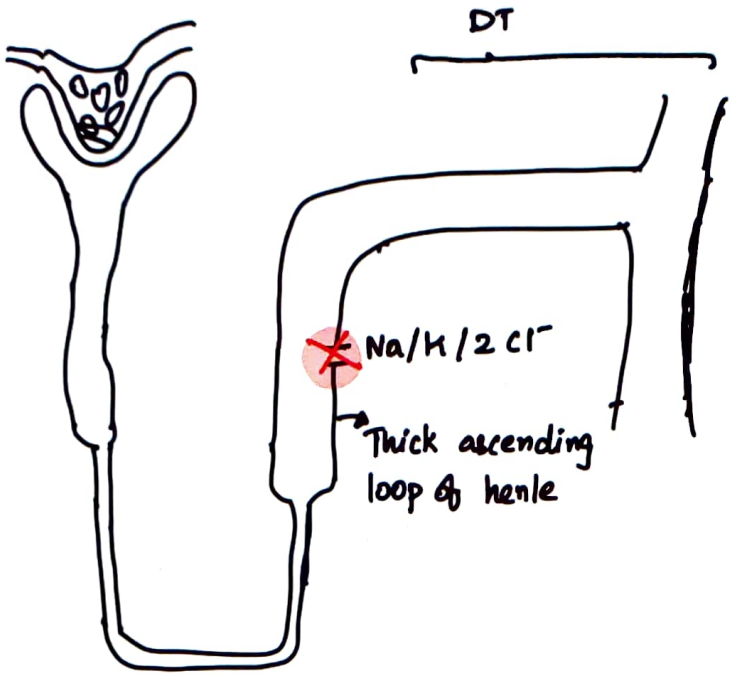
BARTTER'S SYNDROME

37:02



Clinical Findings :

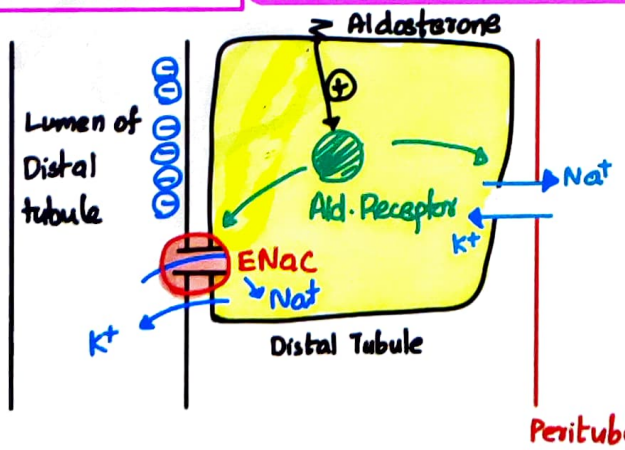
- Polyuria
- Rennin ↑se
- Aldosterone ↑se
- Hypokalemia
- Metabolic alkalosis
- $U_{Ca^{2+}}$ ↑se → Renal stones
- (N) B.P
- ↑se P.G

Rx :-

1) Hydration
2) Spironolactone
3) Indomethacine NSAIDs to block P.G
4) Correction of Electrolytes

LIDDLE'S SYNDROME — PSUEDOHYPERALDOSTERONISM

40:53



- In Liddle's syndrome ENaC channel is HYPERFUNCTIONS
  - ↓
  - Na^+ reabsorbed in excess
  - ↓
  - Loss of potassium in urine

