

الطب والجراحة لجنة

Fluid and Electrolytes

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DISTRIBUTION OF FLUIDS:

• Fluids = **60%** of total body Weight

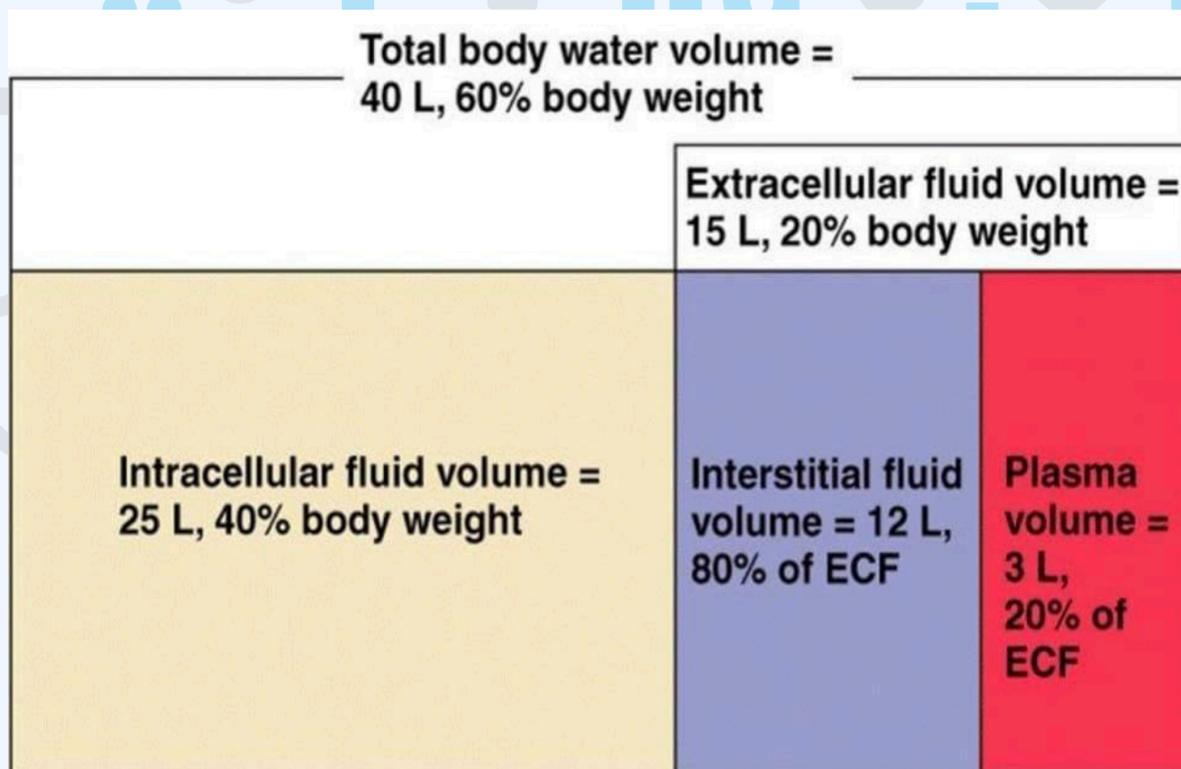
• Two major compartments >>

• Intracellular **2/3rd** , Extra-cellular **1/3rd** .

• Extra-cellular >>

interstitial fluid (in between cells) **3/4th**

intravascular fluid (plasma) **1/4th**



COMPOSITION & PROTEIN

• EXTRA CELLULAR FLUID [I.V.+inters.] HAVE THE **SAME** COMPOSITION

BUT

• INTRAVASCULAR PROTEIN [**MAINLY ALBUMIN**] CONCENTRATION IS **MORE** THAN THE INTERSTITIAL ONE and it is the **most effective intravascular osmotic pressure**. because it has the strongest power to control fluidity

Na IONS [ELECTRICITY] ACCOUNT FOR **MOST** THE **OSMOLALITY** IN THE **EXTRACELLULAR** FLUID

• WHEREVER SODIUM GOES WATER GOES WITH IT PROPORTIONALLY

Osmolarity:

- $\text{Posm}(\text{mOsm}/\text{kg}) = 2 \times \text{Na}^+ + [\text{glucose}]/18 + \text{BUN}(\text{urea}) / 2.8$ (**important**)
- The normal osmolality of plasma ranges from **275** to **290** mOsm/kg.

