
بسم الله الرحمن الرحيم


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The reference range for serum potassium level

3.5 - 5 mEq/L.

Potassium

- < 2 mo — 3 - 7 mmol/L
- 2 -12 mo — 3.5 - 6 mmol/L
- >12 mo — 3.5 - 5 mmol/L

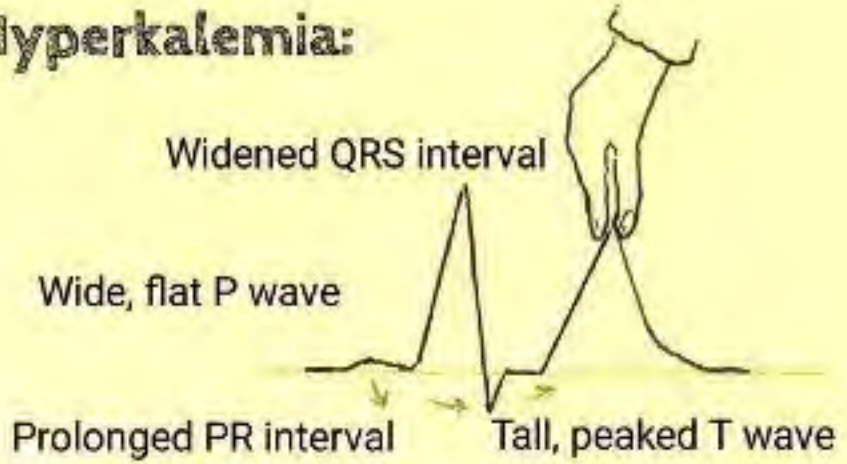
Hypokalemia

Defined as a potassium level < 3.5 mEq/L.

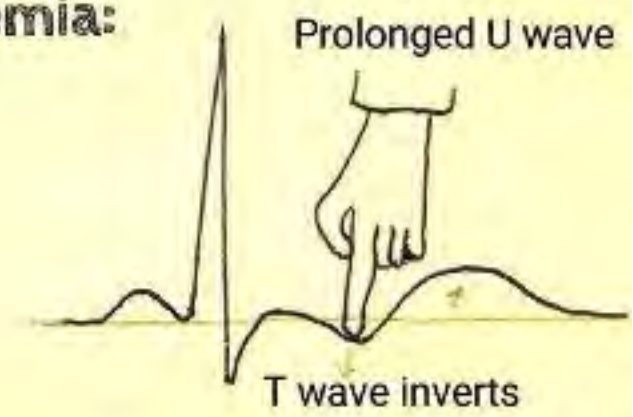
ECG

- Prolongation of QT interval
- ST-segment depression
- T-wave flattening
- Appearance of U waves
- Ventricular arrhythmias
- Atrial arrhythmias

Hyperkalemia:



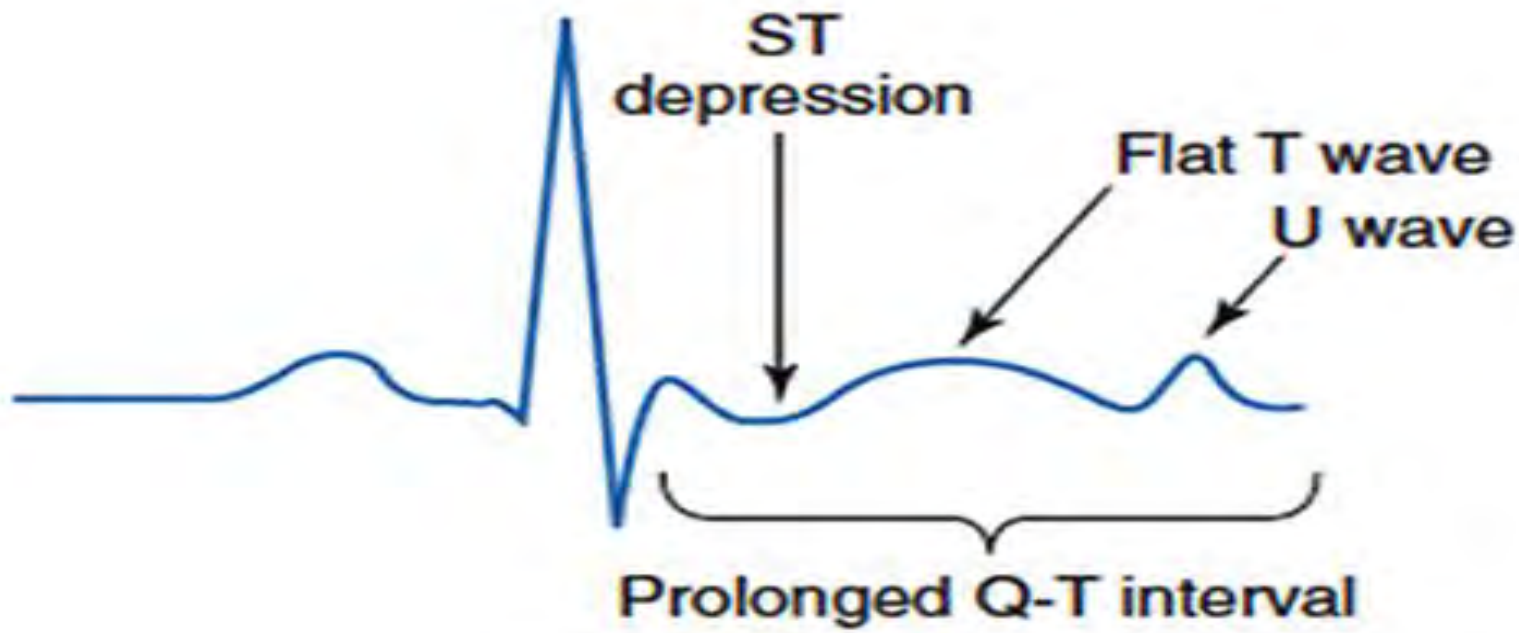
Hypokalemia:



" Pull and Push effects"

of Potassium on T wave of ECG


We are often the cause for
the Prolonged QTc!


