FLUID, ELECTROLYTES, AND ACID–BASE DISORDERS

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DEFINITION OF BODY FLUID COMPARTMENTS

- water constitutes 50% to 70% of lean body weight (decrease with age), 70-80% in infants.
- total-body water is divided into an intracellular fluid compartment and an extracellular fluid compartment, which consists of an intravascular compartment and an interstitial compartment
- Extracellular fluid :

Principle cation : NA+ / anions : CL− AND HCO3 −.

- Intracellular fluid :

Principle cation : K+ and MG2+ / anions : phosphates and (-)

charge proteins.

- knowledge of fluid compartments and their composition becomes

important when considering fluid replacement

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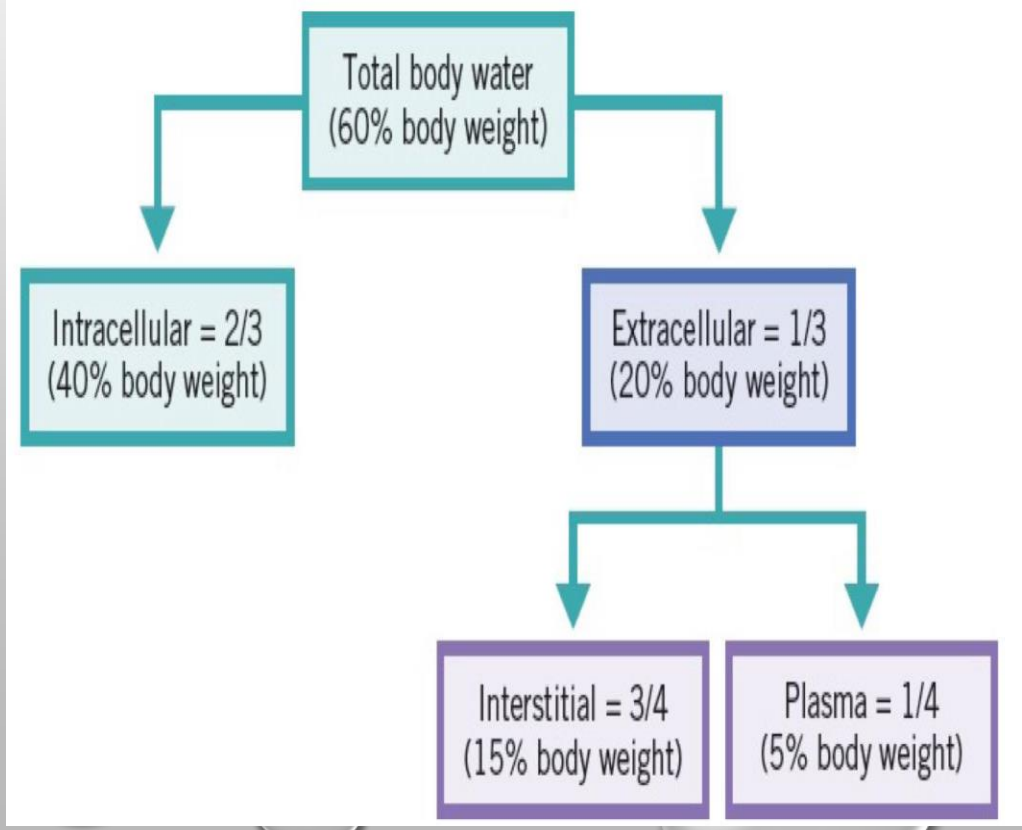


DISTRIBUTION OF INTRAVENOUS FLUID

We have 2 types of Fluid therapy :

1-CRYSTALLOIDS are solutions that contain sodium as the major particles ,OR aqueous solution of mineral salts or water-soluble molecule , inexpensive and used for volume expansion, maintenance infusion, and correction of electrolyte disturbances.

Example :

- ISOTONIC CRYSTALLOIDS (E.G., LACTATED RINGER'S SOLUTION, 0.9% NACL) distribute uniformly throughout the extracellular fluid compartment so that after 1 hour, only 25% of the total volume infused remains in the intravascular space.
- HYPERTONIC SALINE SOLUTIONS
- HYPOTONIC SOLUTIONS (D5W, 0.45% NACL)

2-COLLOID SOLUTIONS: contain high–molecular-weight substances that remain in the intravascular space because its molecular size is large and cannot cross capillary membrane , they increase intravascular space through pull in water via oncotic pressure.

EXAMPLES OF COLLOID SOLUTIONS: ALBUMIN / DEXTRAN / HYDROXYETHYL STARCH (HETASTARCH)

Intravenous isotonic saline (0.9% NACL) is distributed only in the extracellular (ECF) compartment-about 75% in the interstitial compartment and 25% in the plasma. none enters the intracellular fluid (ICF) compartment

Now we will talk Briefly about crystalloids :

❖ **Isotonic fluid :**

1) Normal saline 0.9% :

- Replaces water, sodium, chloride / the only solution we administer with blood.
- Indicated in Intravascular resuscitation and replacement of salt loss e.g. diarrhoea and vomiting.
- When we given in large volume can produce Hyperchloremic metabolic acidocsis , also be aware of fluid overload in case of kidney and heart failure.

2) LR (lactated Ringer's) :

- Lactated Ringer's has water, sodium chloride, sodium lactate, potassium, and calcium.
- Because Normal saline has a higher risk of metabolic acidosis than lactated Ringer's, which is better able to reduce acidity thanks to its lactate content.
- Not for pts with liver disease or lactic acidosis
- Monitor for hyperkalaemia in renal patient.

❖ **Hypertonic fluid**

- Higher osmolarity than the blood , so osmosis will cause water to leave intracellular space (cell shrinks) and expand extracellular space .
- Indicated for treatment of severe hyponatremia , also for brain swelling (cerebral edema)
- Be aware that they could overload extracellular space leading to pulmonary edema , and could cause hypernatremia

❖ **Hypotonic solution :**

- Lower osmolarity than the blood so osmosis causes water to move from extracelluar space to intracellular space (cell swells).
- It helps in dilute extracellular space , so it helps in case of hypernatremia
- Be aware that these can cause brain swelling
- 5% Dextrose is one example of that (D5W)

Now we will talk about colloid product :

Natural – Albumin, FFP Artificial – Gelatin and Dextran and HES

❖ **Albumin :**

- There are 2 solution : 5% leads to 80% initial volume expansion / 25% leads to 200-400% increase in volume expansion
- used mainly for emergency treatment of shock especially due to loss of plasma (acute management of burns) and is given with crystalloids , also in hypoalbuminemia.
- One of the side effect is pruritis and anaphylactic reactions and coagulation abnormalities.
- Disadvantages : cost-effectiveness / volume overload

❖ **Dextran :**

- is a synthetic glucose polymer that expands the intravascular volume by an amount equal to the volume infused
- Side effects include renal failure, osmotic diuresis, bleeding disorder , and laboratory abnormalities (i.e., elevations in blood glucose and protein and interference with blood cross-matching).
- There is no clear benefit to the use of dextran over crystalloid solutions.

❖ **HES :**

- Hetastarch, like 5% albumin, increases the intravascular volume by an amount equal to or greater than the volume infused.

- however, found that fluid resuscitation with hydroxyethyl starch is associated with an increased incidence of mortality, renal failure, and increased use of renal replacement therapy , so now isn't recommended .

COMPARING CRYSTALLOID V/S COLLOID

CRYSTALLOIDS	COLLOIDS
Aqueous solutions of low molecular weight ions with or without glucose	High molecular weight substances, similar to plasma proteins
Readily pass through semi-permeable membrane ("Extra vascular space expanders")	Molecular size is large and do not cross capillary membrane ("Intravascular space expanders").
Intravascular t1/2 = 20-30 minutes	Intravascular t1/2= 2-8 hours
Reduce plasma colloid osmotic pressure	Maintain plasma colloid osmotic pressure
Have poor capillary perfusion	Have good capillary perfusion
Risk of over hydration/tissue edema is obvious	It is insignificant
No anaphylactic reaction	Risk of anaphylaxis is more
Inexpensive	Expensive
Readily available, easy to store and well tolerated by patients – some advantages	Not so
Indications: • Rx of dehydration of any cause, • Hypoglycemia (5% 10% D) • Hypochloremia, hyponatremia of any cause Preloading fluid in regional block(SA) • Intraoperative/postoperative maintenance fluid	Indications: • Fluid resuscitation prior to arrival of blood • Severe hypoglobuminemia • Burns • Fluid boluses in critically ill patient where crystalloid use would be excessive.