Osmolality: is concentration of substance in osmoles in one Kg of solvent

Osmolarity :is concentration of substance in osmoles in one liter of solvent

Fluid requirements/ 24 hours :

- Water : 30-35 ml/kg
- K : 1 mEq/kg
- Na : 1-2 mEq/kg
- Cl : 1.5 mEq/kg

Normal water loss:Normal: Input of fluid=Output ;Normal sites of water loss:

- Urine 1200-1500 ml/24h
- Sweat : 200 ml
- Respiratory losses: 500-700 ml
- Feces :100-200 ml

• **Insensible fluid losses : loss of fluid that is not directly measured ;**

E.g: direct contact, vibration

Daily secretions : Most of them are intraabdominal

- Bile :1 L/24 h
- Gastric : 2L/24 h
- Pancreatic : 600 ML/24 h
- Small intestine : 3 L/24 h
- Saliva : 1500 ml/24 h

• **Most secretions are reabsorbed**

GI ELECTROLYTE LOSSES:

- Sweat - hypotonic (Na concentration 35-65)
- Saliva - K^+ (highest concentration of K^+ in body)
- Stomach - H^+ and Cl
- Pancreas - HCO_3^- : secrete Bicarbonate ↑, so; in the case of patient who had pancreatic fistula, he will lose bicarbonate more than other electrolytes
- Bile - HCO_3^-
- Small intestine - HCO_3^- , K^+ ; in cases of diarrhea or fistula in bowel; will lose K^+ , so hypokalemia occurs
- Large intestine - K^+
- Dialysis can remove K, Ca, Mg, PO, urea, and creatinine

Third spacing ! : interstitial space collection of fluid

•Fluid accumulation in the interstitial of tissue (lumen of the small bowel in case of IO, Pancreatitis , PERITONITIS , Post surgery).

•**INTRVASCULAR , INTRACELLULAR SPACES** as the first two spaces. (1st and 2nd spaces)

•POSTOPERATIVE !

•Around post op **day 3** , Fluids return back to intravascular space so be aware of fluid (لأن الجسم لم يعد بحاجة هذه الكمية من السوائل بعد أول يومين)

INPUT = OUTPUT = EUVOLEMIA

•Assessment of fluid status :

•Skin turgor , mucus membranes

•Vital signs

•Weight changes

•**Urine output : minimum adult = 0.5 ml/kg**

•Jugular vein distention , LL Edema

•Crackles

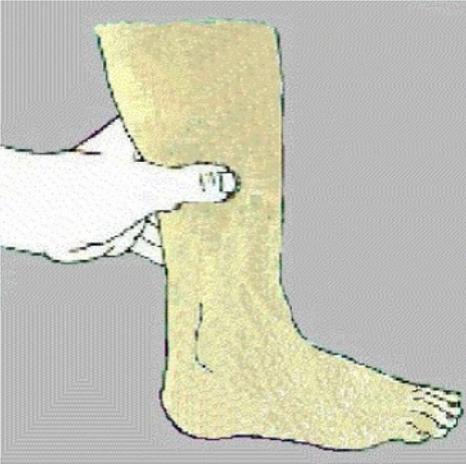
•CVP (central venous pressure) , PCWP (pulmonary capillary wedge pressure)

•Lactic acid

•CXR findings to determine if he was overloaded / not

Those tests are done to determine if the patient is overloaded, Hypervolemic / Not

LEG EDEMA TESTING



CHEST X-RAY:

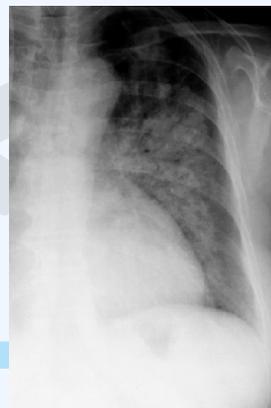
NORMAL:



NECK VEINS DISTENTION



PULMONARY OEDEMA:



Supplied by over fluid ,so fluid accumulates in the pleural space

Electrolyte Imbalance:

SODIUM (135-145 mEq/L)

- Plasma sodium levels are directly affected by fluid balance.
- Hyponatremia results from excess water intake that is not excreted, whereas hypernatremia results from an excessive loss of free water.
- Hyponatremia is defined as a sodium level less than 135 mEq/L
 - mild, 130-134 mEq/L
 - moderate, 120-129 mEq/L
 - severe, <120 meq/L.