ST
depression
Flat T wave
U wave
Prolonged Q-T interval

Hypokalemia

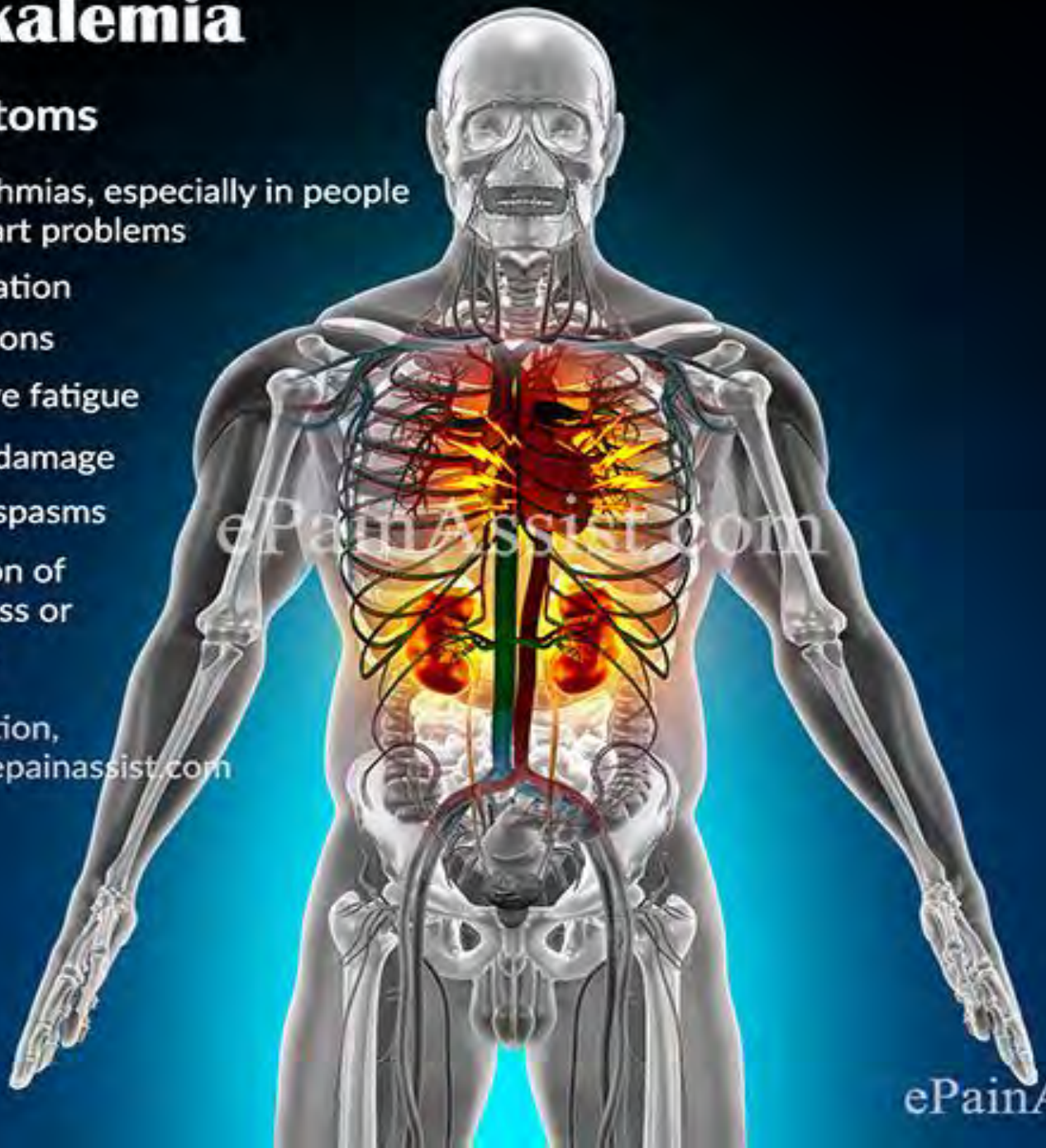
- **Moderate** **2.5 - 3 mEq/L.**
- **Severe** **< 2.5 mEq/L.**

Hypokalemia

Symptoms

- Dysrhythmias, especially in people with heart problems
- Constipation
- Palpitations
- Excessive fatigue
- Muscle damage
- Muscle spasms
- Sensation of numbness or tingling

For Information,
Visit: www.epainassist.com



Signs and symptoms

- Asymptomatic,
- Nonspecific ,
- Underlying cause rather than the hypokalemia itself.

Complaints

- ✓ Polyuria
- ✓ Palpitations
- ✓ Weakness and fatigue
- ✓ Muscle cramps and pain

Physical Findings

- **Decreased muscle strength**
- **Decreased tendon reflexes**

Physical Findings

- **Hypoventilation,**
- **Respiratory distress**
- **Respiratory failure**

Physical Findings

- **HTN**
- **Hypotension**
- **Arrhythmias**

Physical Findings

- **Signs of ileus**

Physical Findings

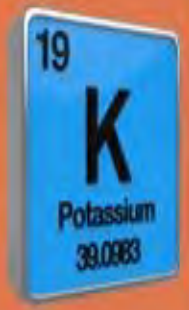
- **Growth failure**
- **Cushingoid appearance**

Hypokalemic Periodic Paralysis

XpertDox
Clinical, Education, Research

Rare disorder characterized by paralysis or muscle weakness

Characterized by fall in serum potassium levels



Affects 1 in 100,000 people

Caused by genetic mutation of potassium channels

Onset of symptoms in adolescence

Symptoms are inability to move muscles or limbs & weakness

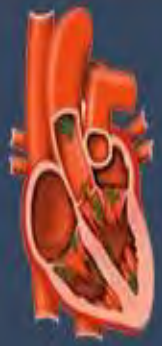
50% patients report recurrent migraine attacks

Symptoms precipitated by exercise, certain food or inactivity

Diagnosed by long exercise test & absent tendon reflexes

Treated by diet, lifestyle modification & potassium supplements

Complications are permanent muscle damage & heart abnormalities



Hypokalemic periodic paralysis

- AD
- Episodic
- Flaccid, generalized weakness
- Hypokalemia during the attacks.

Thyrotoxic periodic paralysis

- Hyperthyroidism.
- Asian males.
- Increased Na-K-ATPase activity.

Hypokalemia can only occur for 4 reasons:

1. Losses
2. Shift into cells
3. Decreased intake
4. Medication effects

Causes:
Diminished intake

- Malnutrition
- Anorexia nervosa
- Parenteral nutrition
- Chronic clay ingestion.

Drugs

- Diuretics,
- Steroids
- Bicarbonate
- Beta-agonist, Theophylline,

Drugs

- Amphotericin B,
- Ampicillin, high-dose penicillins
- Aminoglycosides (Gentamicin),

History

The patient's medications should be reviewed to ascertain whether any of them could cause hypokalemia.

A shift of potassium to the intracellular space

- Alkalosis (metabolic or respiratory)
- Insulin or glucose administration
- Intensive beta-adrenergic stimulation
  - Stress
  - Beta agonists- e.g.: albuterol.

Causes

The most common cause of hypokalemia is extra-renal loss in acute and chronic GIT diseases.

