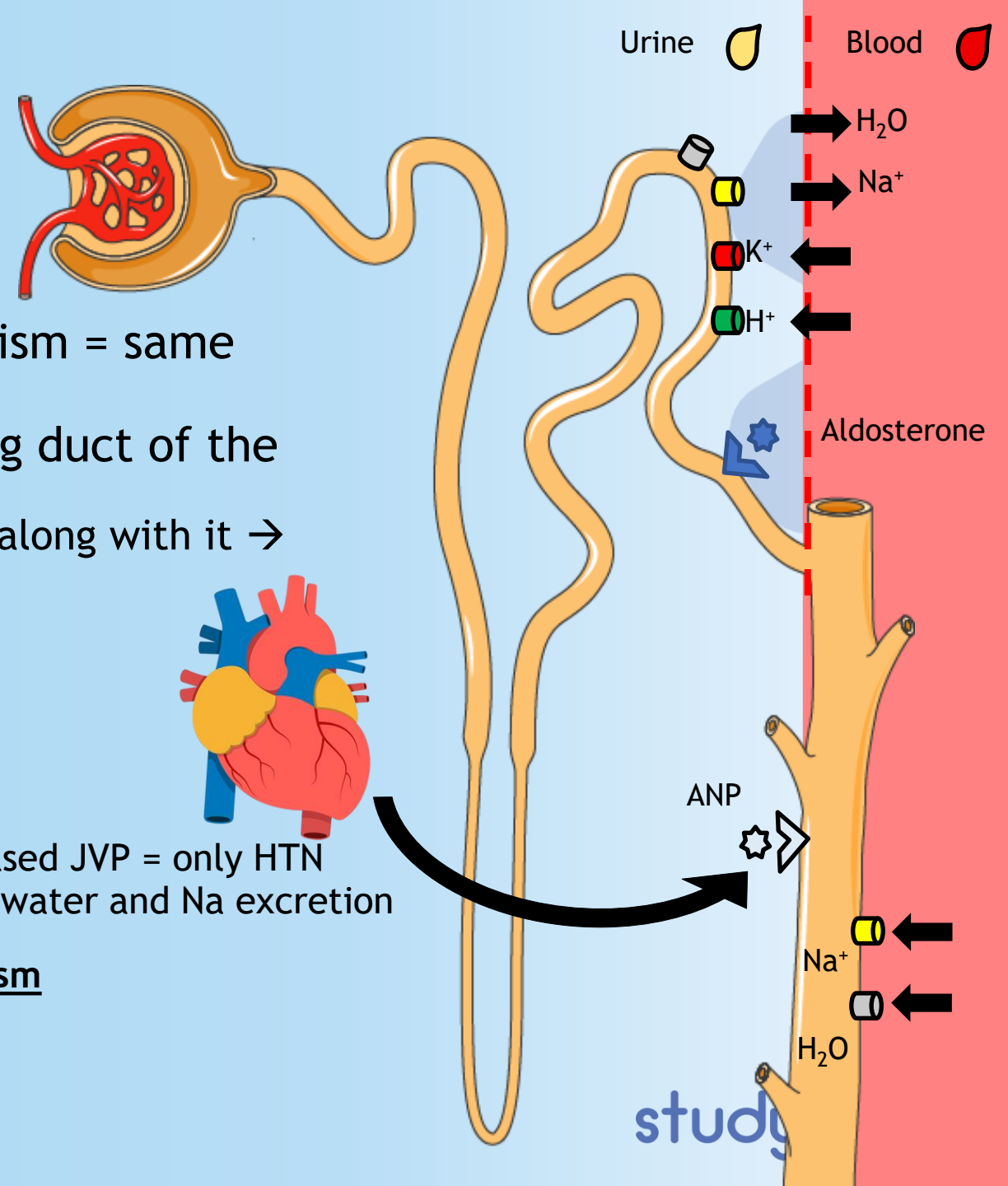
Hyperaldosteronism

- **Excess** secretion of **aldosterone** from the adrenal gland
- Primary hyperaldosteronism
  - Can be caused by:
    - Adrenal adenoma (also called **Conn´s syndrome**) = small portion of the gland overproduce aldosterone
    - Bilateral idiopathic adrenal hyperplasia = the glands enlarge and produce to much aldosterone
    - Adrenal carcinoma – rare
- Secondary hyperaldosteronism
  - Can be caused by:
    - Renal artery stenosis → Renin-Angiotensin-Aldosterone System