HYPONATREMIA

- 1- measure serum osmolality
- Hypo / hyper / iso-tonic

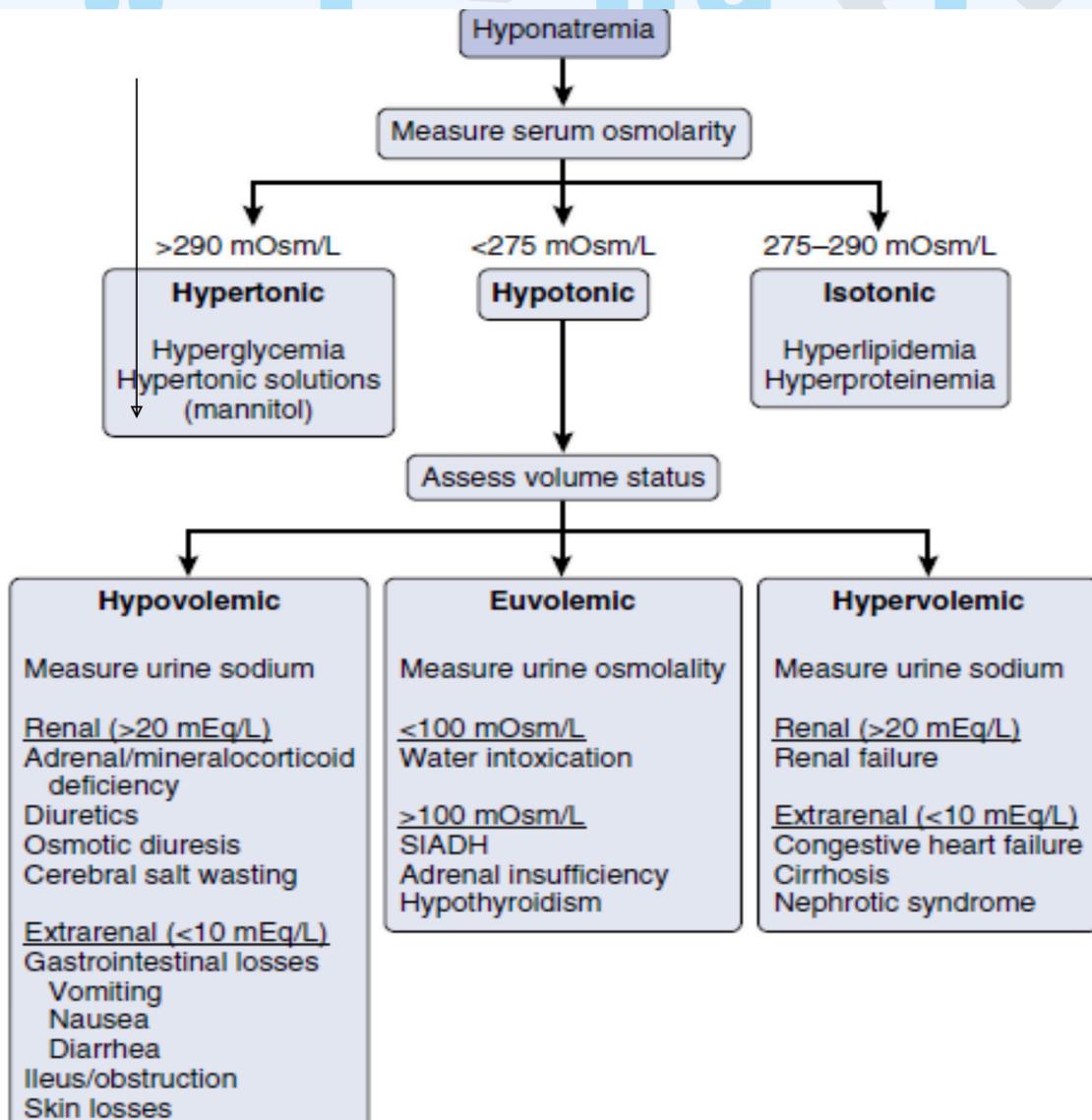
•Hypotonic hyponatremia

•**HYPO-VOLEMIC** (the patient loses a lot of fluid) : NG suction , burns , pancreatitis , diaphoresis >> IV NS & Correct underlying cause

•**EU-VOLEMIC** : SIADH , **CNS** >>> fluid restriction

•**HYPER-VOLEMIC** (the patient has excessive fluid) : RF, CHF , LIVER FAILURE , DILUTIONAL , FLUID **OVERLOAD** >> **fluid restriction and diuretics.**