COMPOSITION OF COMMON PARENTERAL FLUIDS

Solution	Volume[b]	Na^+	K^+	Ca^{2+}	Mg^{2+}	Cl^-	HCO_3 (as Lactate)	Dextrose (g/L)	mOsm/L
Extracellular fluid	—	142	4	5	3	103	27	—	280–310
Lactated Ringer	—	130	4	3	—	109	28	—	273
0.9% NaCl	—	154	—	—	—	154	—	—	308
0.45% NaCl	—	77	—	—	—	77	—	—	154
D5W	—	—	—	—	—	—	—	50	252
D5/0.45% NaCl	—	77	—	—	—	77	—	50	406
D5LR	—	130	4	3	—	109	28	50	525
3% NaCl	—	513	—	—	—	513	—	—	1,026
7.5% NaCl	—	1,283	—	—	—	1,283	—	—	2,567
6% hetastarch	500	154	—	—	—	154	—	—	310
10% dextran-40	500	0/154[c]	—	—	—	0/154[c]	—	—	300
6% dextran-70	500	0/154[c]	—	—	—	0/154[c]	—	—	300
5% albumin	250, 500	130–160	<2.5	—	—	130–160	—	—	330
25% albumin	20, 50, 100	130–160	<2.5	—	—	130–160	—	—	330
Plasma protein fraction	250, 500	145				145			300

COMMON ELECTROLYTE DISORDERS

❖ **SODIUM** (NORMAL CONCENTRATION 135-145):

1-HYPONATREMIA: if infused or ingested water exceeds renal free water excretion capacity, plasma sodium will decrease rapidly. (below 135)

a-clinical manifestations:

- neurologic symptoms result from hypoosmolality a decrease in posm (plasma osmolality) causes intracellular water influx, and cerebral edema / symptoms include lethargy, confusion, nausea, vomiting, seizures, and coma.
- There is acute hyponatremia (below 120-130), chronic hyponatremia (below 110-120)
- 3 types : hypertonic hyponatremia / isotonic hyponatremia / hypotonic hyponatremia (hypovolemic/hypervolemic) as shown in the next slide.
- Treatment :

o isotonic and hypertonic hyponatremia correct with resolution of the underlying disorder

o hypovolemic hyponatremia can be managed with administration of 0.9% NACL to correct volume deficits and replace ongoing losses.

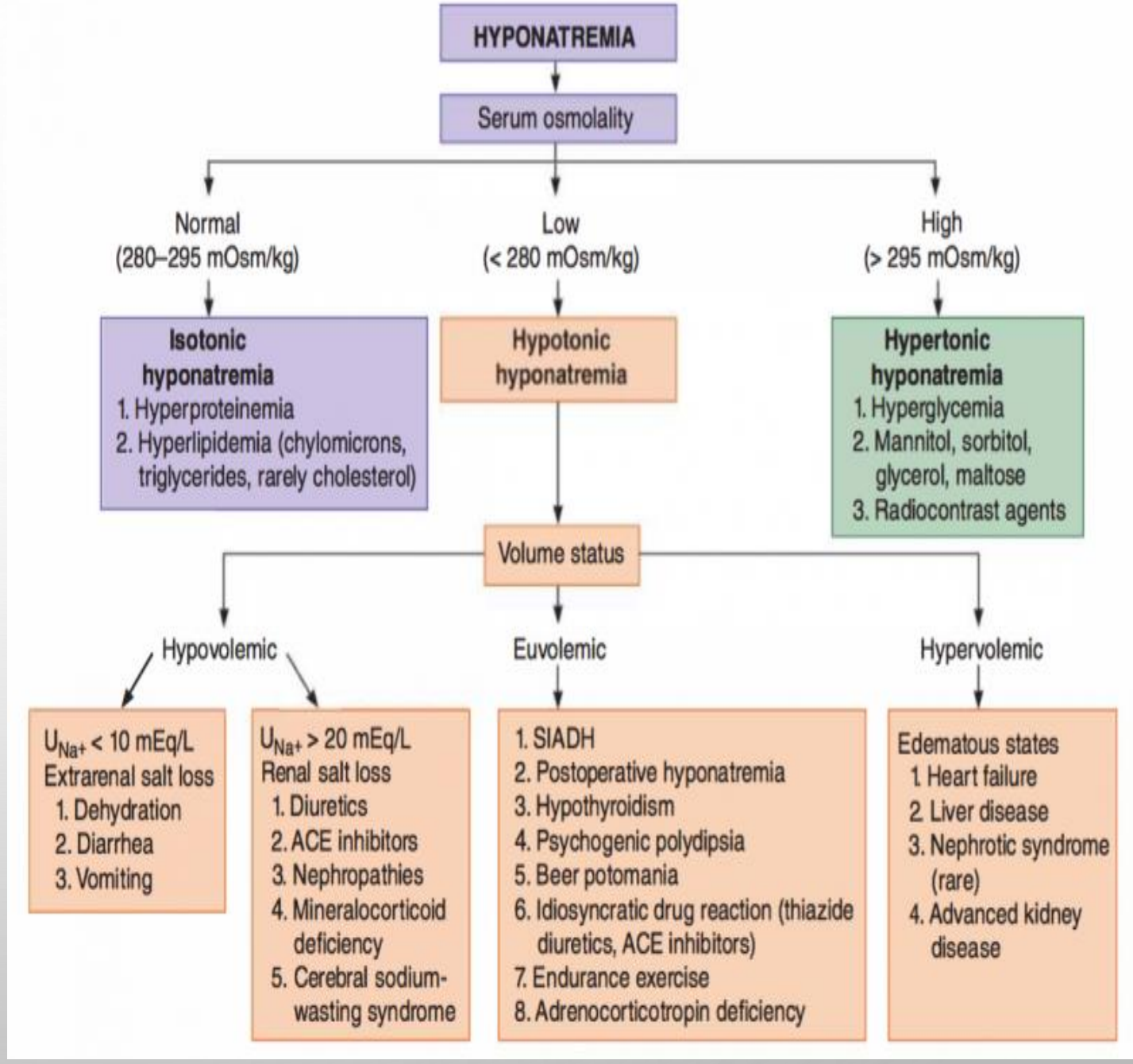
o hypervolemic hyponatremia: water restriction (1,000 ml/day) to return NA+ to greater than 130 mmol/l

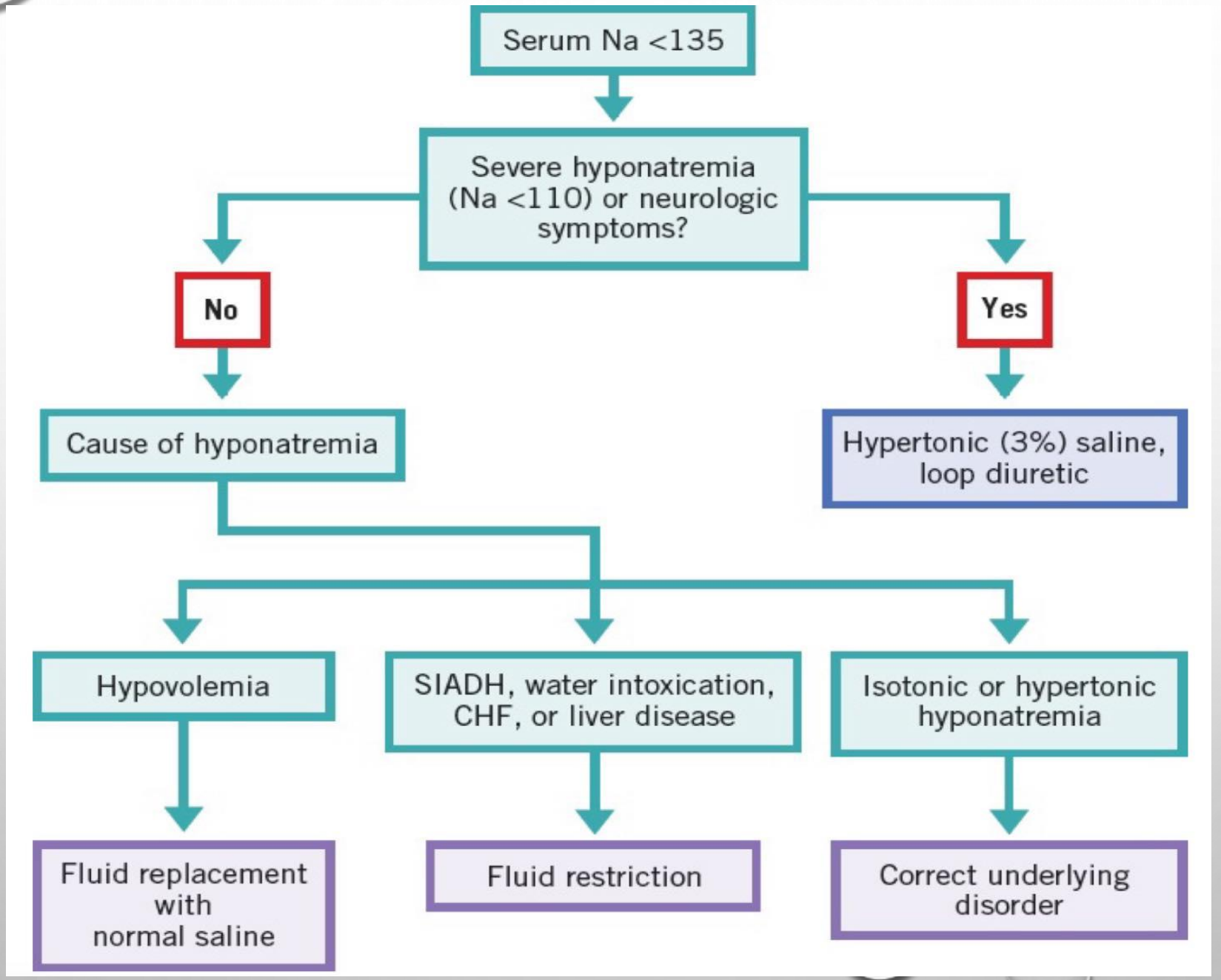
loop diuretic with 3% NACL

❖ False hyponatremia or pseudohyponatremia (isotonic hyponatremia) : it is a low serum sodium concentration associated with normal extracellular osmolality so there is no move of water into the cell and seen in hyperlipidaemia and hyperproteinaemia as in case of multiple myeloma.

