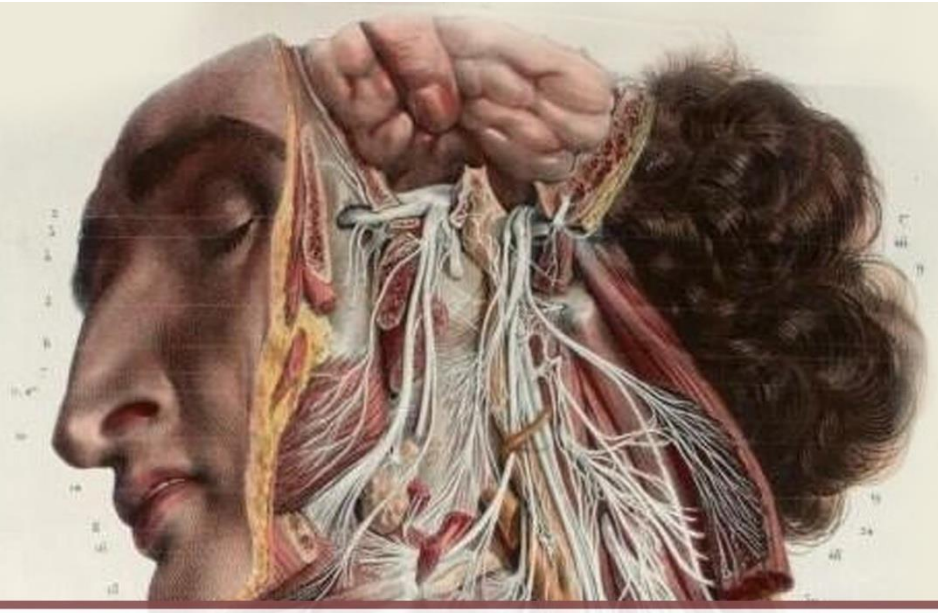
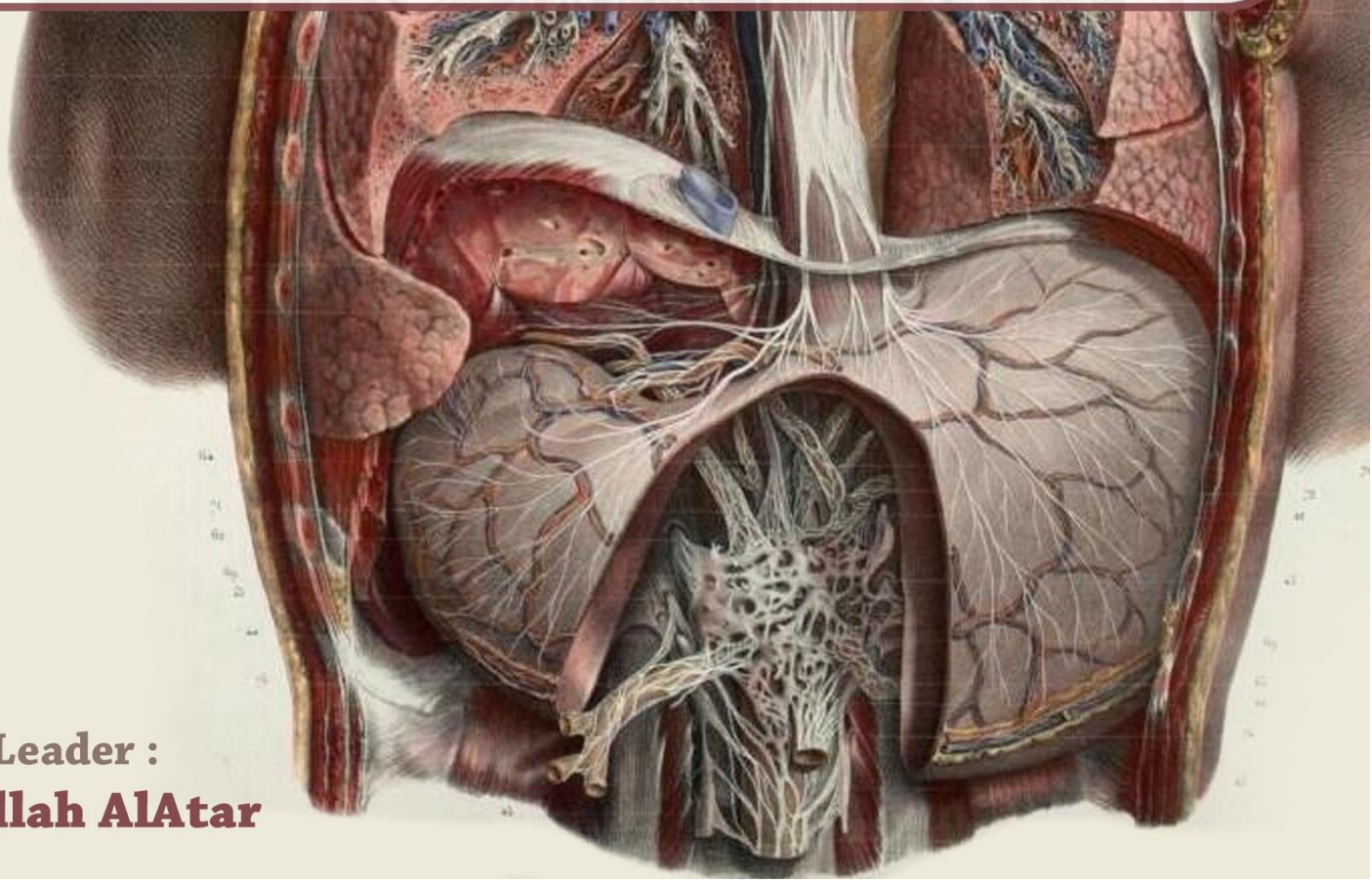


433 SURGERY TEAM

Fluid and Electrolyte Balance

Done By:

Faisal S. AlGhamdi Abdullah Almousa



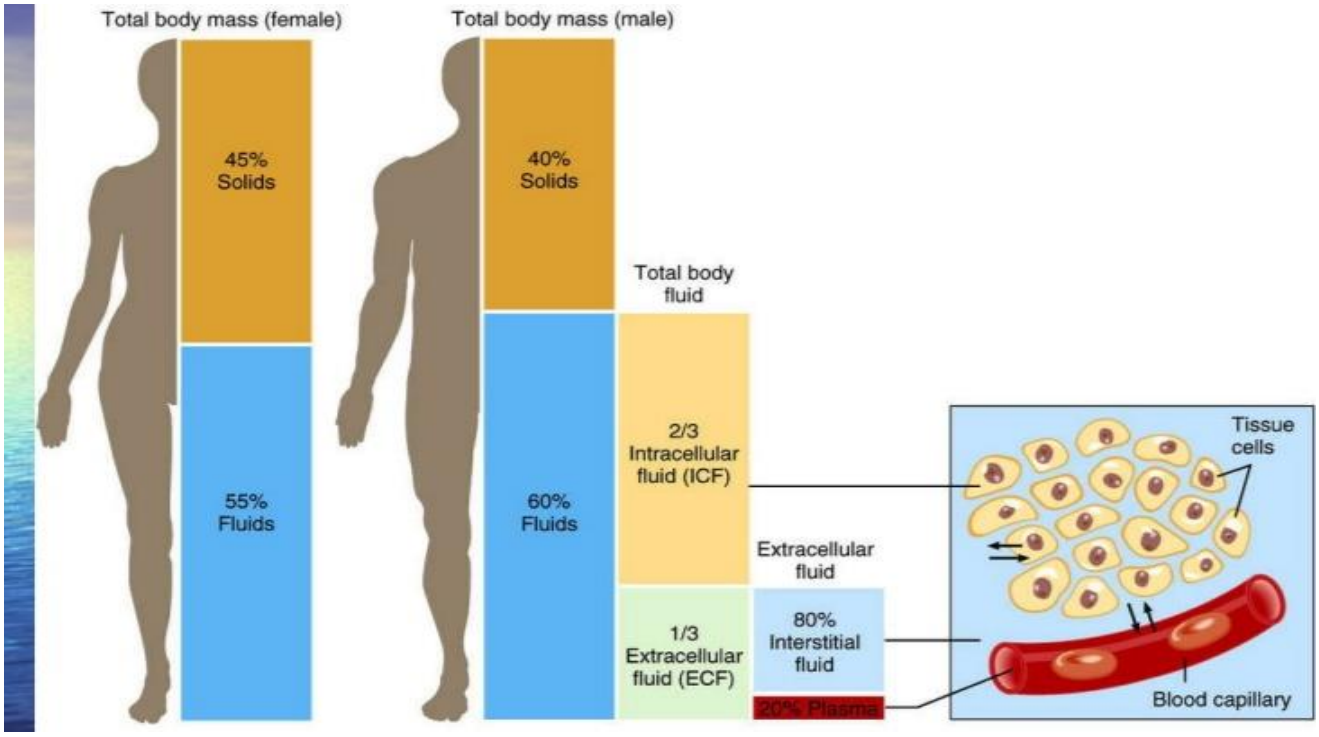
Team Leader :

Abdullah AlAtar

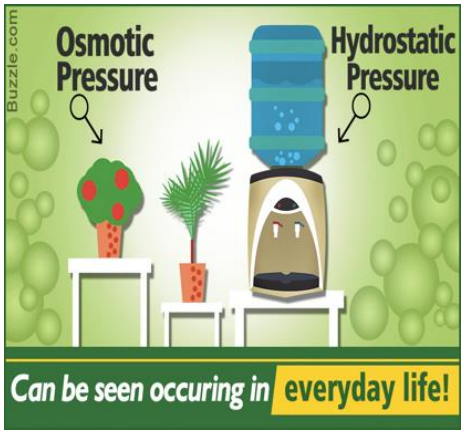
Total Body Fluids and fluids compartment:

- **60% in male**
  of Total body weight
- **55% in female**

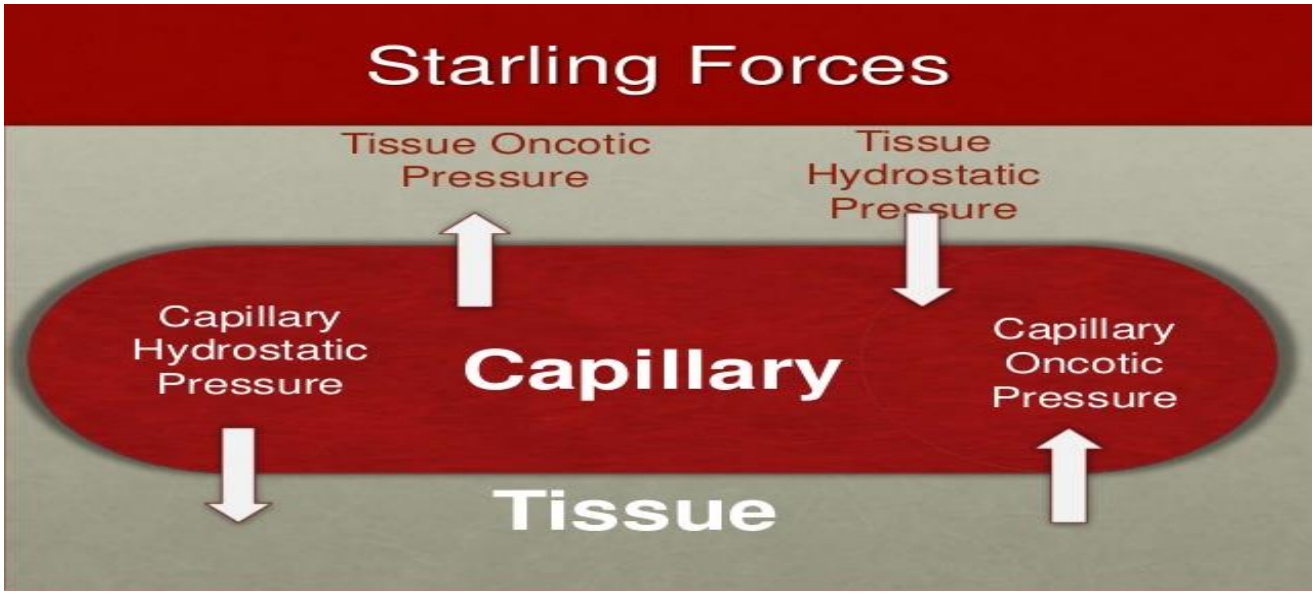
- 2/3 (65%) of TBW is intracellular (ICF)
- 1/3 (35%) extracellular water
  - 25 % interstitial fluid (ISF)
  - 5 - 7 % in plasma (IVF intravascular fluid)
  - 1- 2 % in transcellular fluids – CSF, intraocular fluids, serous membranes, and in GI, respiratory and urinary tracts (third space)



- **Fluid compartments are separated by membranes that are freely permeable to water.**
- **Movement of fluids due to:**
  - **Hydrostatic pressure (Fluid)**
  - **Osmotic/Oncotic pressure (tissue)**