Important

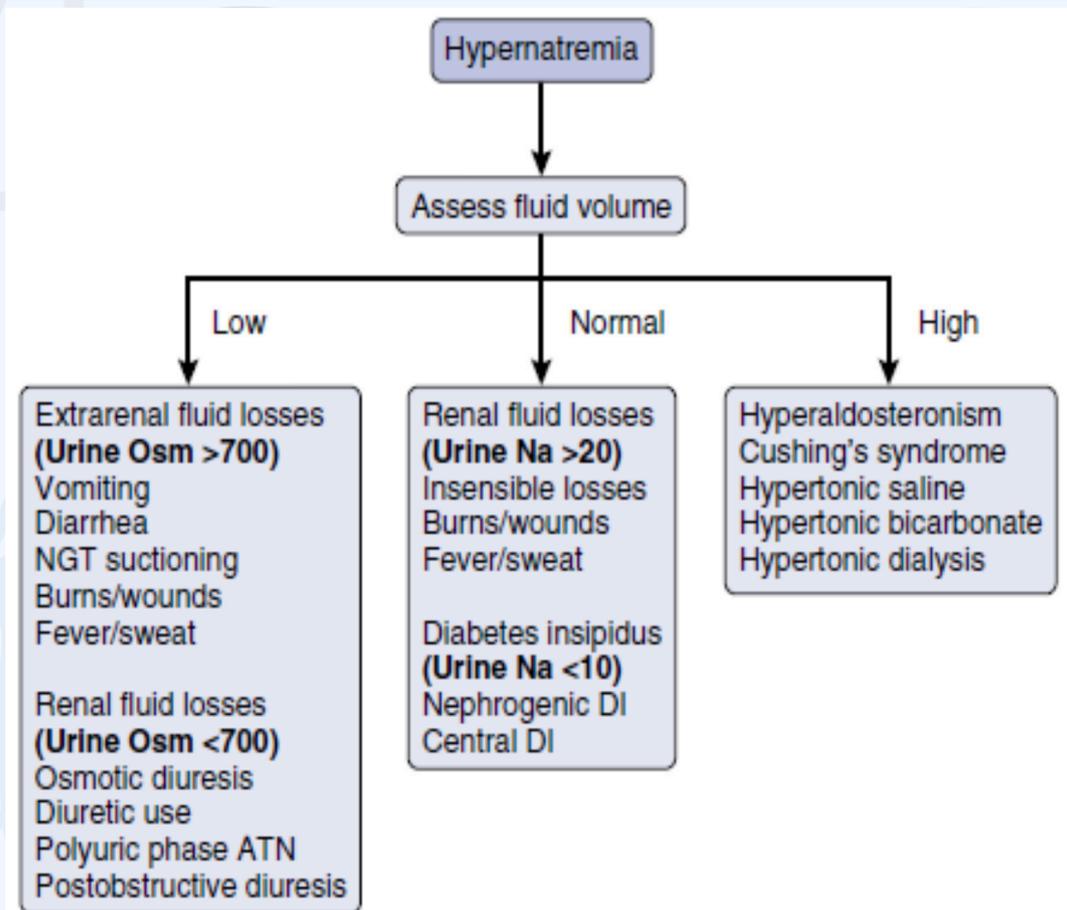


HYPONATREMIA

- Clinically : seizures , confusion lethargy , coma weakness
- Always don't forget to correct hyponatremiaslowly (0.25–0.5 mEq/L per hour) / (12 meq/24h) , **to avoid central pontine myelinolysis.**

HYPERNATREMIA

- Hypernatremia is defined as a sodium level greater than 145mEq/L
- (moderate, 146–159 mEq/L; severe, ≥ 160 Eq/L).
- Causes :
 - In surgical pt. its mostly due to volume depletions (inadequate fluid resuscitation)with inadequate hydration , others : DI Vomiting diarrhea , diuretics
- Clinically :
 - seizures ,confusion, tremors .respiratory paralysis.
- **Slow lowering of serum sodium is very important less than 12 mEq/day TO AVOID SEZIURES . (brain edema)**
- Treatment by hypotonic fluids D5W. (Water and sugar glucose), $\frac{1}{2}$ NS , $\frac{1}{4}$ NS



POTASSIUM [NORMAL SERUM :K 3.5-5 mmol/L]

Cell

- 98% INTRACELLULAR
- 2 % EXTRACELLULAR

H⁺ →

K⁺ ←

- 10% LOSS OF TOTAL BODY K⁺ DROPS SERUM LEVEL FROM 4 to 3 meq
- RENAL EXCRETION OF K IS REGULATED BY **ALDOSTERONE** [Mineralocorticoid]
- **RENAL FAILURE** FAILS TO EXCRETE K, THIS LEADS TO HYPERKALAEMIA
- THERE IS ALWAYS EXCHANGE OF K FOR **HYDROGEN IONS** IN PH CHANGES TO CORRECT THE PH. (تقاس من خارج الخلية K⁺)
- **HYPERKALEMIA LEADS TO ACIDOSIS.** (خرج K⁺ ، دخل الخلية H⁺ ، hyperkalemia (outside) , Acidosis لأن H⁺ زاد تركيزها إذا دخلت الخلية إذا تركيزها زاد H⁺ لأن H⁺ داخل الخلية (inside))
- **ACIDOSIS LEADS TO HYPERKALEMIA**
 - Alkalosis leads to Hypokalemia

Hyperkalaemia due to acidosis

IN ACIDOSIS [H] IONS GO INTO THE CELL TRYING TO REDUCE THE ACIDOSIS .

[K] COMES OUT OF THE CELL CAUSING HYPERKALEMIA WHICH CAN BE LETHAL

HYPOKALAEMIA



- IS THE COMMONEST IN THE SURGICAL WARD PARALYTIC ILEUS (No peristalsis in the Bowel (ileus) causes constipation, occurs postoperatively due to not compensating the hypokalemia)

HYPOKALAEMIA

LOW SERUM POTASSIUM

LOSS THROUGH 1- GIT, 2- RENAL OR INTO THE 3- CELL

☒ 1-GIT.

A-[LOW INTAKE]

B-[HIGH LOSS; VOMIT. N/GTUBE, DIARRHOEA]

☒ 2-RENAL

LOSS BY A-DIURETICS

B-INCREASED ALDOSTERONE ACTIVITY.

☒ 3-ALKALOSIS;

WHERE SERUM POTASSIUM GOES INTO THE CELLS TO REPLACE THE OUTGOING H

IONS IN AN ATTEMPT TO CORRECT THE ALKALOSIS.e.g .; gastric outlet obstruction

☒ 4-INSULIN THERAPY; K goes into the cell ; DM

EFFECT OF HYPOKALAEMIA:

- 1- Decrease G.I. CONTRACTILITY [PARALYTIC ILEUS]
- 2- Decrease RESPIRATORY MUSCLES CONTRACTILITY
- 3- CARDIAC ARRHYTHMIA

ECG Changes in Hypokalemia :

Falt T waves , U waves , ST depression , PAC (premature atrial contraction) PVC (premature ventricular contraction) ,AF (atrial fibrillation)