❖ True hyponatremia (hypotonic hyponatremia): It is low serum sodium concentration and associated with reduction in extracellular osmolality and is further classified into hypovolemic , euvolemic, hypervolemic depend on extracellular volume fluid status.

❖ hypertonic hyponatremia is due to presence of osmotically active solutes in the serum e.g., mannitol or glucose




Serum Na <135
Severe hyponatremia (Na <110) or neurologic symptoms?
No
Yes
Cause of hyponatremia
Hypertonic (3%) saline, loop diuretic
Hypovolemia
SIADH, water intoxication, CHF, or liver disease
Isotonic or hypertonic hyponatremia
Fluid replacement with normal saline
Fluid restriction
Correct underlying disorder

❖ SIADH :

-the syndrome of inappropriate antidiuretic hormone (ADH) secretion (SIADH) is defined by the hyponatremia and hypo-osmolality resulting from inappropriate, continued secretion or action of the hormone despite normal or increased plasma volume, which results in impaired water excretion.

-characterized by

- low plasma osmolality (100 mosm/kg),
- elevated urine sodium (>20 meq/l),
- clinical euvolemia.

-The major causes of SIADH include :

- pulmonary disorders (e.g., atelectasis, empyema, pneumothorax, respiratory failure),
- central nervous system disorders (e.g., trauma, meningitis, tumors, subarachnoid hemorrhage),
- drugs (e.g., cyclophosphamide, cisplatin, nonsteroidal anti-inflammatory drugs),
- ectopic ADH production (e.g., small-cell lung carcinoma).

2-HYPERNATREMIA: is defined as a rise in serum sodium concentration to a value exceeding 145 mmol/l

a-Clinical manifestations:

- primarily neurologic.

- initially lethargy, weakness, and irritability

- may progress to fasciculations, seizures, coma, and irreversible neurologic damage.

b-Types:

- **hypovolemic hypernatremia**:

- any net loss of hypotonic body fluid results in extracellular volume depletion and hypernatremia.

- common causes include diuresis, GI, respiratory, and cutaneous (e.g., burns) fluid losses.

- **hypervolemic hypernatremia**:

- most commonly iatrogenic and results from the parenteral administration of hypertonic solutions (e.g., nahco3, saline, medications, and nutrition).

- IT can also be the result of aldosteronism, cushing disease (secondary hypercortisolism), or mineralocorticoid excess

- **Isovolemic hypernatremia**: - hypotonic losses. - diabetes insipidus

Treatment of Hypernatremia:

- Rapid correction of hypernatremia can result in cerebral edema and permanent neurologic damage.
- Only one half of the water deficit should be corrected over the first 24 hours, with the remainder being corrected over the following 2 to 3 days.
- Serial Na+ determinations are necessary to ensure that the rate of correction is adequate but not excessive.
- If oral intake is not possible, D5W or D5/0.45% NaCl can be substituted.

Clinical manifestations of abnormalities in serum sodium level

BODY SYSTEM	HYPONATREMIA
Central nervous system	Headache, confusion, hyperactive or hypoactive deep tendon reflexes, seizures, coma, increased intracranial pressure
Musculoskeletal	Weakness, fatigue, muscle cramps/ twitching
GI	Anorexia, nausea, vomiting, watery diarrhea
Cardiovascular	Hypertension and bradycardia if intracranial pressure increases significantly
Tissue	Lacrimation, salivation
Renal	Oliguria
BODY SYSTEM	HYPERNATREMIA
Central nervous system	Restlessness, lethargy, ataxia, irritability, tonic spasms, delirium, seizures, coma
Musculoskeletal	Weakness
Cardiovascular	Tachycardia, hypotension, syncope
Tissue	Dry sticky mucous membranes, red swollen tongue, decreased saliva and tears
Renal	Oliguria
Metabolic	Fever

POTASSIUM(Normal concentration3.5-5 mmoL/L)

HYPERKALEMIA: Increase in potassium level above (5mmol/L).

CAUSES:

1- K+ Supplementation, blood transfusion.

2 - Shift from IC to EC space:

- Acidosis
- Rapid rise of EC osmolality (Hyperglycemia Or Manitol)
- Endogenous load / destruction: hemolysis, rhabdomyolysis, crush injury, git hemorrhage, b blocker, digitalis.

3- Impaired excretion:

- K+sparing diuretics , renal insufficiency.

Etiology of potassium abnormalities

Hyperkalemia

Increased intake
- Potassium supplementation
- Blood transfusions
- Endogenous load/destruction: hemolysis, rhabdomyolysis, crush injury, gastrointestinal hemorrhage

Increased release
- Acidosis
- Rapid rise of extracellular osmolality (hyperglycemia or mannitol)

Impaired excretion
- Potassium-sparing diuretics
- Renal insufficiency/failure

Hypokalemia

Inadequate intake
- Dietary, potassium-free intravenous fluids, potassium-deficient TPN

Excessive potassium excretion
- Hyperaldosteronism
- Medications

GI losses
- Direct loss of potassium from GI fluid (diarrhea)
- Renal loss of potassium (to conserve sodium in response to gastric losses)

CLINICAL MANIFESTATION

Mild : asymptomatic. (K+ < 6 mmol/L).

Sever symptomatic (>6.5 mmol/L).

1- **Gastrointestinal**:

Nausea,Vomiting,Diarrhea,Colic.