Causes

- **GI losses**
  - Vomiting ???
  - Diarrhea
  - Nasogastric suctioning
  - Enemas or laxative use

Causes

- **Renal losses**
  - Renal tubular acidosis
  - Bartter and Gitelman syndromes
  - Liddle syndrome
  - Hyperaldosteronism
  - Magnesium depletion

Genetic disorders

- Bartter syndrome
- Gitelman syndrome
- Liddle syndrome

Bartter syndrome

Characterized by urinary wasting of:

- **K**
- **Na**
- **Cl**
- **Ca**

Gitelman syndrome

Hypocalciuric, Hypomagnesemic variant of Bartter syndrome

Liddle s syndrome

- AD inheritance
- Early onset hypertension
- Hypokalemia
- "Pseudoaldosteronism"- Low levels of renin and aldosterone inspite of hypertension and hypokelemia.
- **Gain of function mutations** in the ALPHA subunits of the ditsal nephron epithelial sodium channels.(ENac)

Renal losses of potassium

1. Primary or 2ry aldosteronism

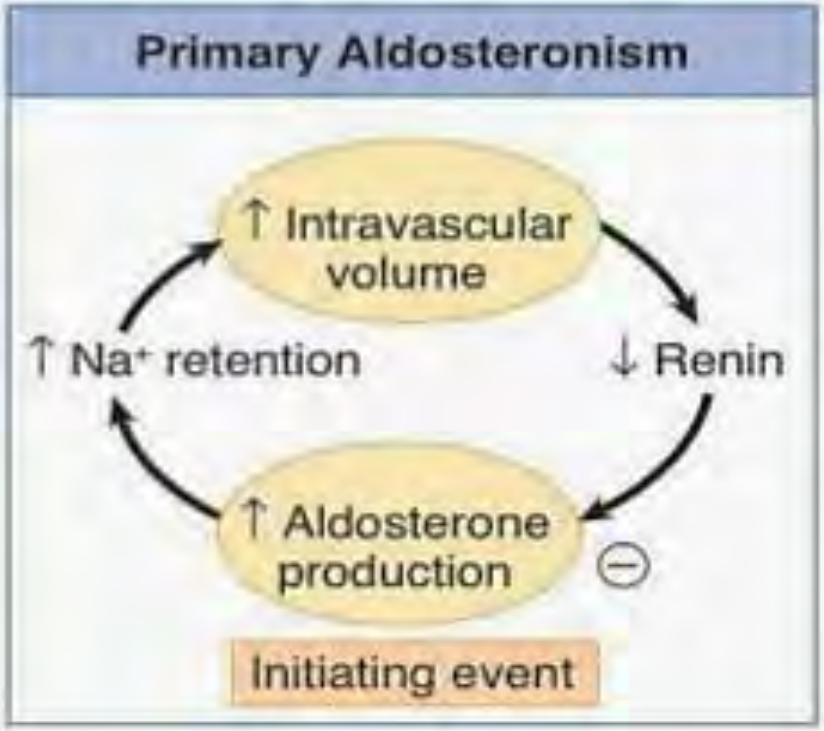
2. Exogenous mineralocortcoid.

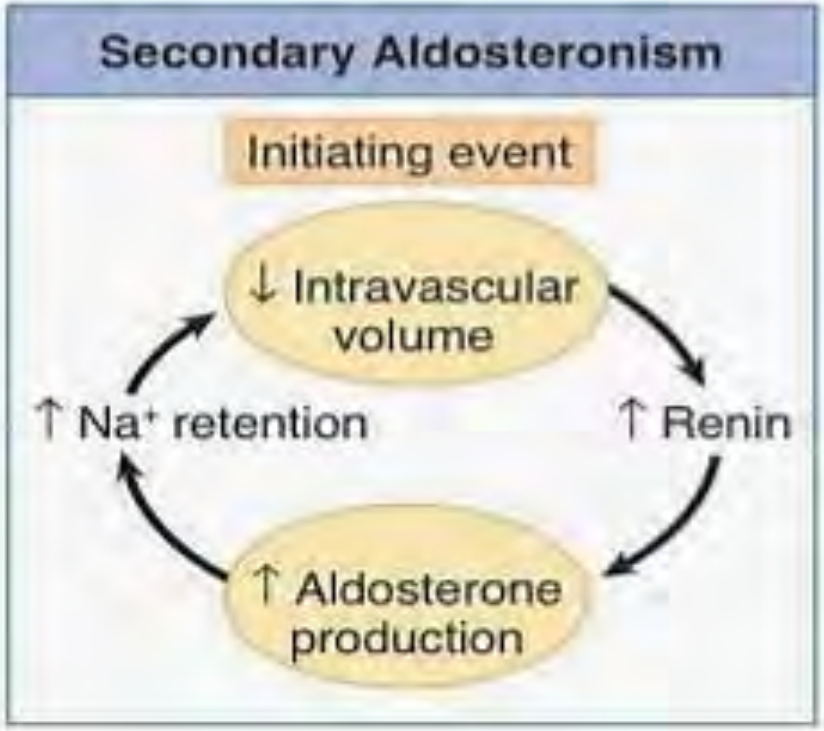
3. Renin-angiotensin-aldosterone cascade activation (Diuretics).

4. Renal tubular defects (RTA)
   - Proximal, especially with therapy
   - Some distal types

5. Presentation of a non-resorbable anion distally, obligating a cation, leading to increased K excretion in the presence of aldosterone.
   - Bicarbonate
   - Penicillin derivatives