TREATMENT OF HYPOKALAEMIA

Treat the underlying cause if possible

☒ **REPLACEMENT:-**

KCL IV causes skin irritation And cardiac arrest if given quickly

☒ MAX RATE THROGH :

☒ **PERIPHERAL IV LINE** : 10 mEq/hour

☒ **CENTRAL LINE** : 20 mEq/ hour

BE CAREFUL OF HYPERKALAEMIA >>RAPID CORRECTION >>> CARDIAC ARREST IN DIASTOLE ,, So hypokalemia treated slowly

PERSISTENT HYPOKALAEMIA

HYPOKALAEMIA INSPITE OF REPLACEMENT THERAPY MEANS COEXISTENT MAGNESIUM DEFICIENCY

**NB:REFRACTORY HYPOKALAEMIA & HYPOCALCAEMIA
COULD BE DUE TO Mg DEFICIT**

HYPERKALAEMIA

**IN BRIEF;
ACIDOSIS**

**DESTRUCTION OF CELLS
ALDOSTERONE SHUTDOWN**

- SEVERE TRAUMA :causes ACIDOSIS & DISTRUCTION OF CELLS
- BURNS :causes ACIDOSIS & DISTRUCTION OF CELLS
- CRUSH INJURY: causes ACIDOSIS & DISTRUCTION OF CELLS
- SEVERE CATABOLIC STATE [SEPSIS] : causes ACIDOSIS
- RENAL FAILURE : causes ACIDOSIS
- ADDISON`S DISEASE : causes ALDOSTERONE SHUTDOWN
- BLOOD TRANSFUION :causes ACIDOSIS & DISTRUCTION OF CELLS

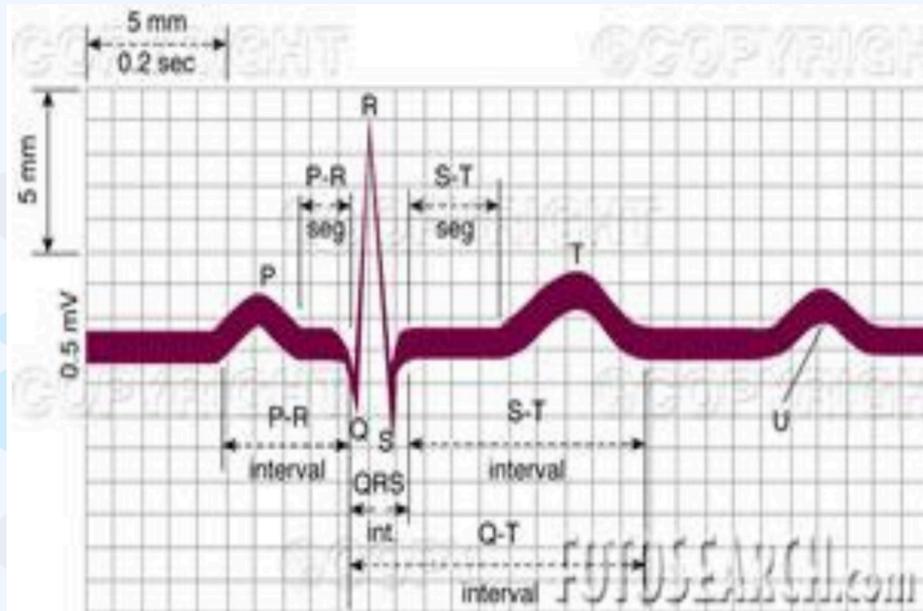
OTHER CAUSES

- 7-ACIDOSIS
- 8-LEUCOCYTOSIS [MARKED]
- 9-THROMBOCYTOSIS [ABOVE ONE MILLION]
- 10-HAEMOLYSIS ; لما عينة دم تُترك في الخارج ; RBCs destructed then K⁺ gets out causes FALSE HYPERKALEMIA
- 11-HAEMOLYSED SPECIMEN

HYPERKALAEMIA

- Clinically :
- Decreased DTR (deep tendon reflexes)
- Weakness
- Parasthesia
- Paralysis
- Respiratory failure

NORMAL ECG



ECG CHANGES in hyperkalemia :

critical value > 6.5.

- Peaked T wave.
- Depressed ST segment
- Prolonged PR
- Wide QRS
- Bradycardia
- V.Fib

ECG CHANGES in hypokalemia:

U wave

Atrial fibrillation

EMERGENCY TREATMENT OF HYPERKALAEMIA:

- **CALCIUM GLUCONATE I.V.** REVERSES THE ACTION OF [K] ON THE HEART (imp)
- 10 UNITS OF REGULAR **INSULIN** IN 100 ML OF 50% DEXTROSE I.V. INSULIN PUSHES [K] INTO THE CELL
- **SOD.BICARB.** CORRECTS ACIDOSIS BUT THIS IS CONTROVERSIAL

SLOW CORRECTION OF HYPERKALAEMIA:

- ORAL CATION EXCHANGE RESIN [SODIUM POLYSTYRENE SULFONATE] **Kayexalate** , TAKES POTASSIUM IN EXCHANGE FOR SODIUM AND GETS RID OF [K] IN THE STOOL
- HYPERKALAEMIA due to **RENAL FAILURE** NEEDS **DIALYSIS**