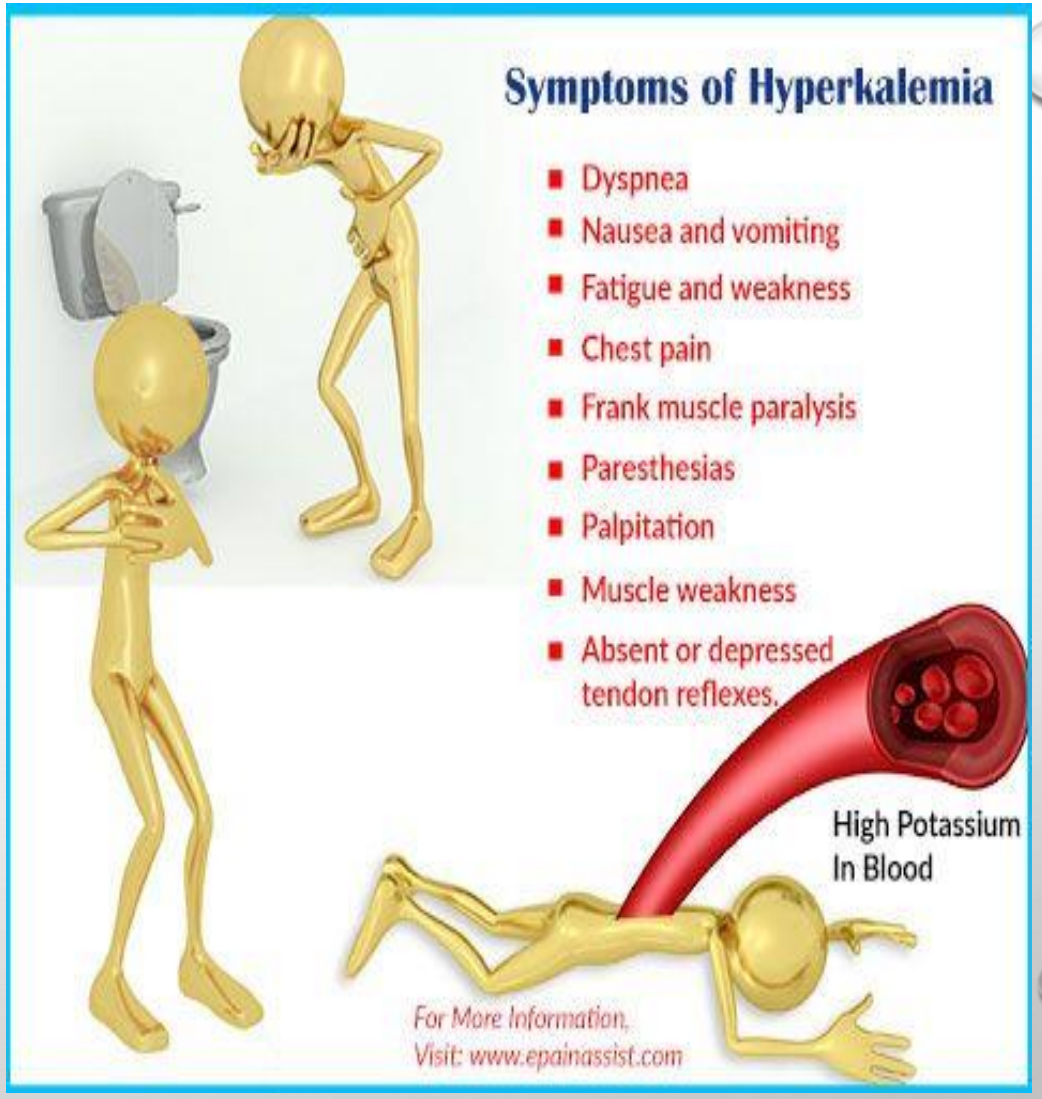
2- **Neuromuscular:**

Hyper/Hyporeflexia, weakness, paralysis RESPIRATORY FAILURE.

3- **Cardiovascular**:

Arrhythmias , Arrest.

ECG changes(peaked T wave, flattened P wave, prolonged pr interval, widened QRS complex, ventricular fibrillation



TREATMENT:

- Mild (5-6mmol/L): can be treated conservatively by the reduction in K+ INTAKE , if needed addition of loop diuretic (e.g furosemide) to promote renal elimination and stop any medication that may cause hyperkalemia.
- Sever (K+>6.5mmol/L): THE GOALS OF THERAPY.

1- **PROTECT THE CELLS FROM THE EFFECTS OF ↑ K+** (COUNTERACT CARDIAC EFFECT) : **Calcium gluconate** 5 – 10 ml of 10% solution(to stabilize the myocardium).

2- **SHIFTING K+ FROM EC TO IC SPACE**: **Glucose** 1 ampule of 50% **dextrose** and **regular insulin** (5 – 10) units i.v, **salbutamol inhaler ,bicarbonate** 1 AMPULE I.V.

3- **REDUCING TOTAL BODY POTASSIUM** (K+ REMOVAL) :

- **Kayexalate** oral administration is 15 – 30 g in 50 – 100 ml of 20% sorbitol rectal administration is 50 g in 200 ml 20% sorbitol
- Hydration with normal saline and loopdiuretic with (adequate renal function)
- **Dialysis** is definitive therapy in severe, refractory, or life-threatening hyperkalemia.

Treatment of symptomatic hyperkalemia

Potassium removal

- Kayexalate
  - Oral administration is 15–30 g in 50–100 mL of 20% sorbitol
  - Rectal administration is 50 g in 200 mL of 20% sorbitol
- Dialysis

Shift potassium

- Glucose 1 ampule of D_{50} and regular insulin 5–10 units IV
- Bicarbonate 1 ampule IV

Counteract cardiac effects

- Calcium gluconate 5–10 mL of 10% solution

D_{50} = 50% dextrose.

HYPOKALEMIA: decrease in potassium level below(3.5mmol/l)

CAUSES:

1- Inadequate intake of potassium (rare)

2- Excessive potassium exertion:

- Hyperaldosteronisn
- Medications (loop diuretics)

3- Losses:

- Git (diarrhea, vomiting, ngt)
- Renal loss
- Burns

4- Intracellular shift

- Metabolic alkalosis, insulin therapy, refeeding syndrome.

CLINICAL MANIFESTATION

Mild: Asymptomatic (3-3.5 mmol/L)

Sever: Symptomatic(K+<3mmol/L).

1-**Gastrointestinal**

Ileus & Constipation

2- **Neuromuscular**

Decreased reflexes, fatigue, weakness, paralysis.

3-**Cardiovascular**

ARREST

ECG changes (ectopy , T wave depression ,U wave)

TREATMENT

Mild(3-3.5mmol/L): Oral repletion with potassium chloride

Sever(K+< 3 mmol/L):

- IF Required , not more than **10** meq/h in unmonitored setting .
- can be increased to **40** meq/h when accompanied by continuous ECG monitoring.

Calcium(Normal concentration 8.9-10.3mg/dL)

- Calcium exists in three forms: ionized (45%), protein bound (40%), and in a complex with freely diffusible compounds (15%). **Only free ionized** ca2+ (4.6-5.1 mg/dL) is physiologically active.
- Normal CA metabolism is under the influence of **PTH AND VIT D**.
- When measuring the total serum CA, the albumin concentration should be taken in consideration. ((Each 1 g/dL reduction in serum albumin will lower the total serum Ca by 0.8 mg/dL , w/o affecting the ionized Ca conc.))
- Unlike changes in albumin, changes in pH affect the ionized Ca concentration. ((Acidosis decreases protein binding, thereby increasing the ionized Ca . While alkalosis increases protein binding , which decreases ionized Ca levels.))

HYPERCALCEMIA: increase in ionized CA or total serum CA.

CAUSES:

- Primary hyperparathyroidism in (the outpatient setting).
- Malignancy in hospitalized patients, from either bony metastasis or secretion of parathyroid hormone–related protein, account for most cases of symptomatic hypercalcemia.
- Hyperthyroidism, vitamin D intoxication, thiazide diuretics ,immobilization.

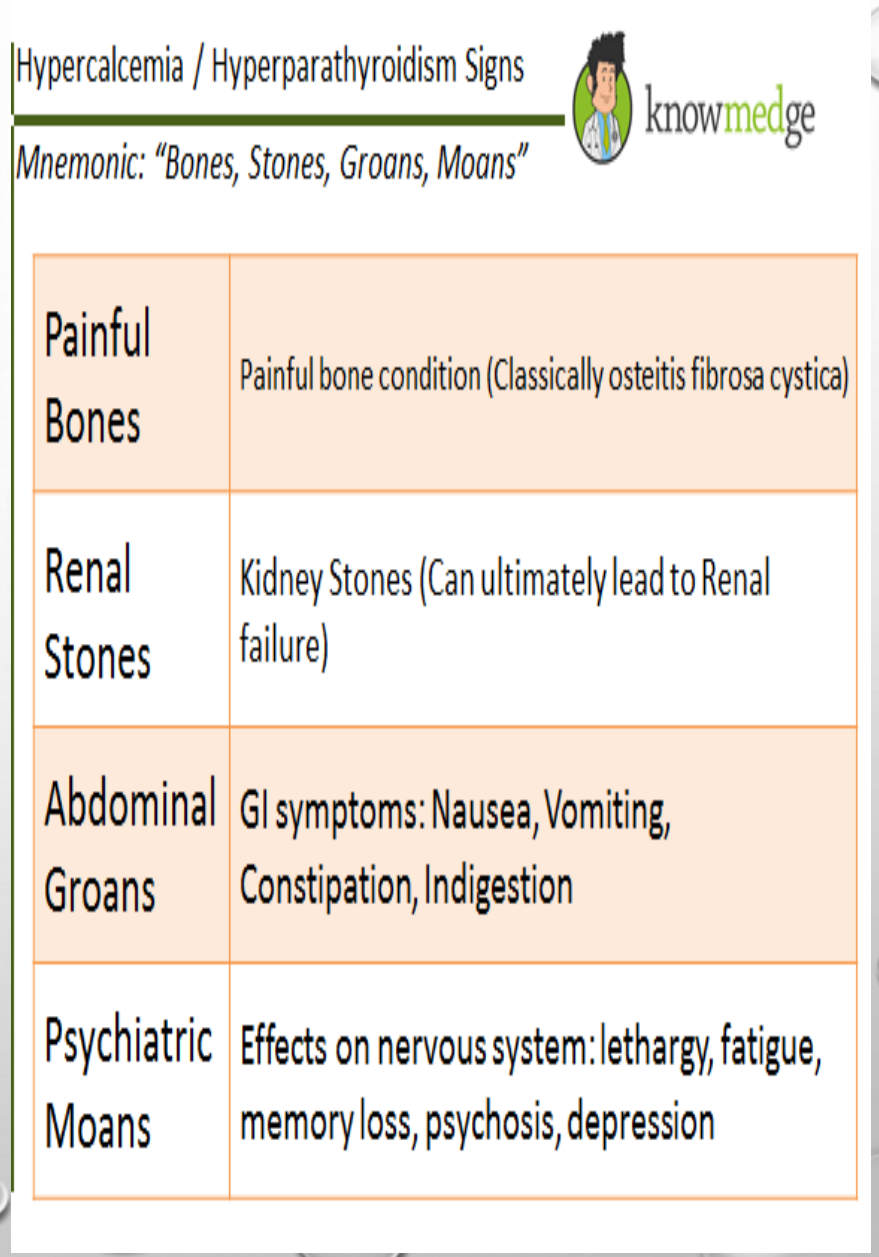
Causes of Hypercalcemia

Mnemonic: "Chimpanzees"

Causes of Hypercalcemia	
C	Calcium supplementation
H	Hyperparathyroidism
I	Iatrogenic, immobilization
M	Multiple myeloma, milk-alkali syndrome, medication (e.g Lithium)
P	Parathyroid hyperplasia or adenoma
A	Alcohol
N	Neoplasm (e.g breast cancer, lung cancer)
Z	Zollinger Ellison syndrome
E	Excessive vitamin D
E	Excessive vitamin A
S	Sarcoidosis

CLINCIAL MANIFESTION

- Mild hypercalcemia (calcium< 12 mg/dL): (ASYMPTOMATIC)
- Severe hypercalcemia: altered mental status, diffuse weakness, dehydration, adynamic ileus, nausea, vomiting, and severe constipation.
- The cardiac effects of hypercalcemia include QT-interval shortening and arrhythmias.