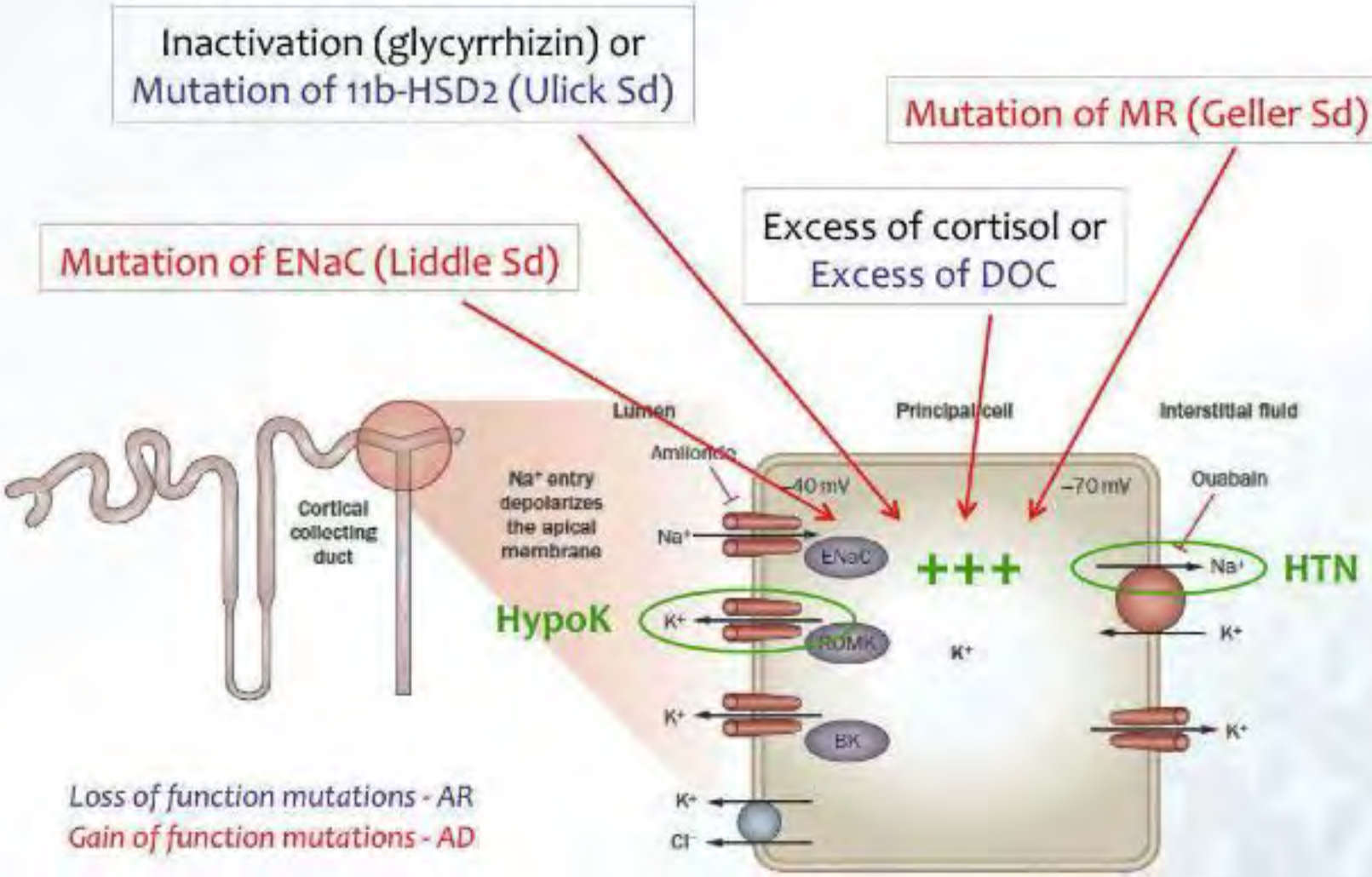
Hyperaldosteronism

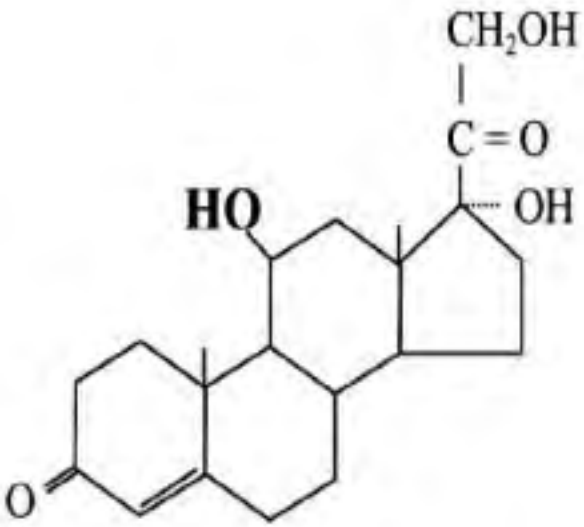




	Renin	Aldo	Ddx
Primary	↓	↑	adrenal adenoma/carcinoma, adrenal hyperplasia syndromes
Secondary	↑	↑	RAS, low effective circulating volume
Mimics	↓	↓	AME, licorice ingestion, Liddle's syndrome