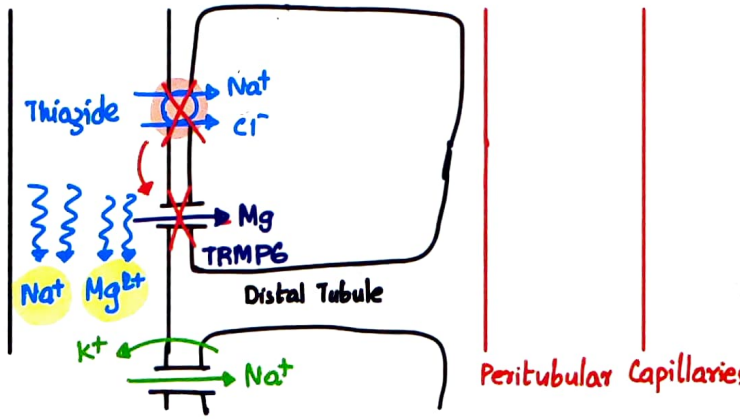
Consequences:

1) Excess Na^+ reabsorbtion → Hypertension
2) Rennin ↓se
3) Aldosterone ↑se
4) Hypokalemia
5) Metabolic Alkalosis
6) Urine Calcium decreases

GITTELMAN'S SYNDROME

44:25

- Defect occuring at a place where thiazide suppose to work.



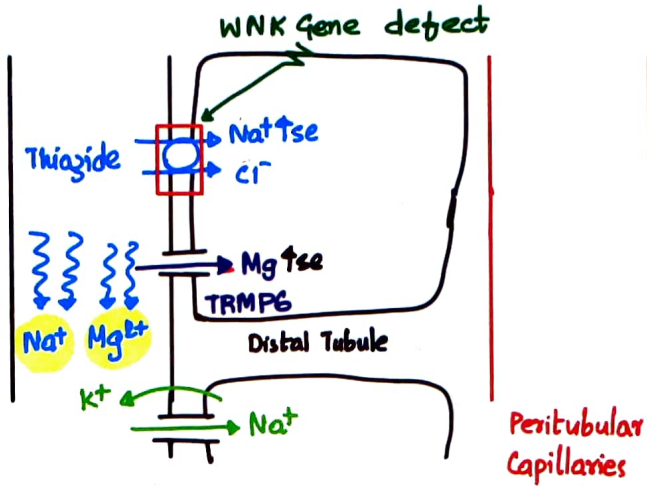
Consequences

- Polyuria
- (N) B.P
- Hypo kalemia
- Metabolic Alkalosis
- Urine Mg. ↑↑↑
- Urine Ca^{2+} ↓se → NO RENAL STONES

GORDENS SYNDROME

47:50

- Psuedohypoaldosteronism Type II



WNK Gene defect leads to hyperfunctioning of thiazide channel which leads to increased Na^+ reabsorbtion.

This also hyperfunctions TRMP6 channel and increased reabsorption of Mg^{2+}.

Consequences

- Hypertension
- Low urine magnesium
- Hyperkalemia
- Metabolic acidosis
- $U_{Ca^{2+}}$ ↑se → Renal Stones
- Rennin ↓se
- Aldosterone ↓se

POLYCYSTIC KIDNEY DISEASE

52:26

Autosomal dominant Polycystic kidney disease

- PKD1 gene - Chromosome 16 → Severe ↑, Early presentation
- PKD2 gene - Chromosome 4 → Less severe, Later presentation

Major manifestations

Renal	Extra Renal
• Flank Pain • Urinary Tract Infection • Renal stones • Hematuria • Hypertension • CKD → progressive → ESRD	• Hepatic cysts • Diverticulosis of gut • Cysts → Pancreas, spleen • Aneurysms

AR-PKD:

- Cyst present at birth
- a/w Caroli's disease in liver
- Hepatic fibrosis

active space

Acid , Base and Electrolytes

NORMAL VALUES

00:45

1) pH = 7.35 - 7.45
2) arterial CO_2 = 35 - 45
3) HCO_3^- = 22 - 28

METABOLIC → Acidosis → HCO_3^- ↓
→ Alkalosis → HCO_3^- ↑

RESPIRATORY → Acidosis → CO_2 ↑
→ Alkalosis → CO_2 ↓

COMPENSATION FORMULAS

01:30

1) METABOLIC ACIDOSIS

Winter's formula =

$$CO_2 = 1.5(HCO_3^-) + 8 \pm 2$$

2) METABOLIC ALKALOSIS

HCO_3^- ⟷ CO_2
10 ↑ 6 ↑

every 10 increase → CO_2 rise by 6.
of HCO_3^-

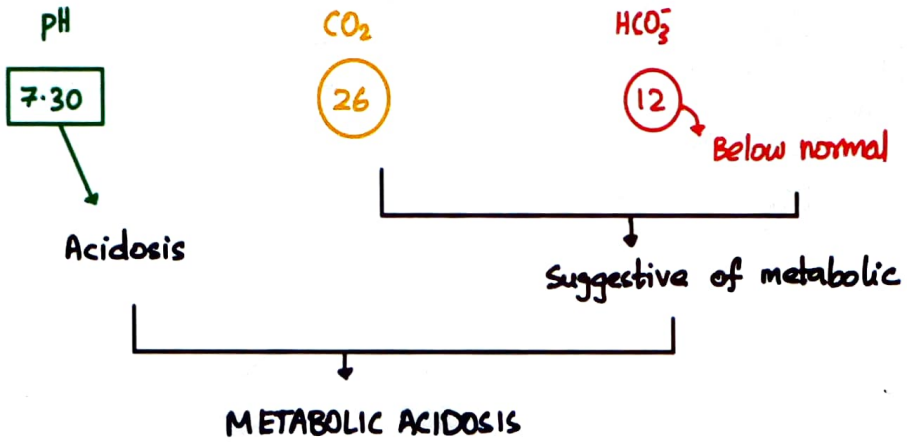
active space

RESPIRATORY

		HCO_3^-	CO_2
• Acidosis	Acute →	1 ↑se	10 ↑se
	Chronic →	4 ↑se	10 ↑se
• Alkalosis	Acute →	2 ↓se	10 ↓se
	Chronic →	4 ↓se	10 ↓se

STEPS IN SOLVING ACID- BASE DISORDERS

03:20



expected CO_2 = $\frac{3}{2}$ × 12 + 8 ± 2

= 24-28 } CO_2 level in the question lies in the range of expected CO_2 level

⇒ ACUTE METABOLIC ACIDOSIS & COMPENSATORY RESPIRATORY ALKALOSIS

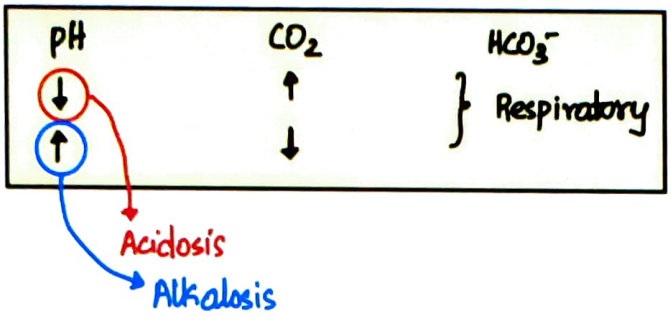
" " " ↑ RESPIRATORY ALKALOSIS

If in the question, if CO_2 value was (16)

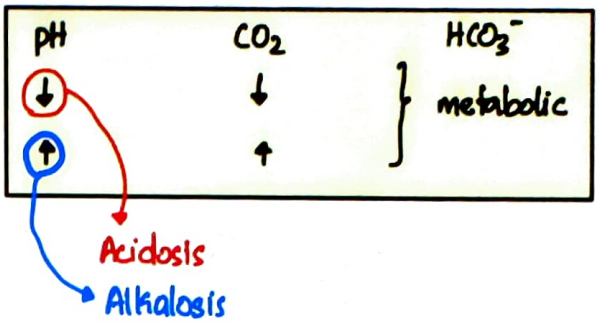
active space

Rome rule suggests :

in respiratory condition
pH and CO_2 move in opposite direction



in metabolic condition
pH and CO_2 move equidirection



pH	CO_2	HCO_3^-
↓	↑	↓
7.10	70	12
⇓	↓	↓
ACIDOSIS	Respiratory acidosis	Metabolic acidosis