- **In Hydrostatic pressure: As the pressure increase as the movement of fluid outside increase**
- **In Osmotic pressure: As the pressure increase as the absorption of fluid increase.**

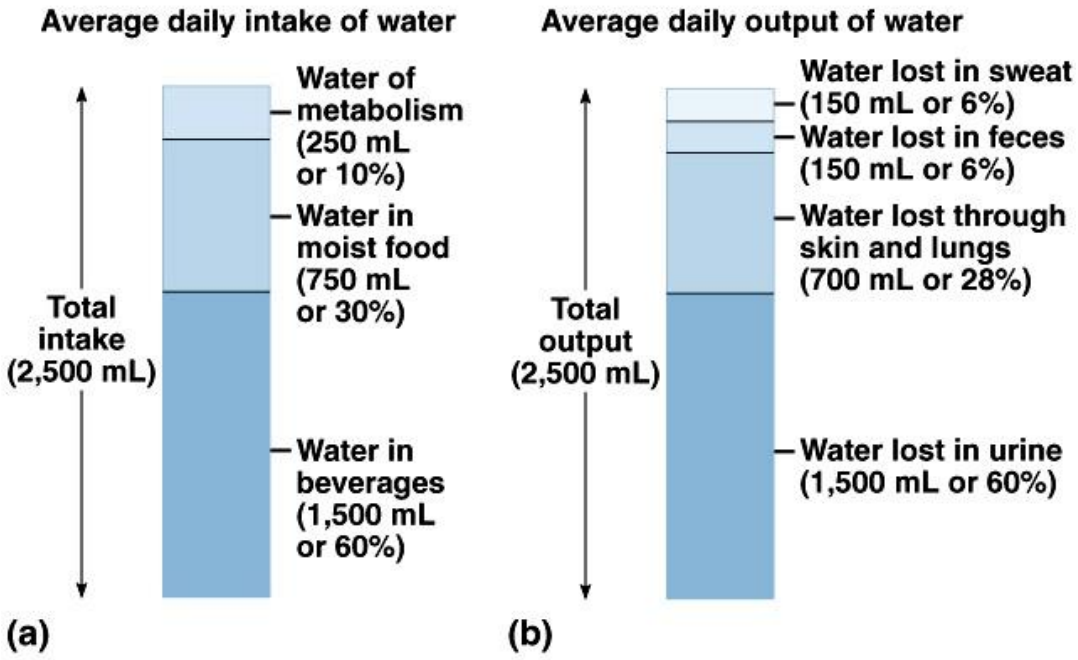


Fluid balance:

- **Neutral balance: input = output**
- **Positive balance: input > output**
- **Negative balance: input < output**
  (+ve lead to edema, and -ve lead to dehydration)

Daily input should = Daily output

- **Most of water intake in Beverages**
- **Most of water output in Urine**



Electrolytes:

- **Cations – positively charged ions**
  - **Na^+, K^+ , Ca^{++}, H^+**
- **Anions – negatively charged ions**
  - **Cl^-, HCO_3^- , PO_4^{3-}**

Intracellular fluid space:

- 40% of body weight
- Largest proportion is in skeletal muscle
- Larger percentage of water is Intracellular in males (large muscle mass)
- **Cations = Potassium & Magnesium**
- **Anions = Phosphates and Proteins**

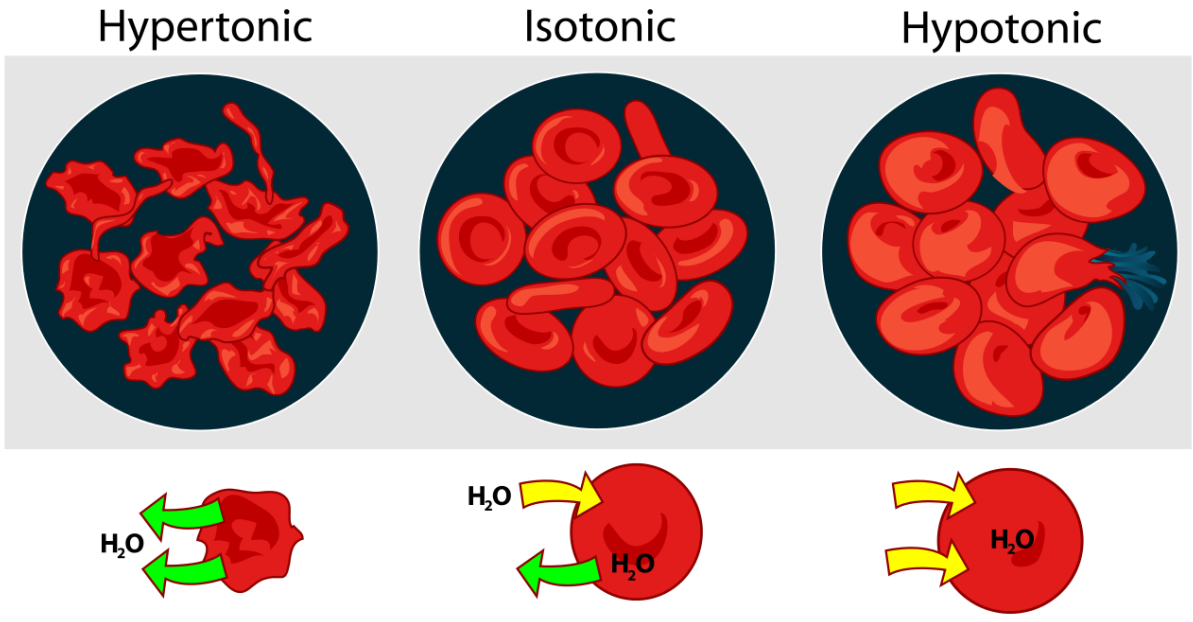
Extracellular fluid space:

- 20% of body weight
- Interstitial 15%, Plasma 5%
- **Cations = Sodium**
- **Anions = Chloride and Bicarbonate**

Homeostasis:

- **Maintained by Ion transport, Water movement and Kidney function.**
- **Tonicity Isotonic, Hypertonic and Hypotonic**

(the difference btw tonicity and osmolarity that tonicity is concentration of solutions in relation to adjacent compartments *like concentration of plasma compared to interstitial space*, but osmolarity take the compartment on it's own)



Movement of body fluids:

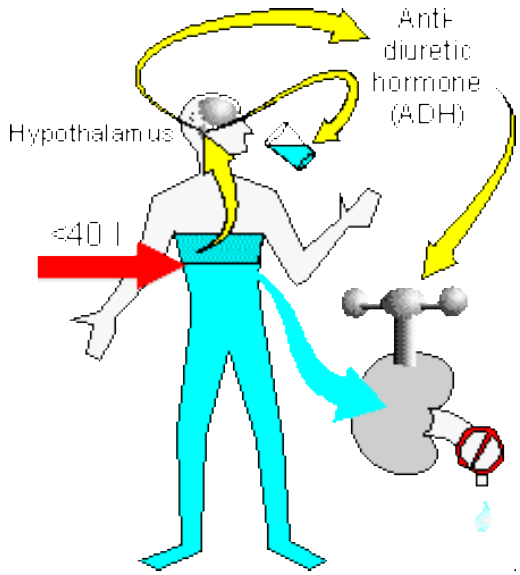
" Where Na goes, H2O follows"

- Diffusion – movement of particles down a concentration gradient.
- Osmosis – diffusion of water across a selectively permeable membrane from low to high osmolarity.
- Active transport – movement of particles up a concentration gradient ''requires energy''.

