Fluid and ELECTROLYTES

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DISTRIBUTION OF FLUIDS

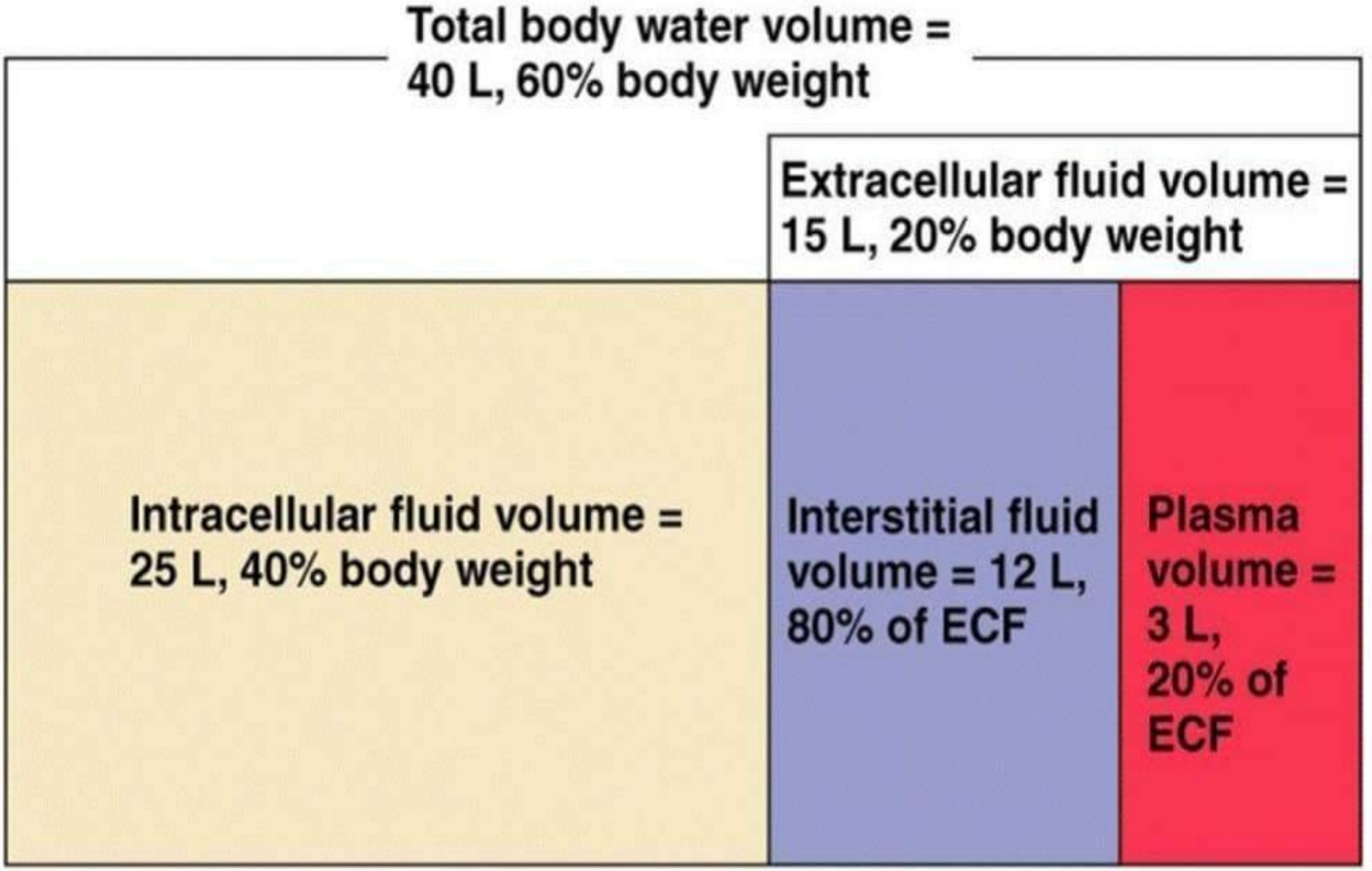
- **Fluids = 60% of total body Weight**

- **Two major compartments** >>
- Intracellular 2/3rd , Extra-cellular 1/3rd .