HYPOCALCEMIA: decrease in ionized or total ca concentration.

CAUSES:

1- Calcium sequestration :

- Pancreatitis.
- Massive blood transfusion with citrate binding.
- Rhabdomyolysis and tumor-lysis syndrome
- Massive soft tissue infections (necrotizing fasciitis)
- Increased osteoblastic activity as in prostate or breast ca.

2- Vit d deficiency: renal failure.

3- Hypoparathyroidism

4- Post parathyroidectomy or thyroidectomy.

5- Abnormalities in mg.

CLINCIAL MANIFESTATION

Symptoms don't occur until ionized fraction falls below 2.5 mg/dl.

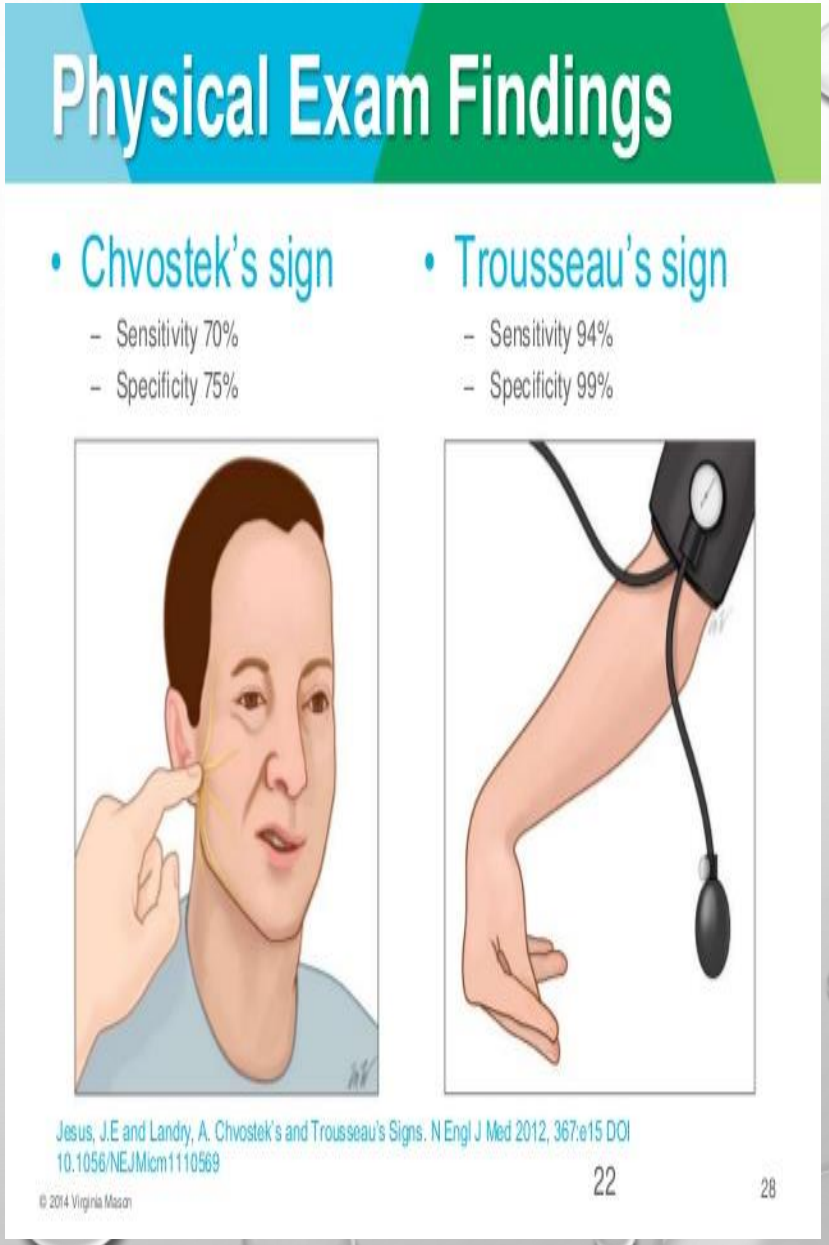
1- Neuromuscular Hyperactive reflexes, Stridor , Tetany, Parasthesia, carpopedal spasm, seizures.

2- Cardiovascular Arrhythmias , Heart failure due to decreased cardiac contractility.

ECG changes: Prolonged QT interval, T-wave inversion, heart block, ventricular fibrillation.

Chevostek's sign: Twitching resulting from taping over the facial nerve.

Trousseau's sign: Spasm resulting from pressure applied to the nerves and vessels of the upper extremity, as when obtaining BP.



TREATMENT

- **Hypercalcemia correction:**
  - Mild hypercalcemia (calcium < 12 mg/dL) can be managed conservatively by restricting calcium intake and treating underling disorder.
  - More severe hypercalcemia may require the following measures:
  - NACL 0.9% and loop diuretics, salmon calcitonin.
- **Hypocalcemia correction:**
  - Asymptomatic: can be treated with oral or I.V. Ca , Ca carbonate and Ca gluconate
  - Symptomatic: should be treated by I.V. Ca , Ca chloride (10 ml calcium of 10% calcium gluconate within first 10 min then maintenance with 1-2 ml/h). Don't forget to check the levels of Mg, Phosphorus, K.

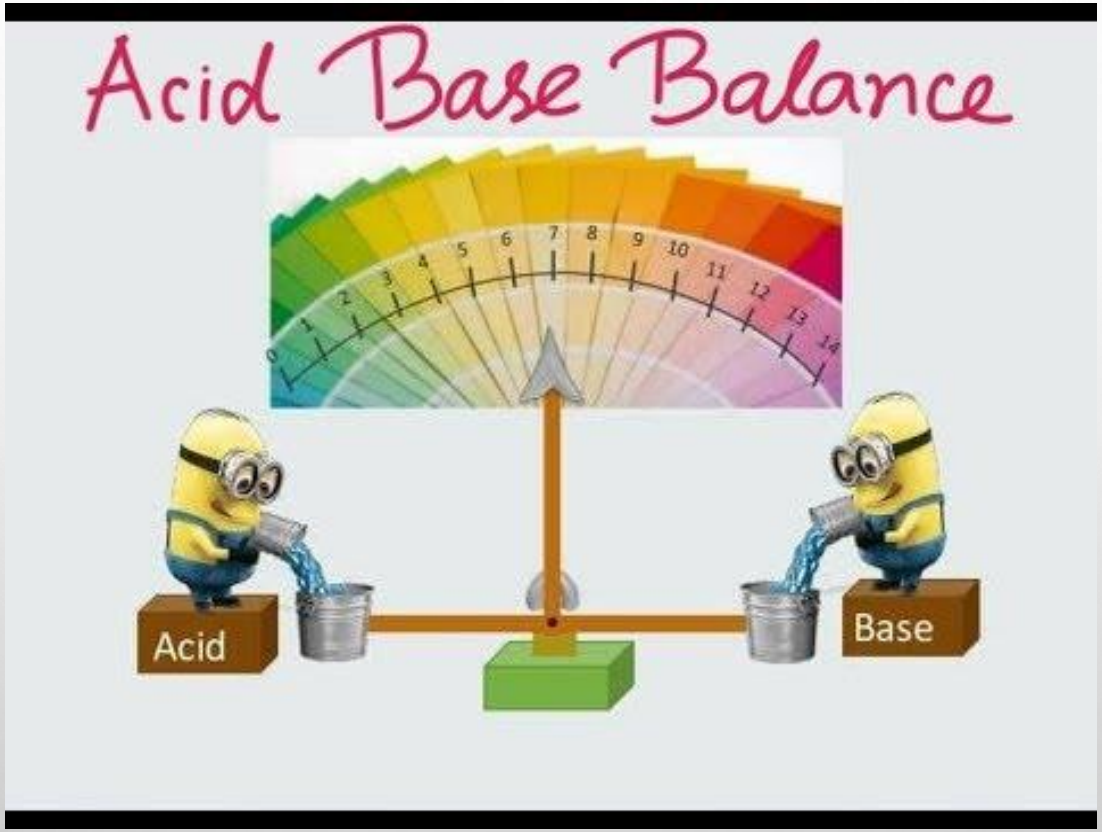
INCREASED SERUM LEVELS

SYSTEM	POTASSIUM	MAGNESIUM	CALCIUM
GI	Nausea/vomiting, colic, diarrhea	Nausea/vomiting	Anorexia, nausea/vomiting, abdominal pain
Neuromuscular	Weakness, paralysis, respiratory failure	Weakness, lethargy, decreased reflexes	Weakness, confusion, coma, bone pain
Cardiovascular	Arrhythmia, arrest	Hypotension, arrest	Hypertension, arrhythmia, polyuria
Renal	—	—	Polydipsia