Regulation of body water: (ADH)

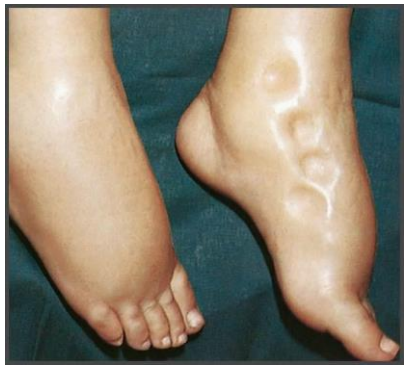
- **Stimulated by osmo-receptors in hypothalamus:**
  - **ADH released from posterior pituitary + thirst**
    - **Decreased amount of water in body**
    - **Increased amount of Na+ in the body**
    - **Increased blood osmolality**
    - **Decreased circulating blood volume**

  - **The result from ADH:**
    ↑ water consumption
    ↑ water conservation
    ↑ water in body
    ↑ volume
    ↓Na+ concentration



Edema:

Accumulation of fluid within the interstitial spaces.

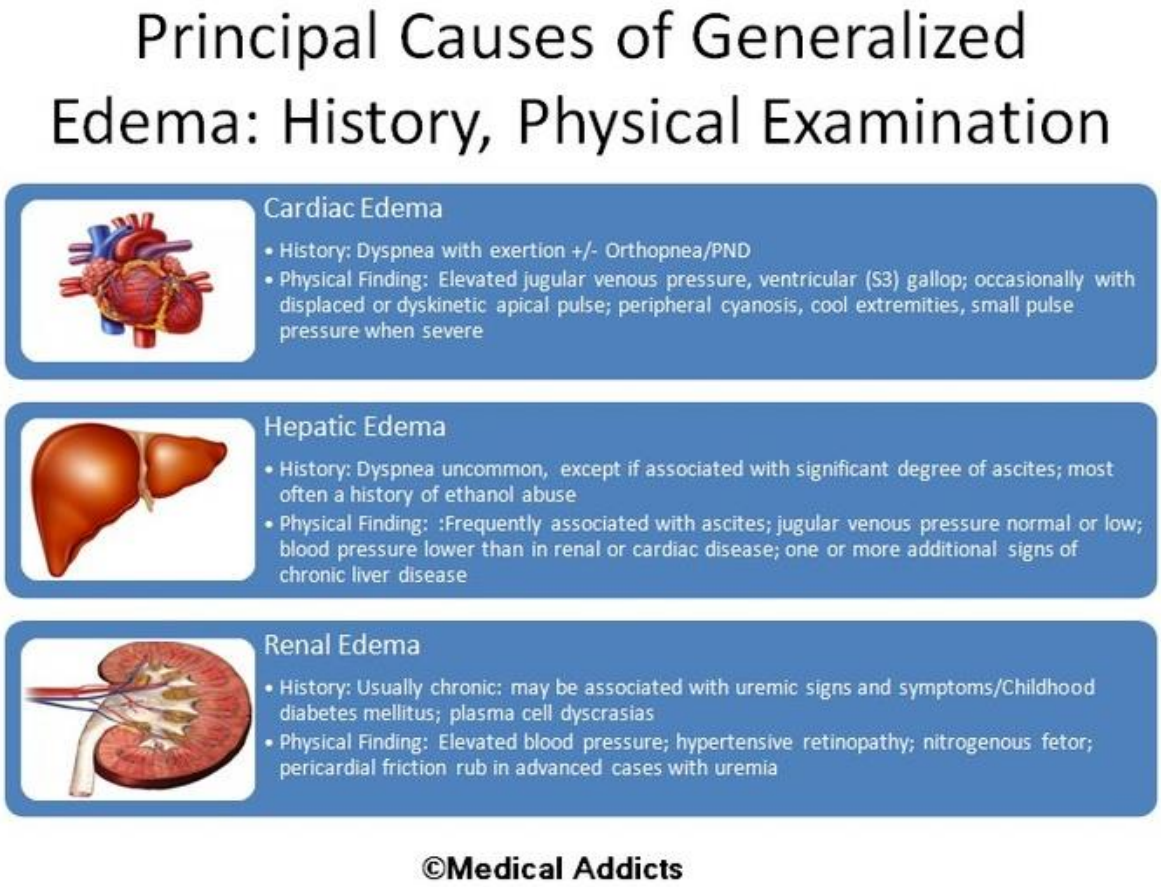


Causes:

- **↑ hydrostatic pressure**
- **↓ plasma osmotic pressure**
- **↑ capillary membrane permeability**

 # Lt. sided heart failure cause pulmonary edema, and Rt. Sided cause edema in body .. with long term each side going to cause failure of the other.

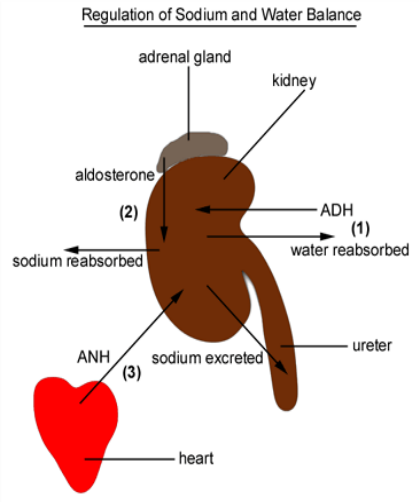
 # Nephrotic syndrome cause edema due to low albumin, leading to low oncotic pressure)



Electrolyte balance:

- **Na^+ (Sodium):**
  - 90 % of total ECF cations
  - **ECF: 136 -145 mEq / L**
  - Pairs with Cl^- , HCO_3^- to neutralize charge
  - Low in ICF
  - Most important ion in regulating water balance
  - Important in nerve and muscle function "Action Potential"

- **Regulation of Na^+ (Sodium):**
  - **Renal tubule reabsorption affected by hormones:**
    - **Aldosterone** (reabsorption of Na)
    - **ADH** (reabsorption of Na)
    - **Renin/angiotensin** (secrete ADH/Aldosterone)
    - **Atrial Natriuretic Peptide (ANP)** (Excrete Na)



- **K^+ (Potassium):**
  - Major intracellular cation.
  - **ECF = 3.5 – 5.5 mEq / L**
  - Resting membrane potential.
  - Regulates fluid, ion balance inside cell.