CALCIUM : NORMAL SERUM Ca .[8.5-10.3mgm/100ml] [2.2-2.5 mmol/l]

- MOST OF IT STORED IN BONE
 - DAILY INTAKE; 1-3 gm
 - MOST OF THE INTAKE IS NOT ABSORBED
 - **CONTROLLED BY VIT. D, PARATHYROID, CALCITONIN**
 - CALCIUM IONS [40% OF THE SERUM CALCIUM] IS NECESSARY FOR NEUROMUSCULAR , ENZYMATIC FUNCTION AND BLOOD COAGULATION [IONIZED] .
 - [50% IS ATTACHED TO ALBUMIN-NOT IONIZED]
 - [IF ALBUMIN IS LOW ; CALCIUM WILL BE LOW]
 - SERUM LEVEL DOES NOT NECESSARILY INDICATE THE LEVEL OF IONIZED [FUNCTIONING] CALCIUM
 - ACIDAEMIA INCREASES IONIZED CALCIUM
 - ALKALAEMIA DECREASES IONIZED CALCIUM
 - NB: Ca absorption needs vit.D. which is activated in the kidney, that is why in renal failure Ca drops. Low Ca leads to hyperparathyroidism. So in renal failure , you may come across low or high Ca level (**Negative feedback**)
- Ca²⁺ : ionized, non-ionized, free

CALCIUM AND ALBUMIN

IF SERUM ALBUMIN DROPS,
SERUM CALCIUM WILL DROP TOO, BECAUSE CALCIUM IS ATTACHED TO ALBUMIN

HYPOCALCEMIA < 1.8 mmol/L

THE COMMONEST; AFTER THYROID SURGERY , RENAL FAILURE AND PANCREATITIS

1-HYPOPARATHYROIDISM

2-HYPOMAGNESEMIA [PATIENTS ON IV.FEEDING FOR LONG TIME]

3-SEVERE PANCREATITIS

4-RENAL FAILURE; ACUTE AND CHRONIC

Ca ABSORPTION IS HELPED BY ACTIVE FORM OF VIT.D WHICH IS DONE IN THE KIDNEY. IN RENAL FAILURE THAT DOES NOT HAPPEN.HOWEVER LOW Ca CAN LEAD TO HYPERPARATHYROIDISM CAUSING HYPERCALCEMIA WITH BONE CHANGES

5-SEVERE TRAUMA [BLOOD LOSS] [ALBUMIN LOSS]

6-MASSIVE BLOOD TRASFUSION

7-CRUSH INJURY [renal failure]

8-NECROTIZING FASCIITIS : infection under the skin

9-RHABDOMMYOLYSIS

ALL SOFT TISSUE INJURY CAN CAUSE HYPOCALCEMIA

HYPOCALCEMIA

CLINICAL MANEFESTATION; **MOST COMMON:** postoperative thyroid surgery

1-CIRCUMORAL NUMBNESS,TINGLING TIPS OF FINGERS AND TOES

2-NEUROMUCULAR HYPERACTIVITY

A-EXAGERATED DEEP REFLEXES

B-POSITIVE CHVOSTEK SIGN

C-CARPOPEDAL SPASM

D-MUSCLE CRAMPS

E-ABDOMINAL CRAMPS

F-CONVULSIONS [RARE]

Carpopedal Spasm





When we measure BP in the arm and we apply the cuff around the circumference of the arm, the arteries closed after raising the pressure then this contraction occurs, that called Carpopedal spasm

ASSESSMENT TIP

Eliciting Chvostek's sign

Begin by telling the patient to relax his facial muscles. Then stand directly in front of him, and **tap the facial nerve** either just anterior to the earlobe and below the zygomatic arch or between the zygomatic arch and the corner of his mouth. A positive response varies from **twitching** of the lip at the corner of the mouth to spasm of all **facial muscles**, depending on the severity of hypocalcemia.



HYPO-CALCAEMIA MANAGEMENT

ACUTE

1-CHECK FIRST BLOOD [PH]

2-ALKALOSIS SHOULD BE TREATED [AS IT REDUCES IONIZED Ca)

3-I.V CALCIUM [Ca GLUCONATE OR CHLORIDE] IN REAL HYPOCALCEMIA

CHRONIC : CHECK Mg level , CHECK ALBUMIN

1. ORAL CALCIUM +

2. VIT.D[1-ALFA-CHOLE-CALCIFEROL]

3. ALUM.HYDROXIDE TO BIND DIETARY PHOSPHATE

HYPERCALCEMIA

1. HYPERPARATHYROIDISM

[Adenoma 90%, Hyperplasia 9% , carcinoma<1%]

2-BONE METASTASIS OF CERTAIN CANCER [OSTEOCLASTIC]; **BREAST** [The **commonest cause**], PROSTATE, BRONCHUS, KIDNEY AND THYROID]

3-NEOPLASM WITH ENDOCRINE SECRETION ; BRONCHUS,KIDNEY AND OVARY

[1+2+3 account for 90% of hypercalcemia]

4-VIT.D INTOXICATION

5-SARCOIDOSIS

6-HYPERTHYROIDISM

7-PROLONGED IMMOBILIZATION

8-MILK ALKALI SYNDROME[Ca + milk used to be ttt of p.u]