DECREASED SERUM LEVELS

SYSTEM	POTASSIUM	MAGNESIUM	CALCIUM
GI	Ileus, constipation	—	—
Neuromuscular	Decreased reflexes, fatigue, weakness, paralysis	Hyperactive reflexes, muscle tremors, tetany, seizures	Hyperactive reflexes, paresthesias, carpopedal spasm, seizures
Cardiovascular	Arrest	Arrhythmia	Heart failure

ACID-BASE BALANCE



QUICK REMINDER

- PH is a measure of how acidic/basic solution. the range goes from 0 - 14, with 7 being neutral. PH of less than 7 indicate acidity(proton donors), whereas a ph of greater than 7 indicates a base(proton acceptor).
- Normal blood PH is kept within a very limited range (7.35-7.45)
- It is very important to keep the PH of the blood and tissues around 7.4, which is suitable to the functions of most body enzymes. (enzymes, which control metabolic reactions, are very sensitive to changes in PH)
- BUT HOW THE BODY KEEP THE PH WITH THIS VERY LIMTED RANGE ?

TABLE 4–1 Normal Blood Gas Values

Blood Gas Value*	Arterial	Venous
pH	7.35 to 7.45	7.30 to 7.40
Pco_2	35 to 45 mm Hg	42 to 48 mm Hg
HCO_3	22 to 28 mEq/L	24 to 30 mEq/L
Po_2	80 to 100 mm Hg	35 to 45 mm Hg

*Technically, only the oxygen (Po_2) and carbon dioxide (Pco_2) pressure readings are true blood gas values. The pH indicates the balance between the bases and acids in the blood. The bicarbonate (HCO_3) reading is an indirect measurement that is calculated from the pH and Pco_2 levels.

PH FIGHTERS

There are 3 lines of defense **(PH fighters)** which regulate PH:

1- The first line of defense is the **blood buffer.**

- Works within seconds (bind acid/base ➡ weaker).

2- The second line of defense is the **respiratory regulation.**

- Works within minutes (eliminate CO2).

3- The third line of defense is the **renal regulation.**

- Works within hours-days (excrete acid/base). the most powerful of the three.

BLOOD BUFFERS

DEF. they are solutions that **resist changes** in their PH when moderate amounts of acids or bases are added.

COMPOSITION: TWO TYPES

1- MIXTURES OF WEAK ACIDS AND THEIR SALT WITH A STRONG BASE.

- Example:: Carbonic acid / NA-bicarbonate mixture ($H_2CO_3/NAHCO_3$).

2- Mixtures of weak bases and their salt with a strong acid.

- Example: Ammonium hydroxide / Ammonium chloride (NH_4OH /NH_4CL) mixture
- Addition of excess amounts of acids or bases to a buffer, **may cause depletion** of the buffer system which is followed by marked change in the PH of the solution.

TYPES OF BUFFER:

1- Physiological buffer systems:

- The most important physiological buffer systems are:
  - a. Bicarbonate system.
  - b. Phosphate system.
  - c. Protein system.

2- Blood buffer systems:

- The protein system includes the plasma proteins, albumin, globulins, and fibrinogen, in the plasma, and the hemoglobin and oxyhemoglobin buffering system in the red blood cells. proteins work as buffer systems because of their components (negatively charged & contain amino acids).
- Buffers act quickly, but not permanently.
- For the final elimination of acids, **the respiratory and renal regulations** are very essential

RESPIRATORY REGULATION

- The second line of defense.
- This is achieved by changing the pCO2.The CO2 diffuses from the cells into the extracellular fluid and reaches the lungs through the blood.
- The rate of respiration (rate of elimination of CO2) is controlled by the chemoreceptors in the respiratory center which are sensitive to changes in the PH of blood.
- when there is a fall in PH of plasma (acidosis), the respiratory rate is stimulated resulting in hyperventilation. this would eliminate more CO2, thus lowering the H2CO3 level.
- However, this cannot continue for long. the respiratory system responds to any change in PH immediately, but it cannot proceed to completion.

RENAL REGULATION

- The third line of defense and the most powerful.
- This achieved by kidneys conserve HC03 and excrete acidic or basic urine depending on body needs.
- The kidney regulate PH through three fundamental mechanism:

  1- secretion H+. 2- reabsorption of HCO3. 3- production new HCO3.
- In acidosis, the kidney do not excrete HCO3 but reabsorb all HCO3 from urine and produce new HCO3.
- In alkalosis the kidney fail to reabsorb HCO3 thus increasing HCO3 excretion.
- Return PH back to normal within 12-14 hours.

ACID-BASE DISTURBANCES

- RESPIRATORY ACIDOSIS
- RESPIRATORY ALKALOSIS
- METABOLIC ACIDOSIS
- METABOLIC ALKALOSIS

RESPIRATORY ACIDOSIS

❖It is caused by increased plasma H_2CO_3 due to failure of the lungs to excrete CO_2 at the proper rate.

Causes:

- In the surgical patient include respiratory center depression (e.g., drugs and organic disease), neuromuscular disorders, and cardiopulmonary arrest.
- chronic respiratory acidosis may occur in pulmonary diseases, such as chronic emphysema and bronchitis.

RESPIRATORY ACIDOSIS CONT.

- In acute respiratory acidosis the $PaCO_2$ is elevated above the upper limit of the reference range (>45 mmHg) with accompanying acidemia (PH<7.35). It occurs following an abrupt failure of ventilation.

(HCO_3 normal --- H_2CO_3 Increased --- PH <7.35) **uncompensated respiratory acidosis.**

- In chronic respiratory acidosis the $PaCO_2$ is elevated above the upper limit with a normal

or near normal PH due to renal compensation and an elevated bicarbonate level.

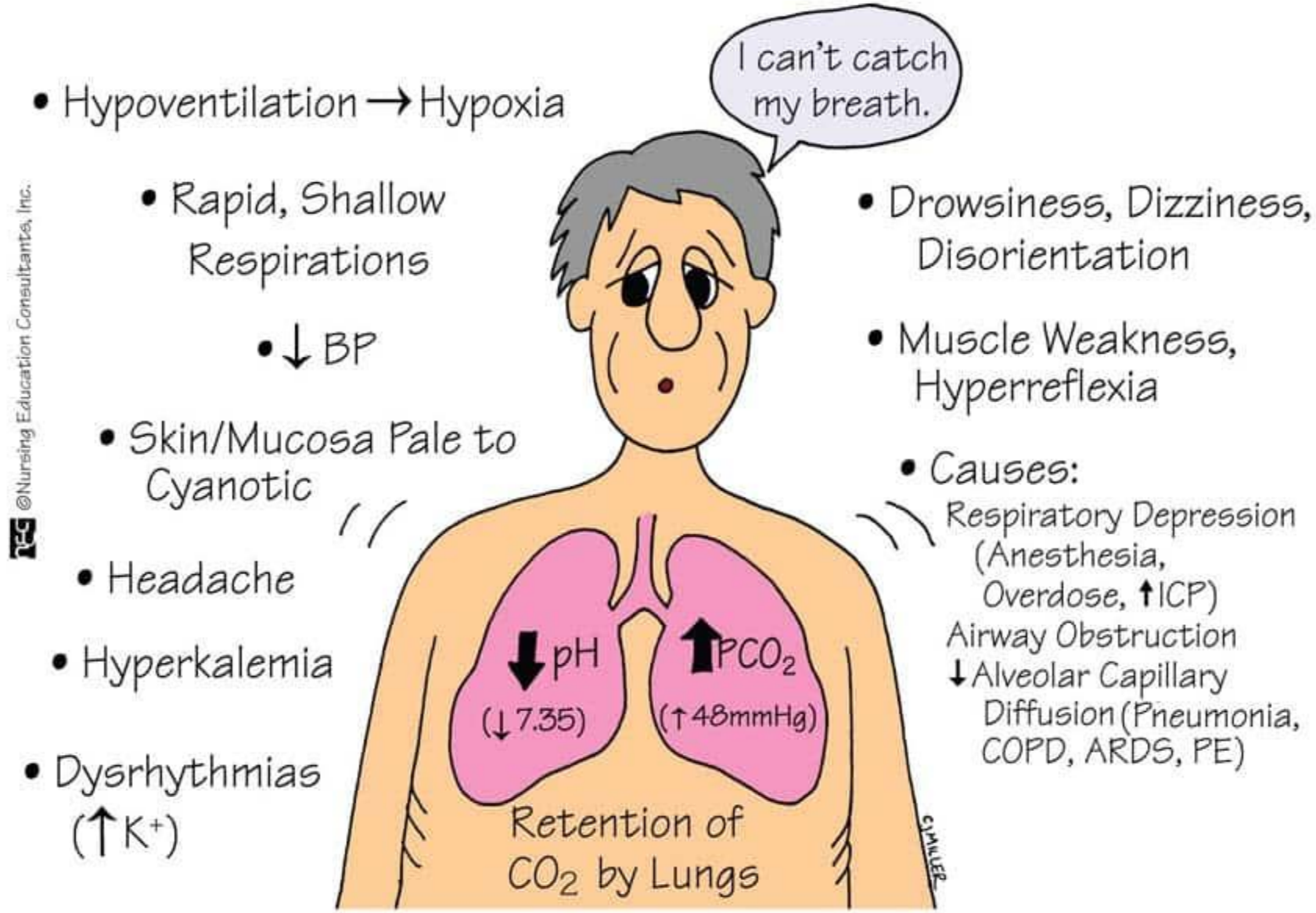
(HCO_3 increased --- H_2CO_3 increased ---PH near 7.35) **compensated respiratory acidosis.**