} MIXED ACIDOSIS

active space

Na^+ Normal : 135 - 145 meq/L

HYPONATREMIA < 135 meq/L

Plasma Osmolarity = $2 Na^+ + \frac{Glu}{18} + \frac{BUN}{2.8}$

↑
- i) D.M
- ii) Mannitol

N
Psuedohyponatremia
- i) ↑ T_4
- ii) ↑ Proteins (Multiple myeloma)

↓
True hyponatremia

HYPOOSMOTIC HYPONATREMIA

ECV

HYPERVOLEMIA
- Congestive cardiac failure
- Cirrhosis
- Nephropathy

EUVOLEMIC
- SIADH
- Hypothyroidism

HYPOVOLEMIC

Urine Na^+ : >20
- i) Diuretics
- ii) Acute Tubular necrosis
- iii) AIN

<20
- i) G.I
  - Diarrhoea
  - Vomiting
  - Fistulas
- ii) Skin
  - Burns
  - Excessive sweating

CORRECTION OF HYPONATREMIA

18:00

- Acute → grossly symptomatic → Rapid correction
- Chronic → Mild / Asymptomatic → Slow correction
  < 6-8 mEq/24 hrs
  - upper limit is 10 mEq/24 hrs
  - >10 mEq/24hr → CENTRAL PONTINE DEMYELINOLYSIS
    CPM : osmotic demyelination

CORRECTION OF HYPERNATREMIA

20:34

$$\text{Water Deficit} = \text{T.B.W} \times \left[\frac{Na^+ - 140}{140}\right]$$

① TBW → male = 0.6 × weight
female = 0.5 × weight

② Ongoing water loss

$$\text{Free water clearance} = V \times \left[1 - \frac{U_{Na^+} + U_{K^+}}{P_{Na^+}}\right]$$

AIIMS '19 ||

③ Insensible loss :

≈ 10ml / kg

eg: 50kg person

insensible loss = 50 × 10

= 500ml

via → . Breathing
. Cuts

① + ② + ③ = Amount of water to be given

active space

CORRECTION OF HYPONATREMIA

22:00

Hyponatremia → Provide (N) saline

↓

- Urine output improves
- B.P starts increasing

↓

- 5% Dextrose
- $N/_2$ saline

POTTASIUM

24:30

- (N) $s.K^+ = 3.5 - 5.5$ mEq/L
- > 5.5 mEq/L → Hyperkalemia

MANAGEMENT OF HYPERKALEMIA :

- Regardless of ECG changes → Patient given Ca^{2+}

Ca^{2+} → Ca Gluconate
Ca^{2+} → $CaCl_2$

- Insulin + glucose
- β_2 Agonist
- Diuretics
- K^+ Binders → i) PATIROMER
  ii) ZIRCONIUM

DO NOT ANSWER C NOT ALLOWED)

- Ca^{2+} Carbonate
- 50%. Dextrose without insulin
- Sodium Polysterene / Kayexelate ⇒ causes intestinal necrosis

MANAGEMENT OF HYPOKALEMIA :-

- Calculate K^+ deficit → $(3.5 - K^+) \times$ weight $\times$ 0.4

① Oral KCl → best method

② I.V correction [if patient cannot take oral]

i) Peripheral I.V line → DO NOT USE >10mEq/hr

ii) Central I.V line → >20mEq/hr

If these limit are met

Use MANNITOL : Increase K^+ by pulling potassium out of the cells
{Hypotonic fluid}

Hypomagnesemia → rare
↳ should be correct
unless corrected Ca^{2+} will not be corrected

active space