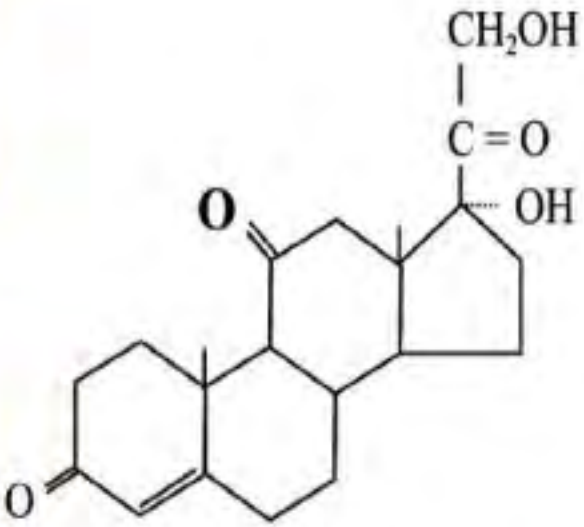
Pseudohyperaldosteronisms





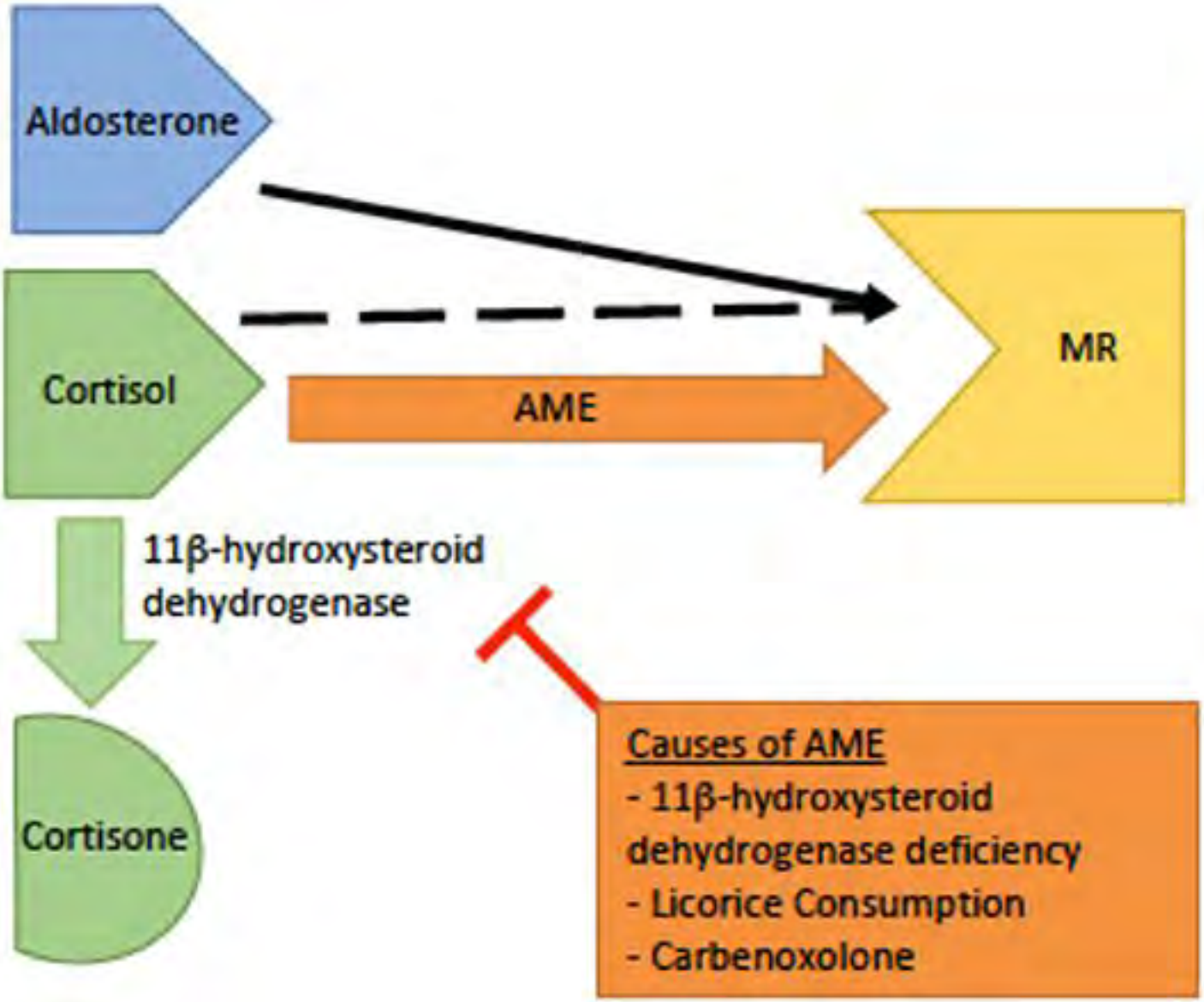
11 β-HSD2
11β-dehydrogenase

11 β-HSD1
11-ketoreductase



active steroids
- cortisol
- corticosterone
- prednisolone

inert steroids
- cortisone
- 11-dehydrocorticosterone
- prednisone


Aldosterone
Cortisol
MR
AME
11β-hydroxysteroid dehydrogenase
Cortisone
Causes of AME
- 11β-hydroxysteroid dehydrogenase deficiency
- Licorice Consumption
- Carbenoxolone

Apparent Mineralocorticoid Excess (AME)

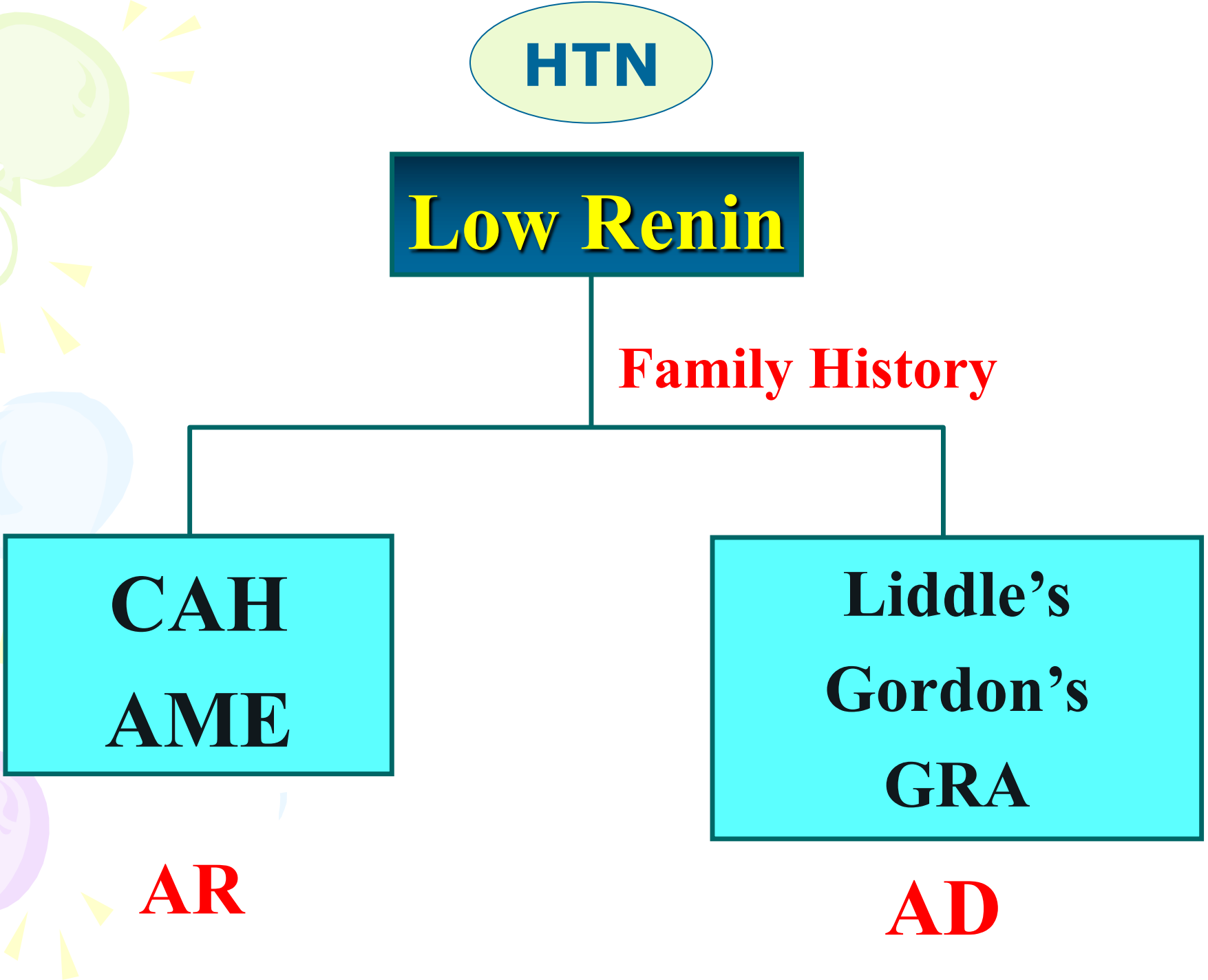
- AR form of monogenic HT
- Results from inactivation of 11-beta-hydroxysteroid dehydrogenase type2 (11B-HSD2)
- Causing reduced metabolism of cortisol to cortisone…local cortisol excess and MN response
- Metabolic clearance of cortisol is prolonged in AME
- Excess urinary excretion of the reduced metabolites of cortisol… increased Tetrahydrocortisol:tetrahydrocortisone Ratio

First-line Approach

- History
- History
- History

First-line Approach

- Drug History
- Family History


HTN
Low Renin
Family History
CAH
AME
Liddle's
Gordon's
GRA
AR
AD

First-line Approach

- Physical examination.
  1. HTN
  2. Getalia

Hypokalemia & HTN

- CAH
- Cushing syndrome
- Liddle syndrome, Licorice,
- Renovascular disease
- Renin secreating tumors
- Adrenal adenoma or hyperplasia

First-line Approach

- History
- Physical examination.
- Urine K (Spot U_K)