HYPERCALCEMIA CLINICAL PICTURE

NEUROMUSCULAR LOW ACTIVITY

- EASY FATIGABILITY
- MUSCLE WEAKNESS
- CONSTIPATION
- ANOREXIA & VOMITING
- DEPRESSION
- POLYUREA THEN OLIGUREA DUE TO DEHYDRATION
- POLYDIPSIA AND VOMITING
- CALCINOSIS [ANYWHERE BUT RENAL IN PARTICULAR]
- SEVERE FORMS CAUSE COMA AND DEATH

BONES pain and fractures,(kidney) **STONES, ABDOMINAL GROANS** pain, constipation **AND PSYCHIC MOANS** mood swings IN

HYPERPARATHYROIDISM

MEDICAL EMERGENCY:>15 mg/100ml

Bone cysts and fractures



SEVERE HYPERCALCEMIA

[>14.5mg/100ml]

1. **CORRECT DEHYDRATION** AND WASH OUT CALCIUM THROUGH THE KIDNEY BY N/S INFUSION
2. FUROSEMIDE [**LASIX**] AND SOD. SULFATE INCREASE URINARY EXCRETION OF CALCIUM
3. I.V INORGANIC **PHOSPHATES**. INHIBIT BONE RESORPTION
3. **PLICAMYCIN** ; FOR BONE METS
4. STEROIDS; FOR 1- **SARCOIDOSIS**
2- **VIT.D INTOXICATION**
3- **ADDISON'S DIS**
5. **CALCITONIN**; FOR **RENAL & CARDIOVAS.DIS.**
6. **HEMODIALYSIS**; FOR **RENAL FAILURE**

MAGNESIUM : Normal range :1.5-2.5 meq/l.

- MAINLY **INTRACELLULAR** LIKE POTASSIUM
- DEFICIENCY SIMILAR TO CALCIUM DEFICIENCY OR POTASSIUM EXCESS [**NEUROMUSCULAR HYPERACTIVITY**]
- HYPOMAGNESEMIA= HYPOCALCEMIA= HYPERKALEMIA**
- NECESSARY FOR ENZYME FUNCTIONS

Hypomagnesaemia: LOW MAGNESIUM

CLINICAL PICTURE

LIKE LOW CALCIUM

1. EXAGGERATED TENDON REFLEXES
2. CHVOSTEK SIGN
3. TETANY

• causes :TPN ,Renal failure , diarrhea , vomiting

IT IS ALWAYS ASSOCIATED WITH Ca AND K DEFICIENCY

REPLACED BY Mg sulfate or chloride solution , IV , MgSO₄ .

Always remember that its **impossible** to correct hypokalemia **without** correction of the Mg.

HYPERMAGNESEIMA : HIGH MAGNESIUM LEVEL

CLINICALY; [LIKE HIGH Ca]

ECG CHANGES [LIKE HIGH K]

- **RENAL FAILURE** : IN ASSOCIATION WITH HIGH K , HIGH Na, LOW Ca.
- **CLINICALY**; [LIKE HIGH Ca] WEAKNESS,LOSS OF REFLEXES , PARALYSIS, COMA, DEATH.
- **ECG CHANGES** LIKE HIGH K.
- **TRETMENT** : CACIUM GLUCONATE IV , INSULIN +GLUCAOSE , DIALYSIS.

PHOSPHATE (2.5-4.5 mg/dl)

- **HYPERPHOSHATEMIA** :Mostly due to **Renal failure**.
- Majority are asymptomatic ,may have symptoms associated with hypocalcaemia.
- **Treatment** : **sevelamer chloride, a phosphate binder** , low phosphate diet , dialysis

HYPOPHOSPHATEMIA

Critical value is less then 1 mg/dl

- Mostly due to **re-feeding syndrome** , ICU patients.
- Clinically : muscle weakness , **failure to wean from the ventilator** , infection risk due to poor leukocyte chemo-taxis from low ATP , encephalopathy .
- **Treatment** : **potassium phosphate.**

ACID -BASE BALANCE

- RESPIRATORY + METABOLIC (kidney) *V.important to differentiate
() respiratory and metabolic
- NORMAL VALUES :
- PH = 7.35-7.45
- PCO₂ = 35-45
- HCO₃ = 22-26
- **LUNG** : CO₂ REGULATION , RAPID EFFECT .
- **KIDNEY** : HCO₃ REGULATION , SLOW EFFECT

RESPIRATORY

- **ALKALOSIS** : HYPERVENTILATION , low pCO₂.
- **ACIDOSIS** : POOR MINUTE VENTILATION (COPD, CNS DEPRESSION
PTX , PLEURAL EFFESION, MORHPINE), high pCO₂.

METABOLIC

- **Loss of bicarbonate** : diarrhea , ileus , fistulas, high output ileostomy.
- **Increase in acid** : lactic acidosis , DKA , Renal failure .