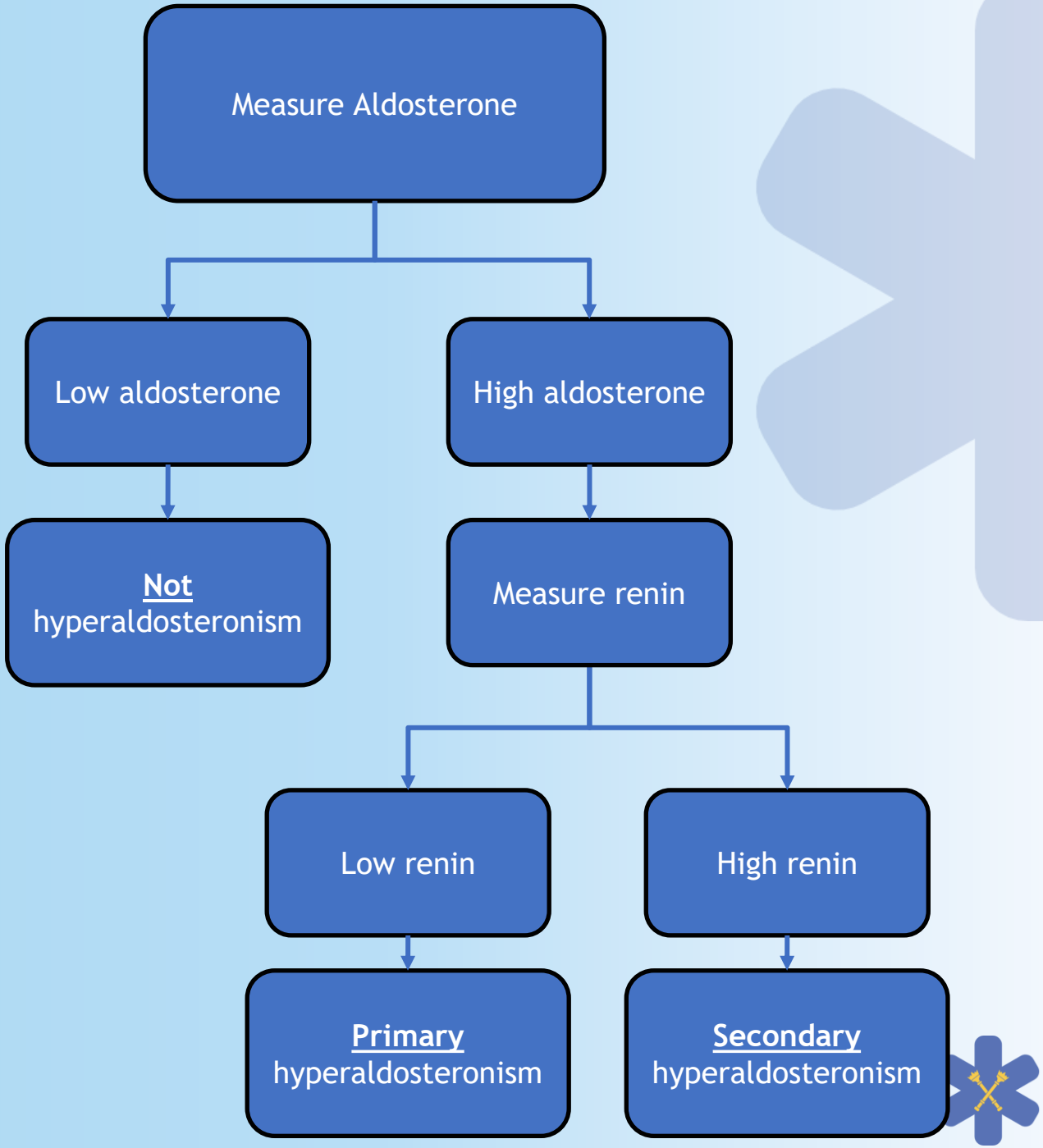
Clinical features

- Primary and secondary hyperaldosteronism = same symptoms but different markers
- Aldosterone = functions at the collecting duct of the nephrones
  - **Na <u>increase</u>** → H2O loves Na and follows along with it → hypertension
    - Resistant to hypertension medication
  - **K <u>decrease</u>** → hypokalemia
  - **H <u>decrease</u>** → metabolic alkalosis
- Aldosterone escape phenomenon
  - Normal volume on physical exam
    - No swelling, no pulmonary rails. no increased JVP = only HTN
    - Caused by heart releasing → ANP causing water and Na excretion – combats aldosterone
    - **<u>Not seen in secondary hyperaldosteronism</u>**