Spot U_K

A random U_K > 20

in presence of hypokalemia indicates renal K wasting

Spot urine potassium & History

Low urine potassium

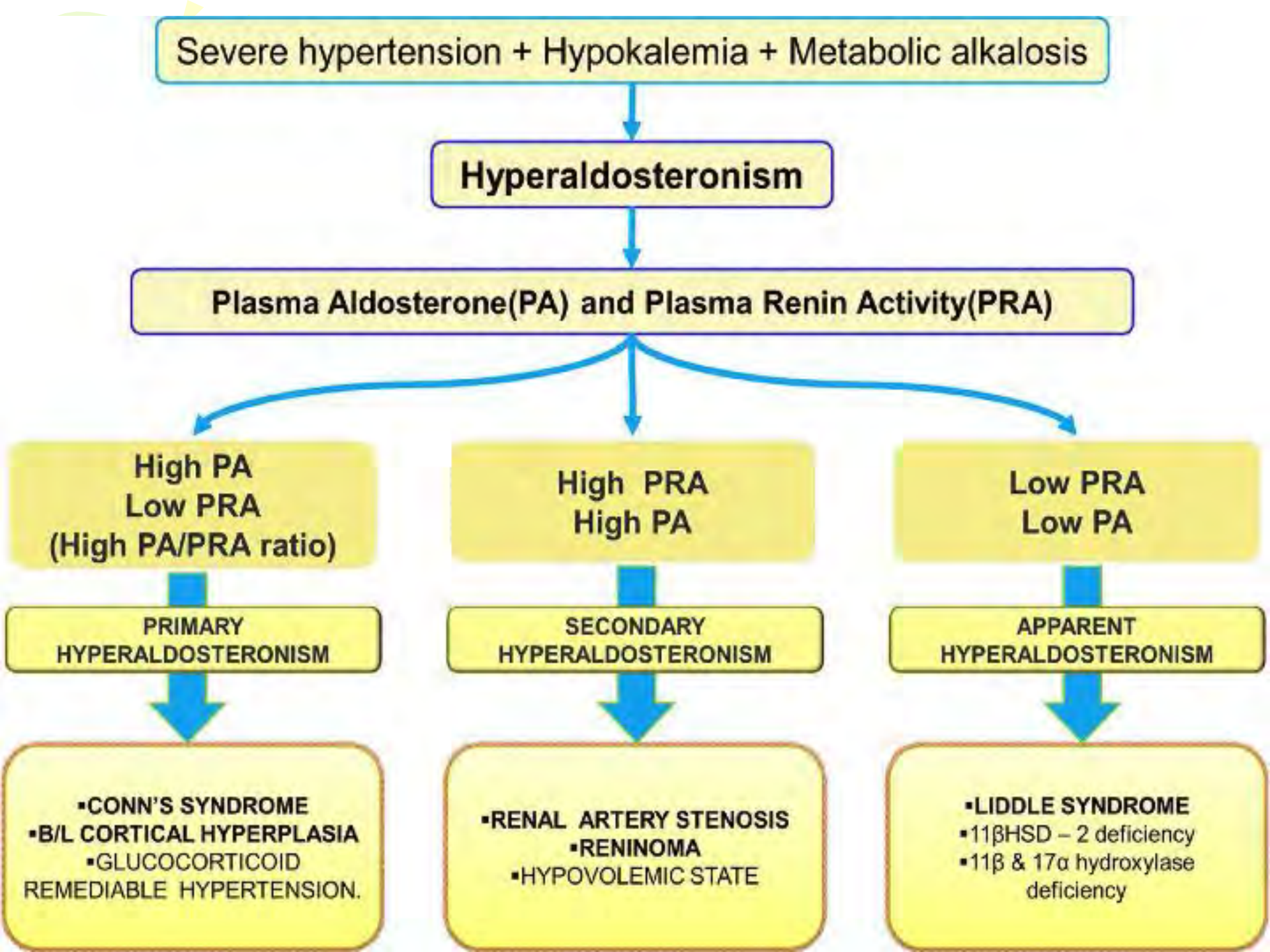
- GIL loss: (Diarrhea , laxatives ?)
- Poor intake (Diet ,TPN ?)
- Intracellular shift (Drugs, episodic weakness ?)

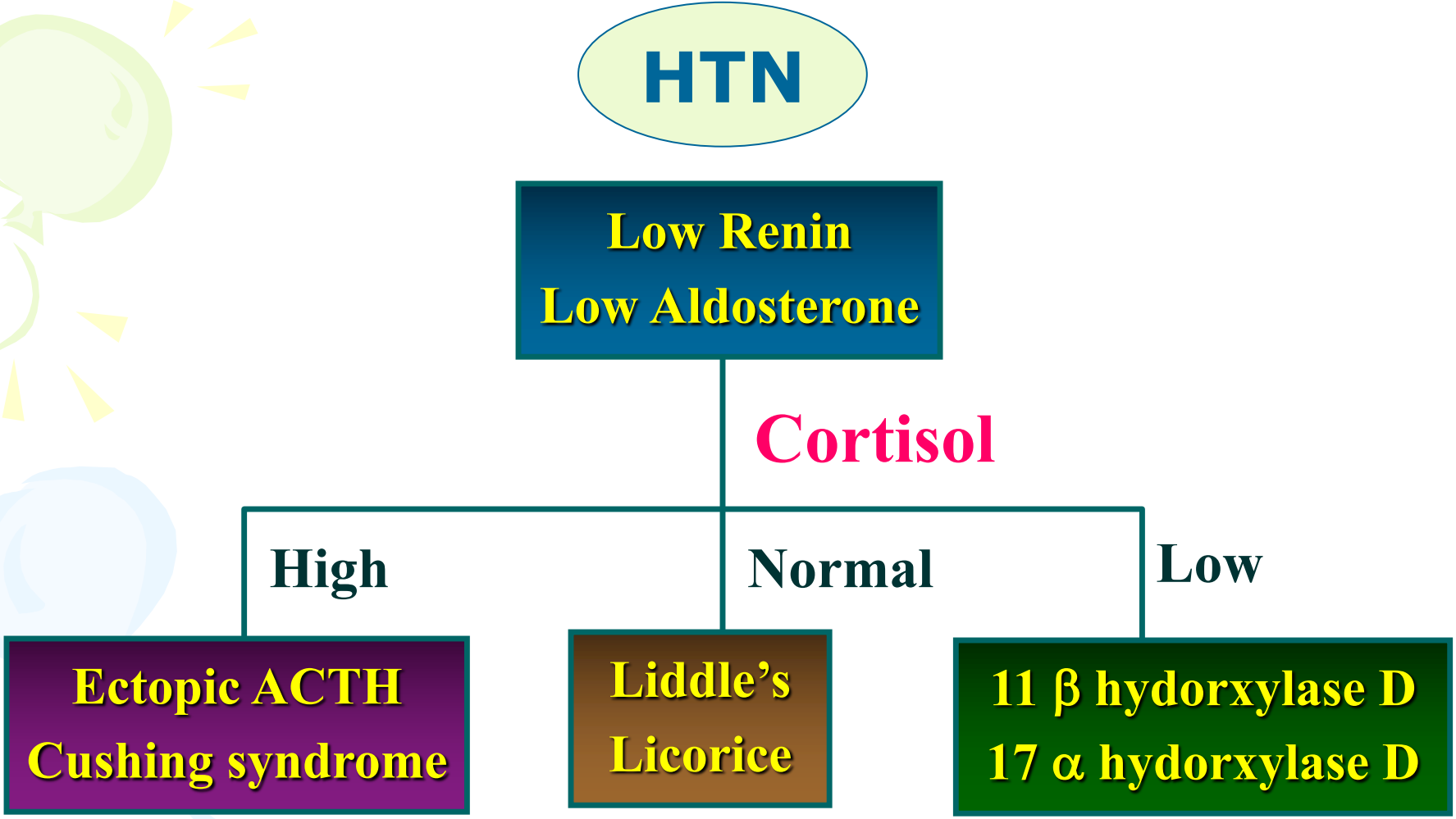
High urine potassium

- Renal loss (Diuretics ?).