Alterations in water balance:

❖ Hypovolemia:

Characteristics:

- ↓ECF volume
- Weight loss
- Dry skin and mucous membranes, cold extremities
- ↓ Urine output
- Rapid heart rate
- Flattened neck veins
- Normal or ↓ B.P – may lead to shock

❖ Volume overload:

Causes:

- Excess IV fluids
- Hypersecretion of aldosterone (e.g. Conn's disease)
- Effect of drugs as cortisone

Characteristics:

- Weight gain
- Decreased hematocrit
- Diluted plasma proteins
- Distended neck veins.
- May lead to edema (↑ capillary hydrostatic pressure) pulmonary edema.

Colloids Vs Crystalloids:

	Crystalliod	Colloid
Intravascular persistance	Poor	Good
Haemodynamic stabilisation	Transient	Prolonged
Required infusion volume	Large	Moderate
Risk of tissue oedema	Obvious	Insignificant
Enhancement of capillary perfusion	Poor	Good
Risk of anaphylaxis	Nil	Low to moderate
Plasma colloid osmotic pressure	Reduced	Maintained
Cost	Inexpensive	Expensive

As there are no difference in benefit, and colloid are more expensive and have more complication, **crystalloid preferred**

EBM **1.1 Crystalloid vs colloid to treat intravascular hypovolaemia**

'There is no evidence that resuscitation with colloids reduces the risk of death, compared to resuscitation with crystalloids, in patients with trauma, burns or following surgery.'

#Dextrose will be metabolized then become hypotonic, so fluid shift to interstitial apace. Isotonic solutions, larger amount will stay intravascular. Colloid like albumin will stay intravascular.

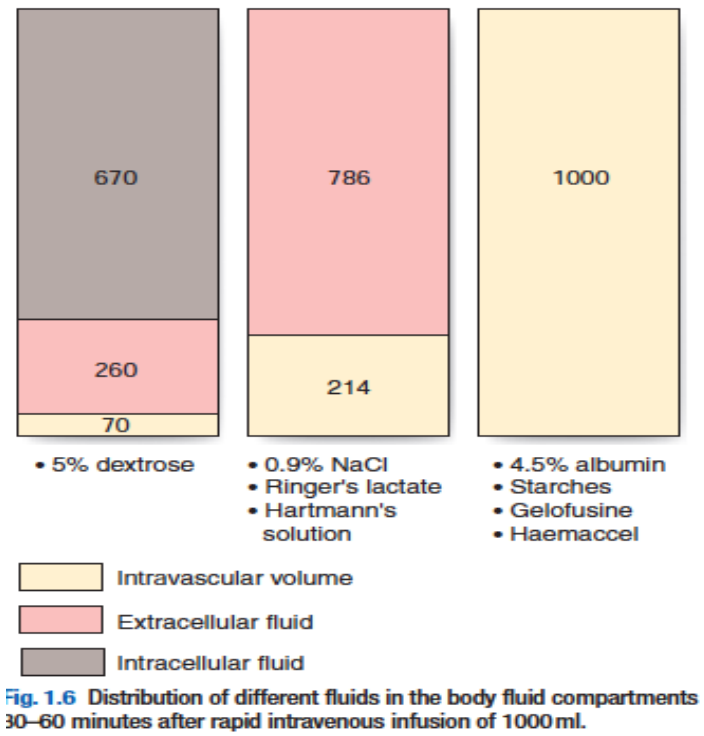


Fig. 1.6 Distribution of different fluids in the body fluid compartments 30–60 minutes after rapid intravenous infusion of 1000 ml.

Table 1.10 Composition of commonly administered intravenous fluids

	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	HCO_3^- (mmol/l)	Ca^{2+} (mmol/l)	Mg^{2+} (mmol/l)	Oncotic pressure (mmH_2O)	Typical plasma half-life	pH
5% dextrose	–	–	–	–	–		0	–	4.0
0.9% NaCl	154	0	154	0	0		0	–	5.0
Ringer's lactate (Hartmann's solution)	131	5	112	29*	1	1	0	–	6.5
Haemaccel (succinylated gelatin)	145	5.1	145	0	6.25		370	5 hours	7.4
Gelofusine (polygeline gelatin)	154	0.4	125	0	0.4	0.4	465	4 hours	7.4
Hetastarch	154	0	154	0	0		310	17 days	5.5
Human albumin solution 4.5% (HAS)	150	0	120	0	0		275	–	7.4

*The lactate present in Ringer's lactate solution is rapidly metabolized in the liver. This generates bicarbonate ions. Bicarbonate cannot be directly added to the solutions because it is unstable (tends to precipitate).

memories it (specially Dextrose, albumin and NS and Ringers)

IV Crystalloids dose:

Maintenance dose: By using 4/2/1 rule

- 1st 10 Kg: 40 ml/hr
- 2nd 10 Kg: 20 ml/hr
- Then each additional 10 Kg: 10 ml/hr

Example:

Male 70 Kg=

40 + 20 + 50 = 110 ml/hr

Electrolytes Imbalance

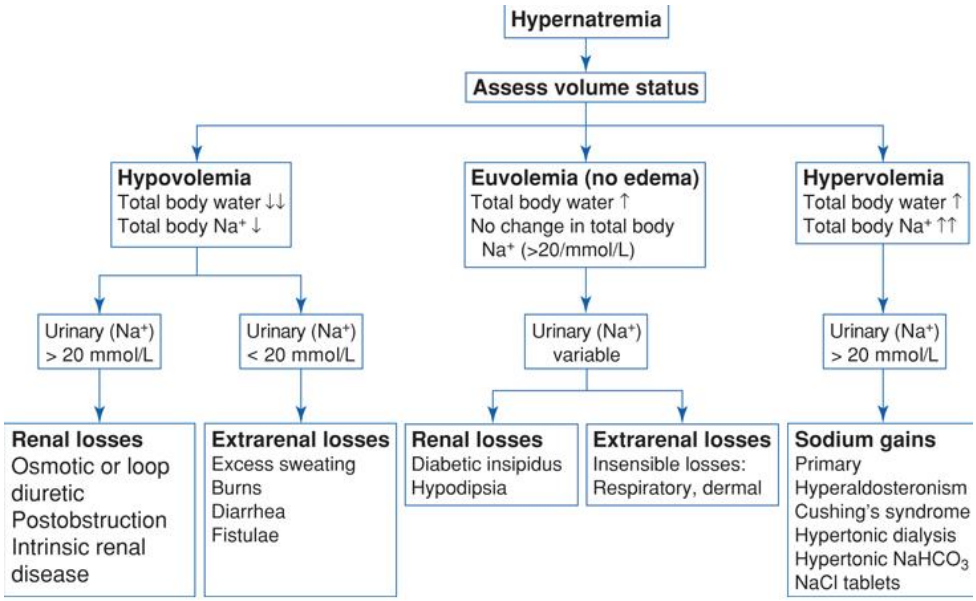
Hypernatremia: (Na > 145 mEq/L)

- **Always remember: Sodium is Brain**
- Sodium is high either due to high intake or low body water

➢ Clinical manifestation of Hypernatremia:

- Lethargy, thirst
- Neurological dysfunction (due to shrinkage of brain cells, as fluid shift from cells to blood because the blood has low osmolarity due to the hypernatremia)

➢ Causes:



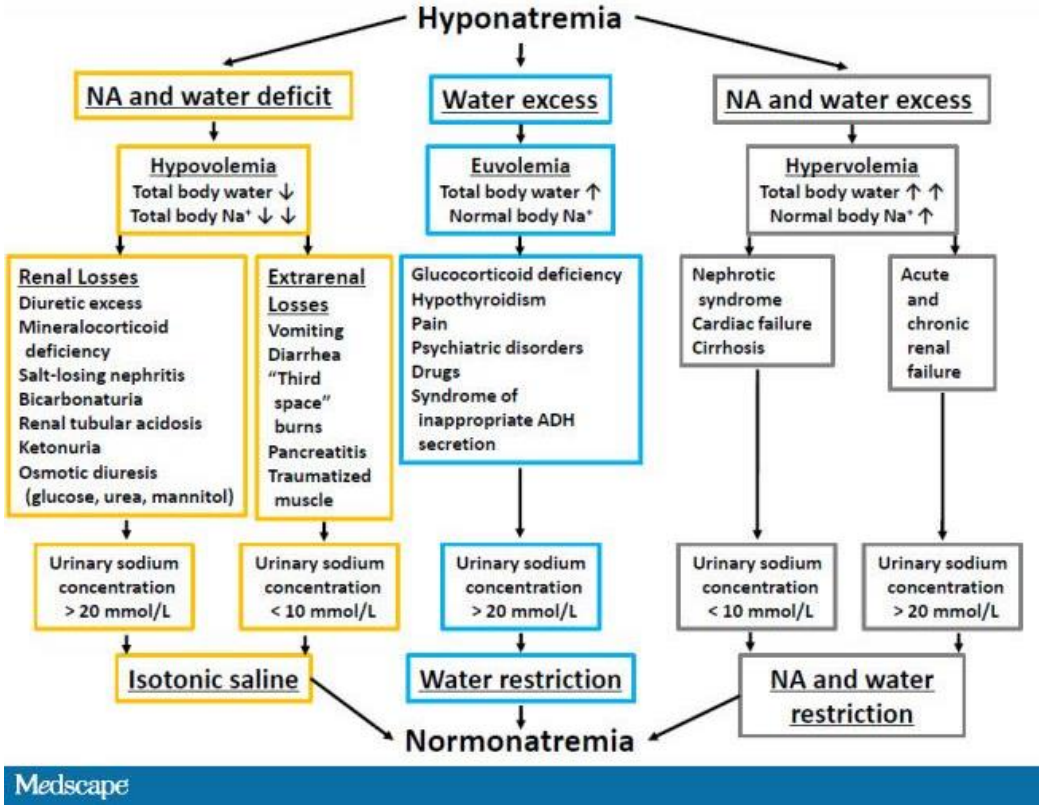
➢ Treatment:

- Isotonic salt-free IV fluid (e.g. Dextrose 5%)
- Oral solutions preferable (in stable patient)
- **Correction of sodium level should be slow, not more than 0.5 mEq/L per hour (to avoid cerebral edema)**

(As written in book, we either give Normal saline because these patient mostly dehydrated then shift to 5% Dextrose because we don't want to increase sodium, or from the beginning start with hypotonic solution like ½ or ¼ normal saline. **Both cases correction of sodium always slow**)

Hyponatremia: (Na <135 mEq/L)

➢ Causes:



➢ Clinical manifestation of Hyponatremia:

- Wide range from asymptomatic to severe neurological symptoms.
- **Neurological symptoms**
  - Lethargy, headache, confusion, apprehension, depressed reflexes, seizures and coma (All due to cerebral edema, as fluid shifts to brain cells due to their higher osmolarity than the blood)
- Muscle symptoms: Cramps, weakness, fatigue
- GI symptoms: Nausea, vomiting, and diarrhea

➢ Treatment:

- Depends on the volume status and etiology (if patient is hypovolemic we give him isotonic solution like NS. If euvolemic we restrict water and if hypervolemic we restrict Na and water)
- Discontinue any offending meds.
- **Slow correction,** don't increase Na more than 0.5 mEq/L per hour to avoid **Osmotic Demyelinating Syndrome**

- **(We never aggressively treat hyponatremic patient with hypertonic solution to increase Na unless he is unstable with severe neurological symptoms like seizure or coma due to the cerebral edema. Because increasing Na quickly *more than 0.5 mEq/L/hr* going to lead to irreversible neurological symptoms due to demyelination of central pontine neurons including dysarthria, dysphagia, mutism, paralysis, locked-in syndrome ☹ and brain death)**

(So, quick correction of Na from low to high= Osmotic demyelinating syndrome. And from high to low= Cerebral edema)

Hyperkalemia: (K > 5.5 mEq/L)

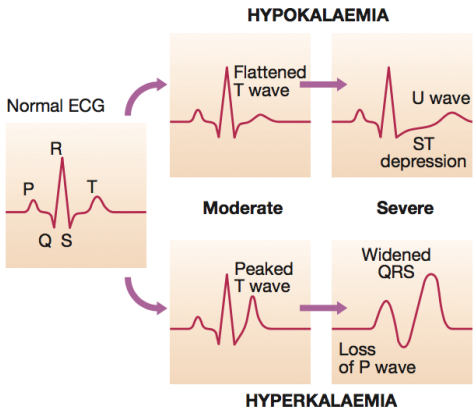
➢ Causes:

- Renal disease
- Massive cellular trauma
- Insulin deficiency
- Addison's disease
- Potassium sparing diuretics (e.g. spironolactone)
- Decreased blood pH
- Exercise causes K+ to move out of cells

(High WBCs, High PLTs, Hemolysis of blood sample and sample taken distal to tourniquet give false high potassium *pseudo-hyperkalemia*)

➢ Clinical manifestation

- **Always remember: Potassium is Heart**
- Early – hyperactive muscles , paresthesia
- Late - Muscle weakness, flaccid paralysis
- **Change in ECG pattern** (peaked T wave, PR prolongation, loss of p wave, widening QRS complex *in severe cases*)
- **Dysrhythmias (V. tach and V. fib)**
- **Bradycardia , heart block, cardiac arrest**



➢ Treatment:

- Decrease intake and increase renal excretion

- Cardiac membrane stabilizer (**calcium gluconate** or calcium chloride) to prevent cardiac arrhythmia and cardiac arrest (in case going to use Ca chloride, should be through central vein because it is sclerotic)
    - **10 ml of 10% calcium gluconate in 10 mins (Rule of 10)**
- Shifting K+ into cells:
    - Insulin + glucose (10 units of short acting insulin + 50 ml 50% Dextrose)
    - HCO3
    - B-agonists (high dose of salbutamol)

Table 1.13 Management of severe hyperkalaemia (K^+ >7 mmol/l)

1. Identify and treat cause. Monitor ECG until potassium concentration controlled.	
2. 10 ml 10% calcium gluconate iv over 3 mins, repeated after 5 min if no response	Antagonizes the membrane actions of ↑ K^+ reducing the risk of ventricular arrhythmias
3. 50 ml 50% dextrose + 10 units short-acting insulin over 2–3 mins. Start infusion of 10–20% dextrose at 50–100 ml/h	Increases transcellular shift of K^+ of into cells
4. Regular salbutamol nebulizers	Increases transcellular shift of K^+ of into cells
5. Consider oral or rectal calcium resonium (ion exchange resin)	Facilitates K^+ clearance across gastrointestinal mucosa. More effective in non-acute cases of hyperkalaemia
6. Renal replacement therapy	Haemodialysis is the most effective medical intervention to lower K^+ rapidly

Hypokalemia: (K < 3.5 mEq/L)

- Not as dangerous as hyperkalemia
- be aware of diabetes (as insulin shift K into cells)

➢ Causes:

- Decreased intake of K^+
- Increased K^+ loss
    - Chronic diuretics
    - Acid/base imbalance (alkalosis cause hypokalemia, as cells uptake potassium in exchange with H+ to decrease pH)
    - Trauma and stress
    - Increased aldosterone
    - Insulin

➢ Clinical manifestation:

- Neuromuscular disorders
  - Weakness, flaccid paralysis, respiratory arrest, constipation
  - **Dysrhythmias** (ECG: flattened T wave, appearance of U wave)
  - Postural hypotension
  - Cardiac arrest

➢ Treatment:

- K^+ intake, but slowly, preferably orally (unless patient unstable)
- Must correct hypomagnesemia (then K will be corrected)

Calcium Imbalance:

- Most in ECF
- Regulated by:
  - Parathyroid hormone
    - ↑Blood Ca^{++} by stimulating osteoclasts
    - ↑GI absorption and renal retention
  - Calcitonin from the thyroid gland
    - Promotes bone formation
    - ↑ renal excretion

Hypercalcemia:

➢ Causes:

- **Hyperparathyroidism** (Most common in young)
- **Malignant tumors** (Most common in old)
- Hypothyroid states
- Renal disease
- Excessive intake of vitamin D
- Milk-alkali syndrome
- Certain drugs

➢ Clinical manifestation:

To remember: Stones, Bones, Abdominal Groans, Psychic Moans

- **Nonspecific** – fatigue, weakness, lethargy
- Increases formation of kidney stones and pancreatic stones
- Pain, Muscle cramps
- Depression, hellocinations
- Bradycardia, cardiac arrest
- Abdominal pain, GI upset also common
- Metastatic calcification

Hypocalcemia:

- Hyperactive neuromuscular reflexes and muscle spasm differentiate it from hypercalcemia
- Convulsions in severe cases

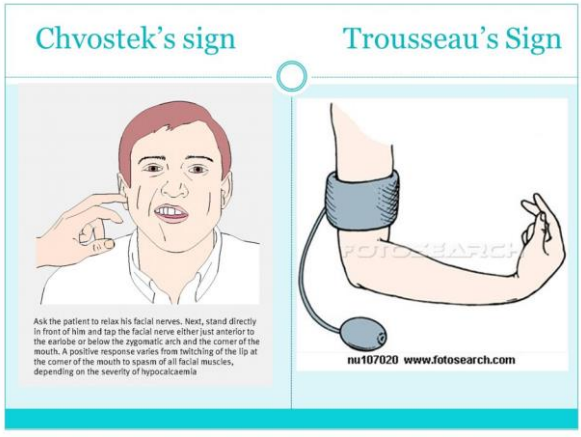
➢ Causes:

- Renal failure
- Vitamin D deficiency or hypomagnesemia
- **Post – thyroidectomy or irradiation of neck**
- Infiltrative or autoimmune disease
- Suppression of parathyroid function
- Hypersecretion of calcitonin
- Malabsorption states
- Abnormal intestinal acidity and acid/ base balance.
- Widespread infection or peritoneal inflammation **(Acute pancreatitis cause hypocalcemia due to soapification)**

➢ signs:

- Chvostek's sign
- Trousseau's sign

(They also have hyperreflexia. Sometime they develop laryngospasm which is serious)



➢ Treatment:

- IV calcium for acute
- Oral calcium and vitamin D for chronic

Hypomagnesemia: (common in surgical patients)

(hypomagnesemia is the most common. Hypermagnesemia only common as a complication of magnesium given in preeclampsia)

➢ Clinical manifestation:

- muscle weakness, altered mentation, tremors, hyper-reflexia, seizure.
- Tachyarrhythmias (long QT interval leading to **Torsades de pointes**, atrial fibrillation)

➢ Treatment:

- IV Magnesium

Acid-Base Balance

- $H2O \Leftrightarrow H^+ + OH^-$ (lead to steady state: pH 7) Henderson-Hasselbach Equation
- **Source of H^+:** intake, metabolism, CO
- **Elimination of H^+: kidney**, lung (CO_2), gastrointestinal tract, liver

Buffers:

- A buffer is a substance, that has the ability to bind or release H^+ in solution, thus keeping the pH of the solution relatevily constant despite of considerable quantities of acid or base
- The important buffer systems in blood include proteins, carbonic acid-bicarbonate buffers.