RESPIRATORY ACIDOSIS CONT.

- **Symptoms:**
  - Dyspnea, disorientation or coma.
  - Dysrhythmias.
  - HYPERKALEMIA OR HYPOXEMIA.
- **Treatment:**
  - Treat the underlying cause.
  - support ventilation correct electrolyte imbalance.
  - IV sodium bicarbonate

in cases of acute respiratory acidosis, there is no indication for NaCHCO3 administration.

RESPIRATORY ACIDOSIS



RESPIRATORY ALKALOSIS

❖It is caused by decreased plasma H_2CO_3 due to increased loss of CO_2 through the lungs.

Causes:

- Acute hypoxia (e.g., pneumonia, pneumothorax, pulmonary edema, and bronchospasm)
- Chronic hypoxia (e.g., cyanotic heart disease and anemia)
- Respiratory center stimulation (e.g., anxiety, fever, gram-negative sepsis, salicylate intoxication, central nervous system disease, cirrhosis, and pregnancy)
- Excessive ventilation in the mechanically ventilated patient.

RESPIRATORY ALKALOSIS CONT.

- in acute respiratory alkalosis the PaCO2 level below the lower limit of normal (<35mmhg) and serum PH is alkalemic (PH>7.45).

(HCO3 - normal---H2CO3 decreased---PH>7.45) **uncompensated respiratory alkalosis**.

- In chronic respiratory alkalosis the PaCO2 level below the lower limit of normal with a normal or near normal PH due to renal compensation and an decreased bicarbonate level.

(HCO3 - decreased---H2CO3 decreased---PH near 7.45) **compensated respiratory alkalosis**.

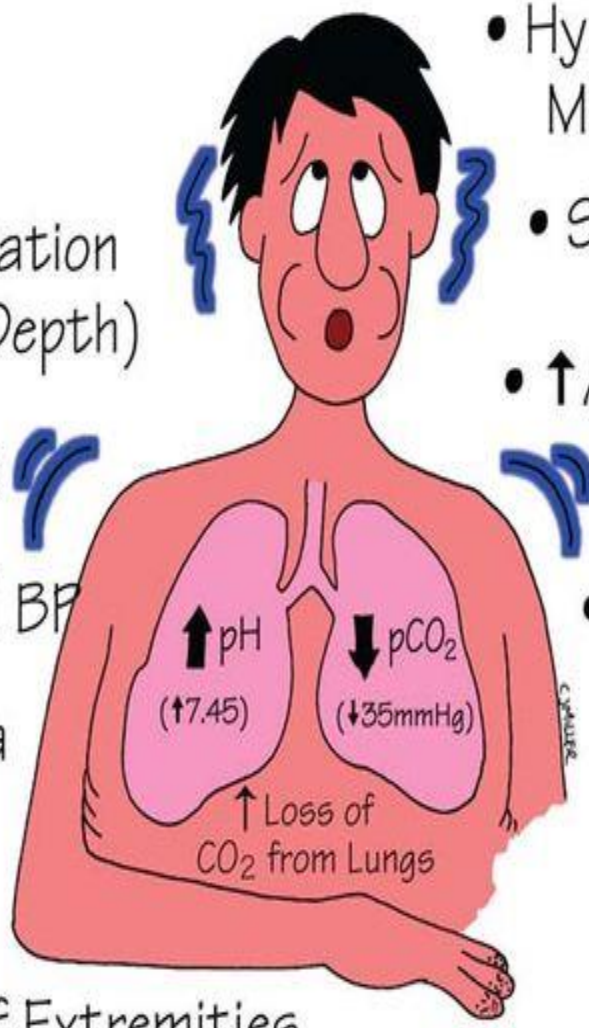
RESPIRATORY ALKALOSIS CONT.

- **Symptoms:**
  - TACHYPNEA OR HYPERPNEA.
  - CHEST PAIN.
  - LIGHT-HEADEDNESS, SYNCOPE, COMA, SEIZURES.
  - NUMBNESS AND TINGLING OF EXTREMITIES.
  - DIFFICULT CONCENTRATING, TREMORS, BLURRED VISION.
  - WEAKNESS, PARESTHESIA, TETANY.

RESPIRATORY ALKALOSIS CONT.

- **Treatment:**
  - Treating the underlying cause.
  - Decreasing the respiratory rate and tidal volume of the patient. On respiratory support.
  - Reassure anxious patient.
  - Sedation and pain control.

RESPIRATORY ALKALOSIS



- Hyperventilation (↑Rate & Depth)
- Tachycardia
- ↓or Normal BP
- Hypokalemia
- Numbness & Tingling of Extremities
- Hyper Reflexes & Muscle Cramping
- Seizures
- ↑Anxiety, ↑Irritablity
- Causes:
  - Hyperventilation (Anxiety, PE, Fear)
  - Mechanical Ventilation

METABOLIC ACIDOSIS

❖Occurs when the body produces excessive quantities of acid or when the kidneys are not removing enough acid from the body.

Causes:

- Increased anion gap (normochloremic): diabetic ketoacidosis, lactic acidosis, starvation, toxic ingestion (salicylates, ethylene glycol, methanol), renal failure.
- Normal anion gap (hyperchloremic): diarrhea, renal tubular acidosis, carbonic anhydrase inhibitors, Addison's disease.

(HCO3 decreased---H2CO3 normal---PH <7.35) **uncompensated metabolic acidosis.**

(HCO3 decreased--H2CO3 decreased---PH near 7.35) **compensated metabolic acidosis.**

METABOLIC ACIDOSIS CONT.

❖anion gap: the difference between measured cation and measured anion.

In body fluids: total cations= total anions. NA =CL- + HC03- + unmeasured anions.

Unmeasured anions = NA - CL- - HCO3 = ANION GAP. 142 - 108 - 24=10 mEq/L.

Normal anion gap = 8-16 mEq/L

- it help us to know if metabolic acidosis is due to:
  - loss of bicarbonate.
  - accumulation of volatile (organic anions).
- Based on this concept metabolic acidosis is categorized into normal anion gap(hyperchloremic) and increased anion gap (normochloremic).