Second-line Approach

- PRA, PAC & cortisol
- ABG
- Spot U_{cl}


Severe hypertension + Hypokalemia + Metabolic alkalosis
Hyperaldosteronism
Plasma Aldosterone(PA) and Plasma Renin Activity(PRA)
High PA
Low PRA
(High PA/PRA ratio)
High PRA
High PA
Low PRA
Low PA
PRIMARY HYPERALDOSTERONISM
SECONDARY HYPERALDOSTERONISM
APPARENT HYPERALDOSTERONISM
▪CONN'S SYNDROME
▪B/L CORTICAL HYPERPLASIA
▪GLUCOCORTICOID REMEDIABLE HYPERTENSION.
▪RENAL ARTERY STENOSIS
▪RENINOMA
▪HYPOVOLEMIC STATE
▪LIDDLE SYNDROME
▪11βHSD – 2 deficiency
▪11β & 17α hydroxylase deficiency



Lin SH, et al. Am J Med Sci 2003; 325: 153-156.

Liddle syndrome

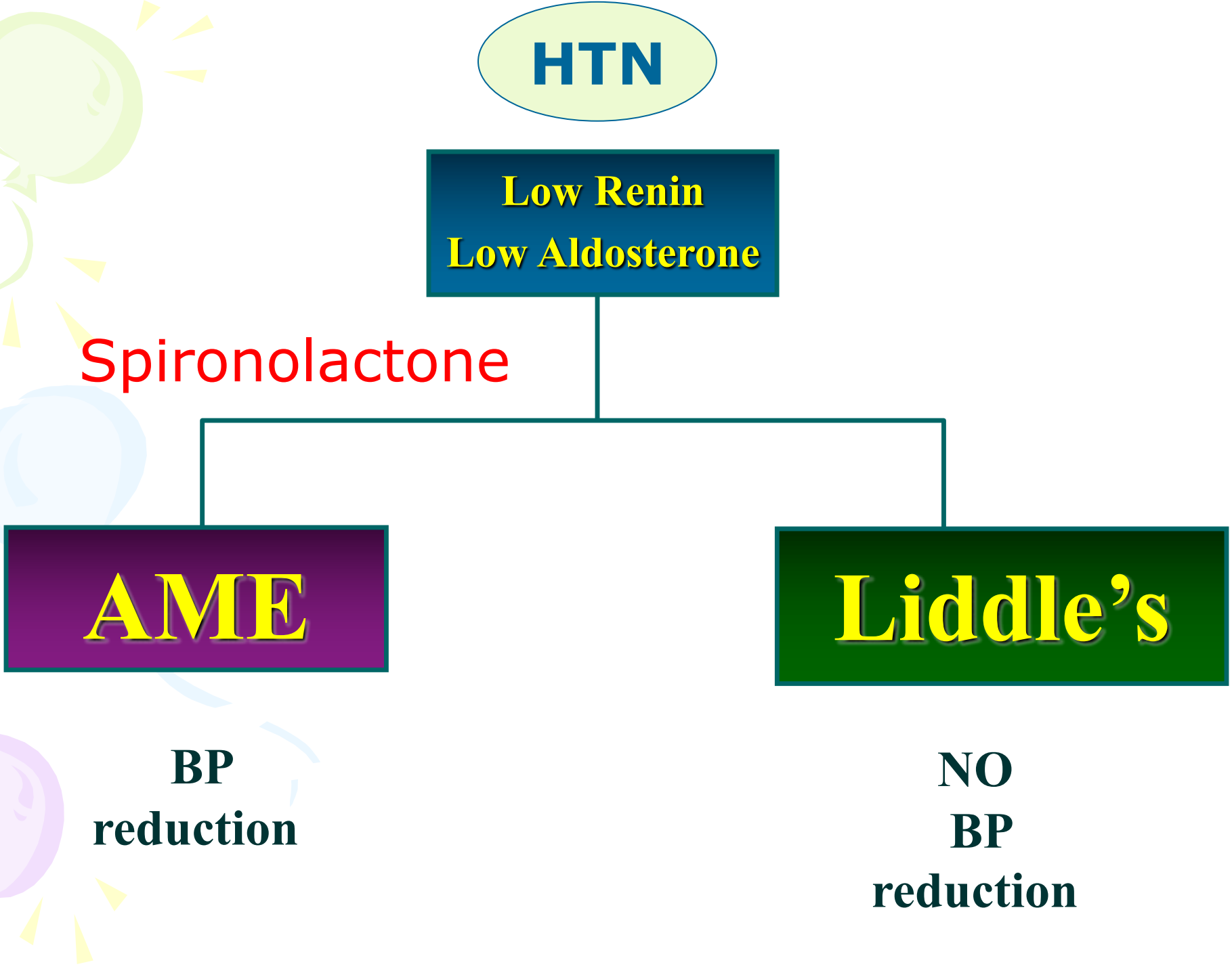
Amiloride & Triamterene

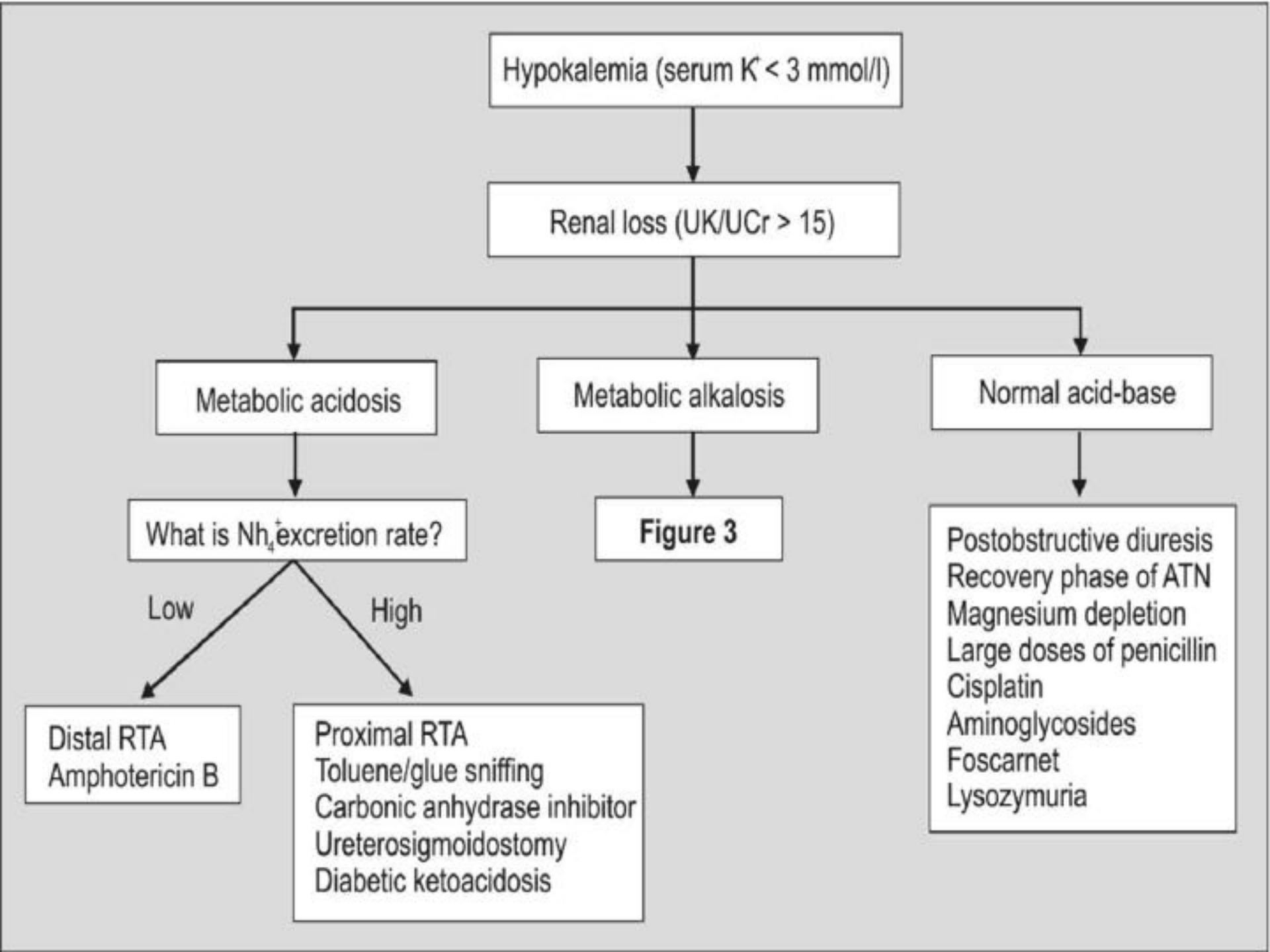
are

effective treatments

but

Spironolactone is not.


HTN
Low Renin
Low Aldosterone
Spironolactone
AME
Liddle's
BP
reduction
NO
BP
reduction


Hypokalemia (serum K+ < 3 mmol/l)
Renal loss (UK/UCr > 15)
Metabolic acidosis
Metabolic alkalosis
Normal acid-base
What is NH_4^+ excretion rate?
Figure 3
Low
High
Distal RTA
Amphotericin B
Proximal RTA
Toluene/glue sniffing
Carbonic anhydrase inhibitor
Ureterosigmoidostomy
Diabetic ketoacidosis
Postobstructive diuresis
Recovery phase of ATN
Magnesium depletion
Large doses of penicillin
Cisplatin
Aminoglycosides
Foscarnet
Lysozymuria

Third-line Steps Based on

High clinical index of suspicion for the disorder

Third-line Steps Based on

High clinical index of suspicion for the disorder

- Adrenal imaging (adenoma)
- Evaluation for renal artery stenosis
- Enzyme assays for 17-beta hydroxylase deficiency

Third-line Steps Based on

High clinical index of suspicion for the disorder

- Drug screen (diuretics, sympathomimetics)
- Thyroid function in Asians with tachycardia.
- Serum anion gap (toluene toxicity)

Third-line Steps Based on

High clinical index of suspicion for the disorder

- Pituitary imaging (Cushing syndrome)

Treatment

1. Treat the cause