Buffer	pK	Concentration	Buffer capacity
Bicarbonate	6,1	24 mmol/l	75 %
Hgb	8,25	24 mmol/l	25 %
oxyHgb	6,95		
Proteins	-		
Phosphates	6,8		

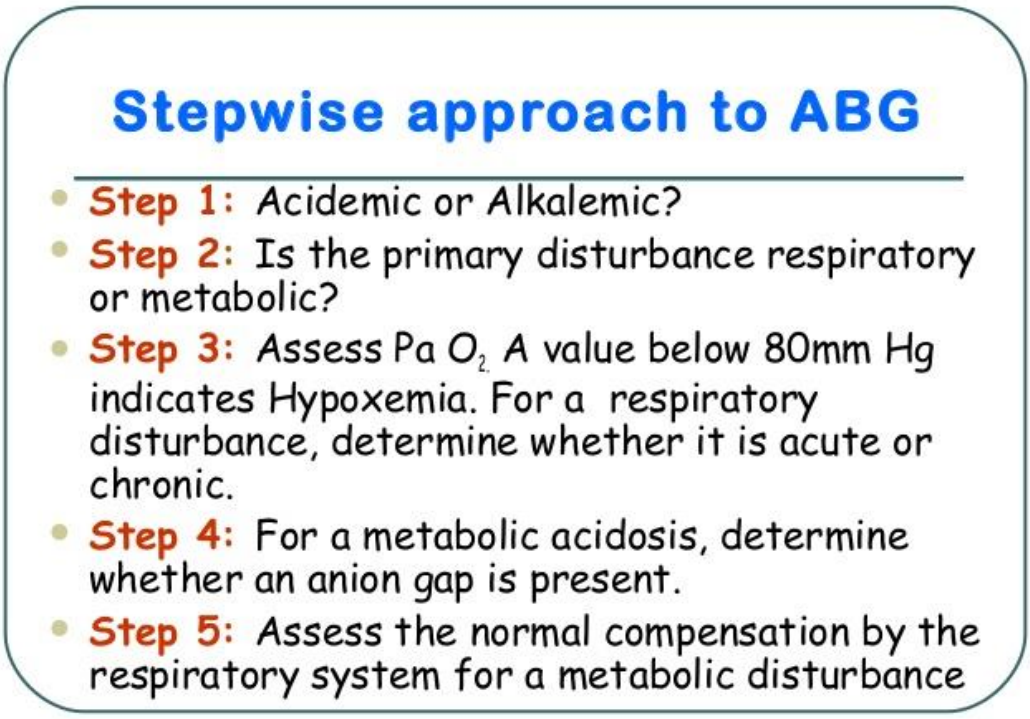
So bicarbonate has the largest capacity. Hb is also very strong buffer

Acid-base disorders:

- Normal pH: 7.35-7.45
- More than 7.45 is alkalosis, and less than 7.35 is acidosis (acidemia is whenever pH itself is less than 7.35, and acidosis is the process of decreasing pH. So patient could have a pH of 7.46, he has alkalalemia, but before that it was 7.50 so we know the pH is going down so he has acidosis)
- Metabolic disturbance: addition of acid or alkali (stronger than the buffers) or removal of acid or alkali
- Respiratory disturbance: rise or decline in arterial pCO_2
- Compensation by:
  - Kidneys (HCO3)
  - Lung (CO2)

Arterial blood gases:

- pH 7.35-7.45
- pCO2 Hgmm (kPa) 35-45
- bicarbonate (mmol/l) 21-29
- Anion gap (mmol/l) 10-14



Doctor said the 5th step about measuring the compensation is not required from us

For explanation, the easiest way to analyze ABG is as following:

Step 1: Analyze the pH

The first step in analyzing ABGs is to look at the pH. Normal blood pH is 7.4, plus or minus 0.05, forming the range 7.35 to 7.45. If blood pH falls below 7.35 it is acidic. If blood pH rises above 7.45, it is alkalotic. If it falls into the normal range, label what side of 7.4 it falls on. Lower than 7.4 is normal/acidic, higher than 7.4 is normal/alkalotic. Label it.

Step2: Analyze the CO2

The second step is to examine the pCO2. Normal pCO2 levels are 35-45mmHg. Below 35 is alkalotic, above 45 is acidic. Label it.

Step 3: Analyze the HCO3

The third step is to look at the HCO3 level. A normal HCO3 level is 22-26 mEq/L. If the HCO3 is below 22, the patient is acidotic. If the HCO3 is above 26, the patient is alkalotic. Label it.

Step 4: Match the CO2 or the HCO3 with the pH

Next match either the pCO2 or the HCO3 with the pH to determine the acid-base disorder. For example, if the pH is acidotic, and the CO2 is acidotic, then the acid-base disturbance is being caused by the respiratory system. Therefore, we call it a respiratory acidosis. However, if the pH is alkalotic and the HCO3 is alkalotic, the acid- base disturbance is being caused by the metabolic (or renal) system. Therefore, it will be a metabolic alkalosis.

Step 5: Does the CO2 or HCO3 go the opposite direction of the pH?

Fifth, does either the CO2 or HCO3 go in the opposite direction of the pH? If so, there is compensation by that system. For example, the pH is acidotic, the CO2 is acidotic, and the HCO3 is alkalotic. The CO2 matches the pH making the primary acid-base disorder respiratory acidosis. The HCO3 is opposite of the pH and would be evidence of compensation from the metabolic system.

Step 6: Analyze the pO2 and the O2 saturation. Finally, evaluate the PaO2 and O2 sat. If they are below normal there is evidence of hypoxemia.

Anion gap:

Anion Gap Metabolic Acidosis

MUDPILES anion gap = Na – [Cl⁻ + HCO_3^-]

Methanol
Uremia
Diabetic ketoacidosis
Paraldehyde
Iron, isoniazid
Lactic acidosis
Ethylene glycol
Salicylates

In case it is metabolic acidosis:
Anion Gap (AG) = (Na^+) - (Cl^-+HCO_3^-)

- **Elevated anion gap:** lactic acidosis, ketoacidosis, renal failure, exogenous administration of acids
- **Non-anion gap acidosis**: Renal Tubular Acidosis, Vomiting, Iatrogenic acidosis (administration of Cl)