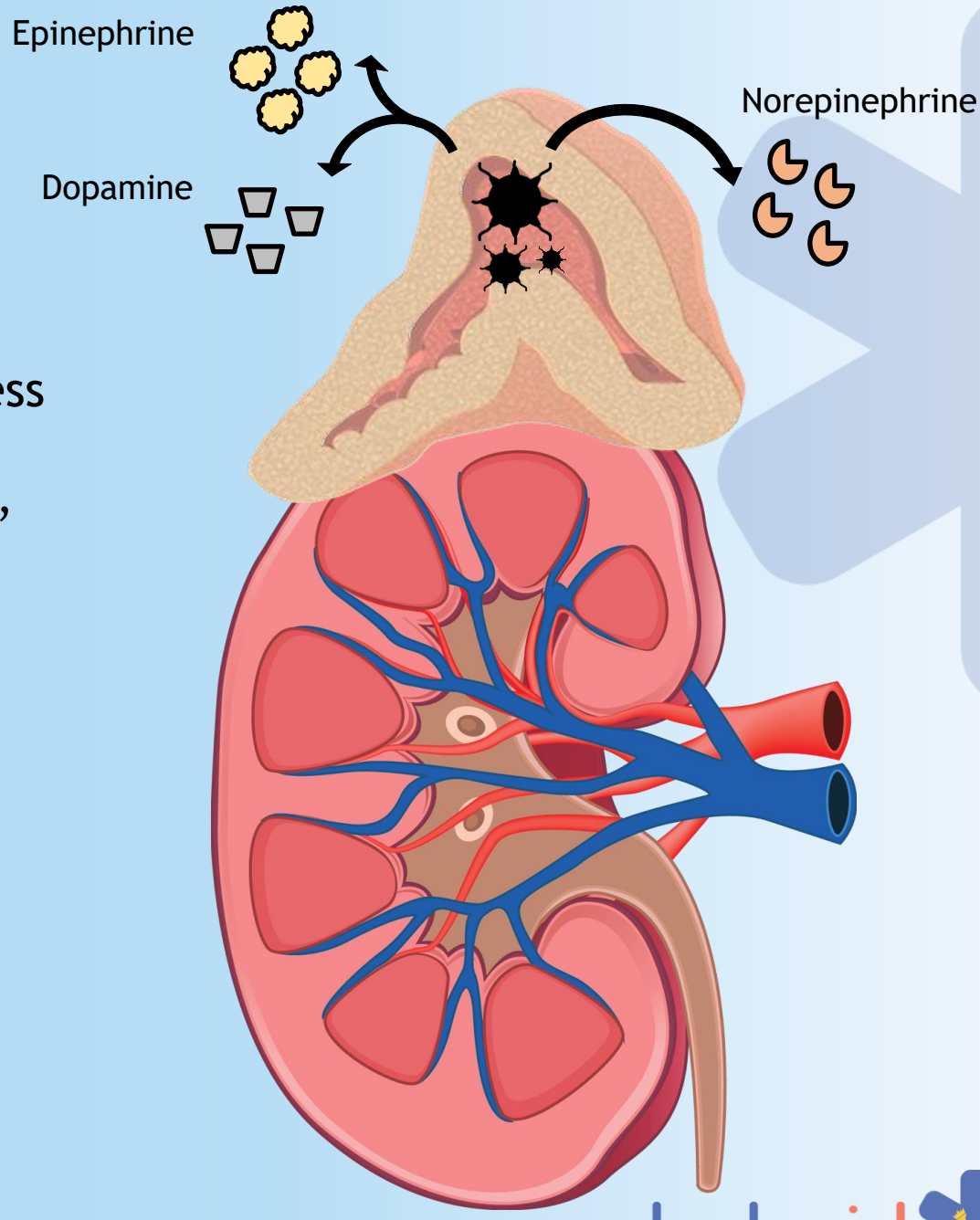
Diagnosis

- Typical patient = patient with hypertension that is not responding to any medication and with hypokalemia
- Two blood tests:
  - Plasma **renin** activity (PRA)
  - Plasma **aldosterone** concentration (PAC)
  - Take the ratio of these two
    - If high = primary hyperaldosteronism
    - If low = secondary hyperladosteronism



Pheochromocytoma

Pheochromocytoma

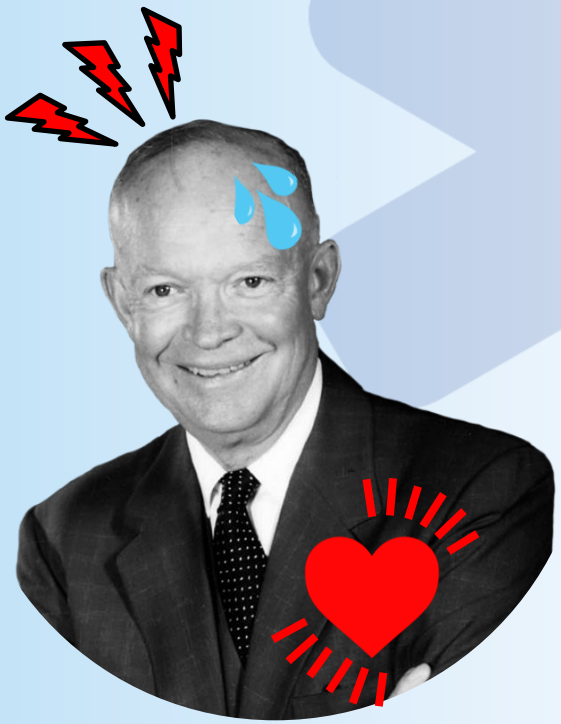


- Tumor in the adrenal medulla, causing an excess of cathecolamine
- **Cathecolamine** = epinephrine, norepinephrine, dopamine
- Stimulates the sympathetic nervous system
- Tumor secreting epinehrine = episodic hypertension
- Tumor secerting norepinephrine = less often hypertension, more often hyperglycemia and glycosuria
- Called the «**10% tumor**»
  - 10% malignant
  - 10% occur in children
  - 10% occurs outside of the abdomen
  - 10% are bilateral

studyaid

Clinical features

- Norepinephrine and epinephrine activates adrenergic receptors
- **α1** = causes HTN
- **β1** = causes an increase in HR
- **β2** = causes bronchodilation
- + ↑Glucose, ↑fat metabolism
- Symptoms are fluctuating in tumors
- **The classic triad:** headache, tachycardia and excessive sweating