METABOLIC ALKALOSIS

- Vomiting , NG suction , diuretics , **Gastric Outlet Obstruction** leads to **hypochloremic hypokalemic** , pyloric stenosis.
- Loss of CL & H from stomach >> alkalosis hypocholermic .
- Water loss > >kidney >> NA/K exchange >> hypokalemia
- **Hypochloermic hypokalemic metabolic alkalosis**
- KEY to correct CL - deficit >> give N.S

FLUID MANAGEMENT

- IV fluid replacement includes maintenance, deficit, and ongoing losses. (Daily requirements of the patient unless he is NPO)

Calculation of maintenance:

- **100/50/20 rule:** per 24 hour
 - 1st 10 kg: **100 mL/kg/day**
 - 2nd 10 kg: **50 mL/kg/day**
 - Rest: **20 mL/kg/day**

E.g: 75 kg / day

1st 10 kg: $10 \times 100 = 1000$ ml/kg/day

2nd 10 kg: $10 \times 50 = 500$ ml/kg/day

Rest: $20 \times 55 = 1100$ ml/kg/day

$1000 + 500 + 1100 = 2600$ ml/kg/day

- **4/2/1 rule (per hour)**

- 1st 10 kg: **4 mL/kg/hour**
- 2nd 10 kg **2 mL/kg/hour**
- Rest: **1 mL/kg/hour**

Solutions

- **o Crystalloids:** Most commonly used
 - Isotonic
 - Hypertonic
 - Hypotonic
- **o Colloids:**
 - Albumin
 - Dextran
 - Hydroxyl starch

Isotonic Crystalloids

for maintenance and can be used for resuscitation

- **0.9%** normal saline and ringer's lactate.
- Distribute uniformly through the extracellular compartment.
- After one hour, only **25%** of the total volume remains in the intravascular space. (In the case of resuscitation, you have to give massive amounts of fluid ,so usually Ringer test **colloid والأكثر استخداما** for resuscitation)

- The lactate in Ringer's lactate will be converted to bicarbonate >Patients will become alkalotic. **IMPORTANT**
- Ringer's lactate is designed to mimic the extracellular fluid; it is called a balanced salt solution.
- **o The most common trauma resuscitation fluid is Ringer's lactate**

Hypertonic solutions

- **Examples** include 7.5% normal saline and 3% normal saline.
- **o Indications:**
 - Used in **shock/burns** (usually in combination with colloids like dextran)
- **o Effects:**
 - ✓ Studies have shown that it causes significant blunting of neutrophil activation with a transient increase in serum sodium that normalizes within 24 hours.
 - ✓ This effect may help in **decreasing** widespread tissue **damage** and **multiorgan dysfunction** seen after a **traumatic injury**.
- **o Side effects:**
 - Hyponatremia (hyposmolarity) and hypochloremia
 - Hypokalemia
 - Central pontine demyelination

Hypotonic solutions

- **Examples** include **D5W** and **0.45% NaCl**.
- Should not be used for volume expansion, because they only expand 10% of the infused volume.
- Indicated to replace free water deficits.

FLUID MANAGEMENT

☒ IF ELECTROLYTES ARE **NORMAL**

REPLACEMENT IS BY **1- N/S** WHICH CONTAINS:

Na 154 meq

Cl 154 meq

that may reduce the ph.value WHICH IS GOOD IN STOMACH OUTLET OBSTRUCTION [FOR CORRECTION OF ALKALOSIS] but might cause acidosis in normal PH

OR 2- RINGER LACTATE WHICH CONTAINS:

Na 130 meq , k 4 meq , Ca 3 meq

Cl 109 meq , Lactate 28 meq that changes to bicarbonate in the liver which causes Alkalosis

Suitable for metabolic acidosis

Fluid loss associated with alkalosis: Replace with N/S

Fluid loss associated with acidosis: Replace with Ringer lactate

Colloid solutions

has multiple types

- They contain **high** molecular weight substances that remain in the intravascular space
- More **expansive** than crystalloids.

• INDICATIONS:

- ❖ When crystalloids fail to sustain plasma volume. This is due to the low colloid osmotic pressure in **burn** patients and in cases of **peritonitis**.

• SIDE EFFECTS:

- Pulmonary edema
- Renal failure
- Bleeding disorders

• Early use of colloids in the resuscitation regimen may result in more prompt resuscitation of tissue perfusion. Moreover, it might decrease the total volume of required fluids.

• Albumin preparations: **5% or 25% albumin**. Indicated for volume expansion.

• However, they are not indicated for patients with adequate colloid oncotic pressure (albumin >2.5)

Thank you!

لله در أهل الطب . . لله درهم ودر عنائهم

لله هلاك أبدانهم ، لله انشغال عقولهم ، لله كل خلية من خلايا عقولهم
استخدمت في فهم أعراض مرض ما وحفظها ، لله حساباتهم لكل ساعة
ودقيقة ، لله نومتهم القلقة بين جبال الكتب ، لله إخفاء أحزانهم عن أهلهم
براً ولطفاً بقلوبهم ، لله أعوامٌ يقضونها من أجل تشخيص مريض في دقائق

لله . . كلنا لله

نعوذ بك من العجز والكسل ومن ارتخاء الأيدي قبل الوصول

يا ويلنا إن تعبنا هنا وهناك