2. Reduction of losses
3. Replenishment of stores
4. Evaluation for potential toxicities
5. Prevent future episodes, if possible

The magnesium level

K level cannot be corrected until hypomagnesemia has been corrected.

Decreasing Potassium Losses

- ✓ Treat diarrhea or vomiting
- ✓ Discontinue diuretics/laxatives
- ✓ K-sparing diuretics
- ✓ Control hyperglycemia
- ✓ H2 blockers to patients on NG suction

Replenishment of Potassium 2.5-3.5 mEq/L

- Need only oral K replacement therapy.
- If cardiac arrhythmias or significant symptoms are present, treat as severe hypokalemia.

Replenishment of Potassium <2.5 mEq/L

- Hospital admission;
- Continuous ECG monitoring,
- IV potassium should be given.
- Gradual replacement over a few hours.
- Replace Mg if low.
- Check serial K levels.

Pitfalls

- Take ongoing K losses into consideration.
- Avoid glucose-containing parenteral fluids.
- If the patient is acidotic, correct the K first.
- Evaluation for potential toxicities.
  - ✓Digitalis
  - ✓Overcorrection hyperkalemia

Severe Hypokalemia

- 0.5 mEq/kg/dose;
- 0.5 mEq/kg/hr;
- Max 10 mEq/hr;
- Max 40 mEq/dose;
- Max 200 mEq/day;

Severe Hypokalemia

- evaluate 1-2 hr after end of infusion;
- repeat as necessary based on lab values;
- ongoing losses may require >200% of normal daily maintenance


Thank you

MCQ

- **A 12 YEARS OLD BOY COMPLAINS OF FATIGABILITY AND WEAKNESS**

- **PHYSICAL EXAM:** BP 122/68, HR 72/MIN, NO ORTHOSTATIC CHANGES, NO EDEMA

- **LABS (mEg/L):** Na :135, K: 2.1, Cl: 85, Hco3:45, U_{Na}=80, U_K=70

MCQ1:

- **WHY IS HIS POTASSIUM LOW?**

A. VOMITING

B. DIURETIC USE

C. BARTTER/GITELMAN

D. None of the above

E. All of the above

MCQ2:

WHAT TEST(S) WILL HELP YOU MAKE THE DIAGNOSIS?

A. Spot Urine K
B. Spot Urine Cl
C. Spot Urine K/Cr
D. None of the above
E. Any of the above

MCQ3:

WHICH SALT YOU ARE GOING TO USE TO TREAT THIS PATIENT?

A. k acetate
B. K citrate
C. K chloride
D. None of the above
E. Any of the above