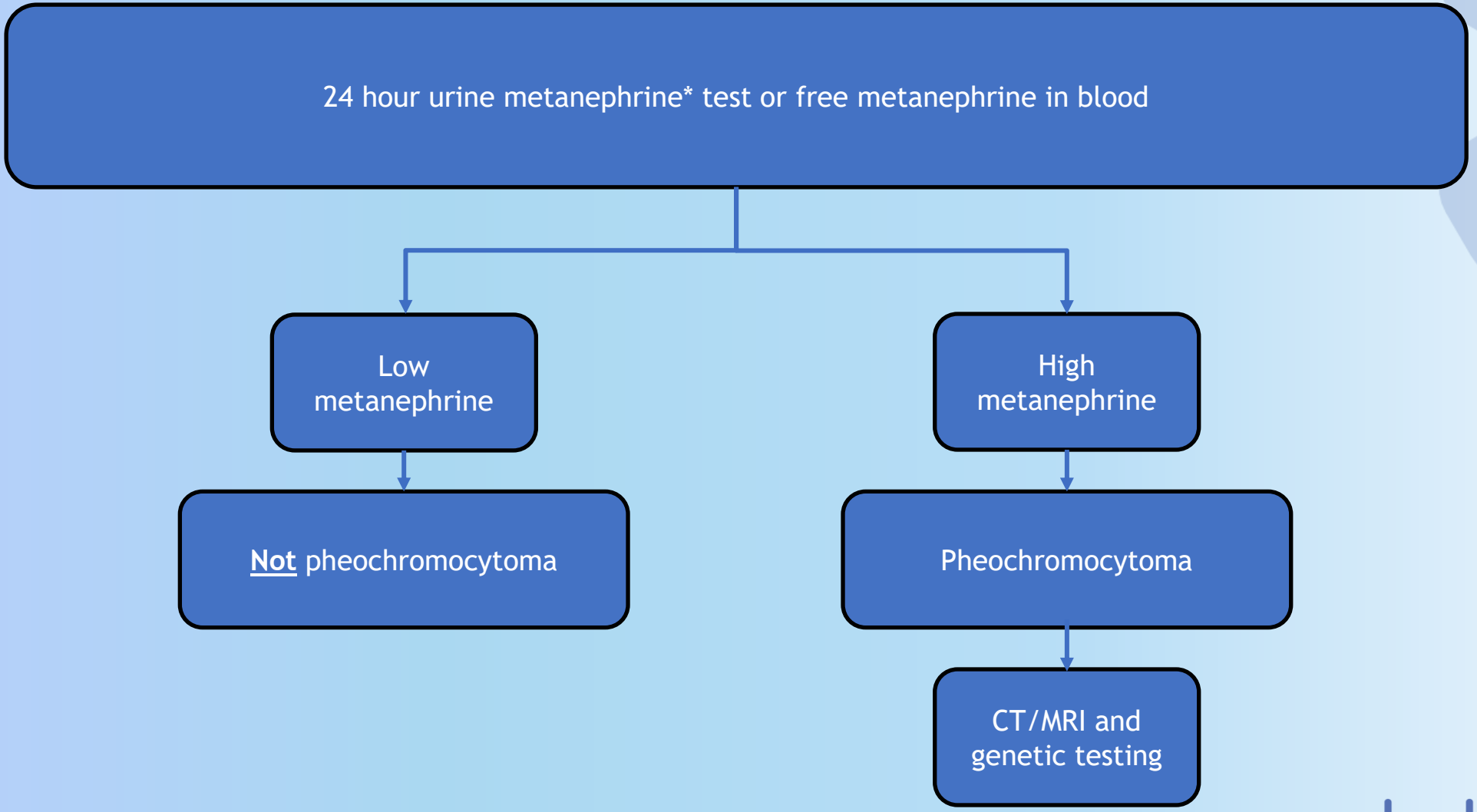


Dwight D. Eisenhower, 34th president of the United States had an undiagnosed pheochromocytoma

studyaid

*Metanephrine = breakdown product of cathecolamine

Diagnosis



studyaid