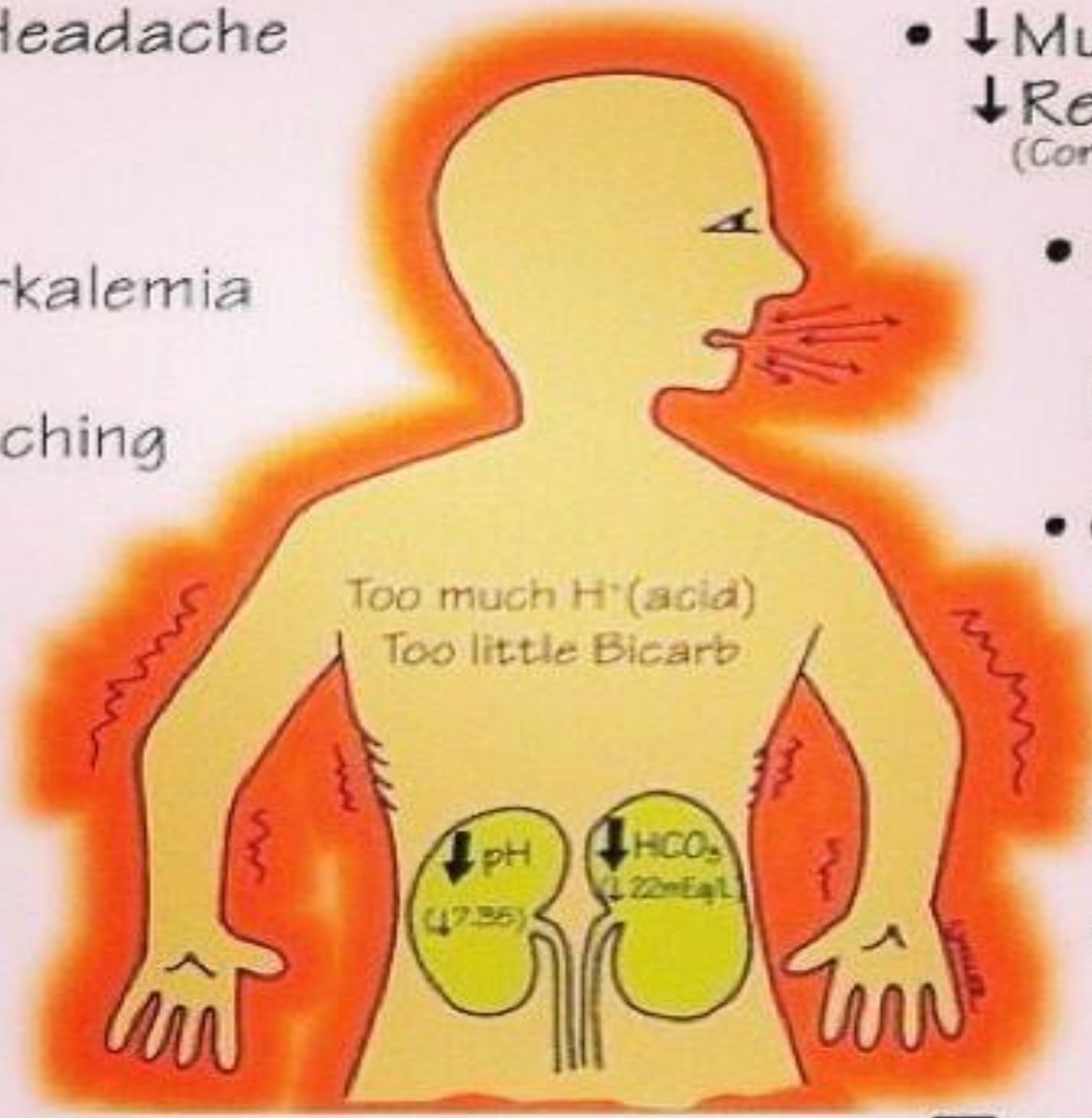
METABOLIC ACIDOSIS CONT.

- **Symptoms:**
  - Kussmaul's respiration.
  - Lethargy, confusion, headache, weakness .
  - Nausea, vomiting.
  - Warm flushed skin.
- **Treatment:**
  - Treat underlying cause.
  - Bicarbonate therapy should be considered in patients with moderate-to-severe metabolic acidosis only after the primary cause has been addressed.

METABOLIC ACIDOSIS

@nurse_elsie

- Headache
- ↓BP
- Hyperkalemia
- Muscle Twitching
- Warm, Flushed Skin (Vasodilation)
- Nausea, Vomiting



- ↓Muscle Tone, ↓Reflexes (Confusion, ↑Drowsiness)
- Kussmaul Respirations (Compensatory Hyperventilation)
- Causes:
  - ↑H⁺ Production (DKA, hyper-metabolism)
  - ↓H⁺ Elimination (renal failure)
  - ↓HCO_3 Production (dehydration, liver failure)
  - ↑HCO_3 Elimination (diarrhea, fistulas)

METABOLIC ALKALOSIS

❖Refers to a disorder involving a primary increase in HCO3 resultant rise in PH.

Causes:

- Excessive loss of acid: vomiting , gastric lavage, hypokalemia.
- Volume contraction: diuretic therapy(thiazid, loop diuretic), hypovolemia.
- Excessive alkali intake.
- Mineralocorticoid excess: cushing syndrome, primary aldosteronism.

(HCO3 - increased---H2CO3 NORMAL---PH>7.45) **uncompensated metabolic alkalosis**.

(HCO3 - increased---H2CO3 INCREASED---PH near 7.45) **compensated metabolic alkalosis**.

METABOLIC ALKALOSIS CONT.

- **Symptoms:**
  - Hypoventilation.
  - Dysrhythmias, dizziness.
  - Paresthesia, numbness, tingling of extremities.
  - Hypertonic muscles, tetany.

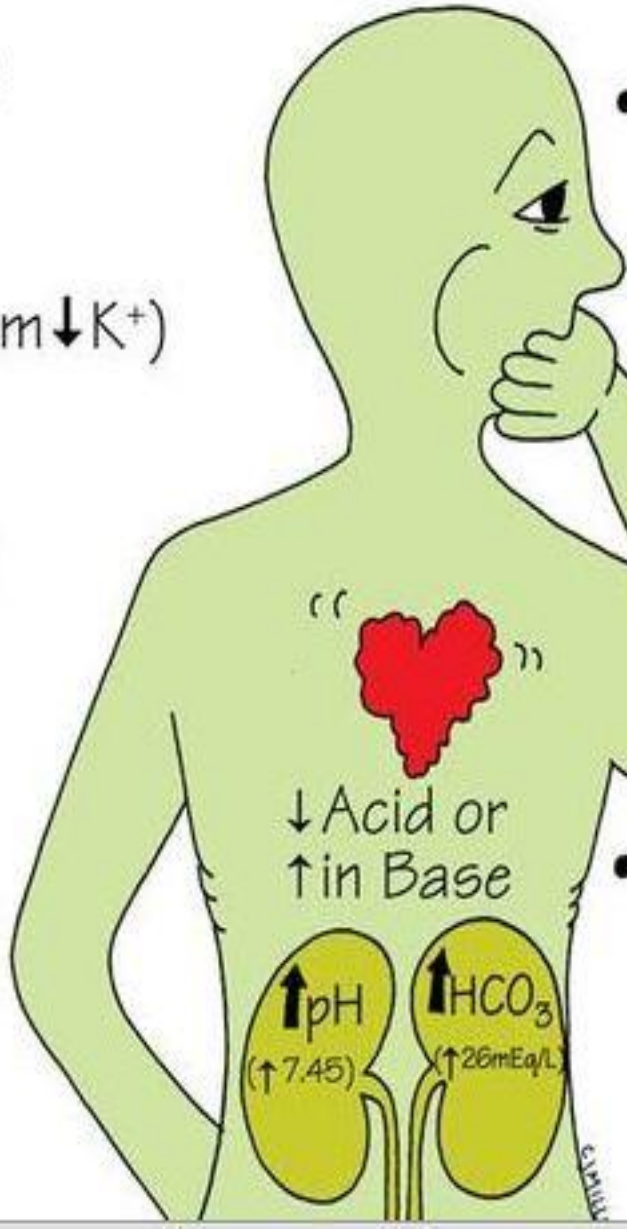
METABOLIC AKLALOSISCONT.

Treatment:

- Treat underlying cause.
- initial therapy should include the correction of volume deficits (with 0.9% NACL) and hypokalemia.
- severe alkalemia ($HCO_3 > 40$ mmol/L):ammonium chloride (NH_4CL) is hepatically converted to urea and HCL
- Dialysis can be considered in the volume-overloaded patient with renal failure and intractable metabolic alkalosis.

METABOLIC ALKALOSIS

- Confusion
- Dysrhythmias (Tachycardia from ↓K^+)
- Compensatory Hypoventilation
- Causes:
  - ↑ HCO_3 (Antacids, admin of sodium bicarbonate)
  - ↓H^+ (NG Suctioning, Prolonged Vomiting, Hypercortisolism)



- Dizzy, ↑Irritability
- Nausea, Vomiting, Diarrhea
- ↑Anxiety, Seizures
- Tremors, Muscle Cramps, Tingling of Fingers & Toes (↓serum Ca^{++})

DIAGNOSIS OF ACID BASE BALANCE DISTURBANCES

1. Comprehensive history and clinical examination.
2. Areteral blood gases analysis(ABG).
3. Serum electrolytes.

History & examination

- History of underling disease eg DM may aid in raising suspiscion for certain acid base disorder, history of poisoning or drug abuse.
- Clinical signs such as hyperventilation, kussmaul breathing , wheezing , vomiting and diarrhea may help in diagnosis.

ABGS

- Important for assessing patients ventilation, oxygenation and acid base status.
- PH and PCO2 are measured values while HCO3 is calculated using the henderson-hasselbalch equation.

THANK YOU