- Extra-cellular >>

  interstitial fluid (in between cells) 3/4th

  intravascular fluid (plasma) 1/4th


Total body water volume =
40 L, 60% body weight
Extracellular fluid volume =
15 L, 20% body weight
Intracellular fluid volume =
25 L, 40% body weight
Interstitial fluid volume = 12 L, 80% of ECF
Plasma volume = 3 L, 20% of ECF

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.**

- **Na IONS**[**ELECTRICITY**] ACCOUNT FOR MOST THE **OSMOLALITY** IN THE **EXTRACELLULAR** FLUID
- WHEREVER SODIUM GOES WATER GOES WITH IT PROPORTIONALLY

- **Posm(mOsm/kg) = 2× Na+ + [glucose]/18+BUN/2.8**
- 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:

- 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 .

Daily secretions :

- 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^-
- Bile – HCO_3^-
- Small intestine – HCO_3^-, K^+
- Large intestine – K^+
- Dialysis can remove K, Ca, Mg, PO_4, urea, and creatinine

Third spacing !

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

- INTRVASCULAR , INTRACELLAUR SPACSES as the first two spaces.

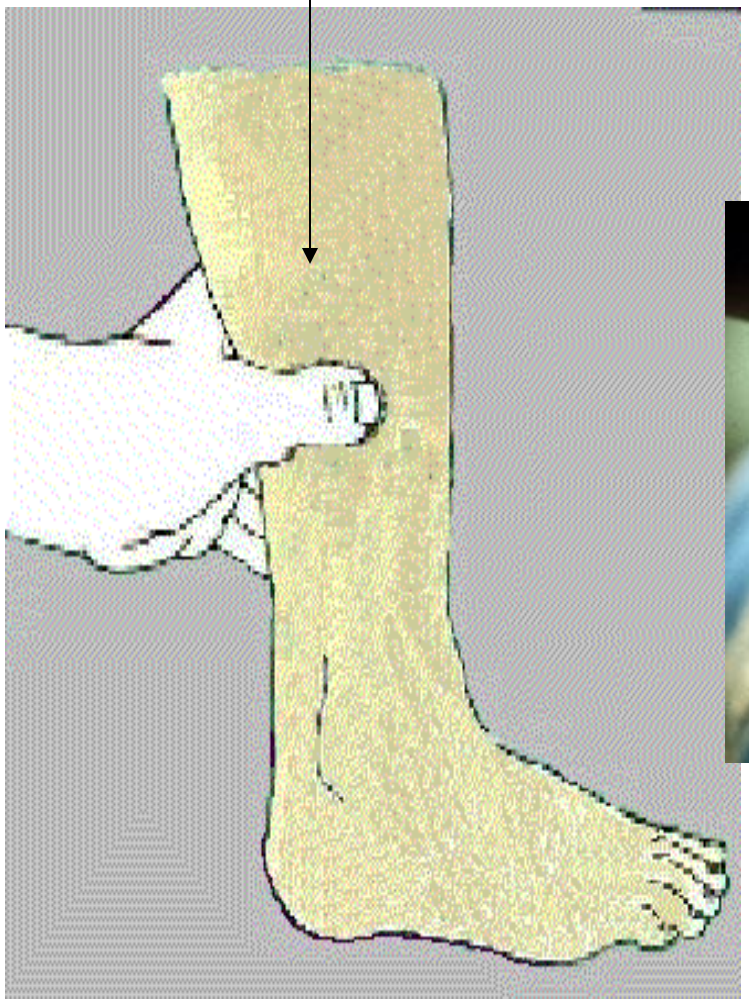
- **POSTOPERATIVE !**
- Around post op **day 3 ,** Fluids return back to intravascular space so be aware of fluid overload at this time.

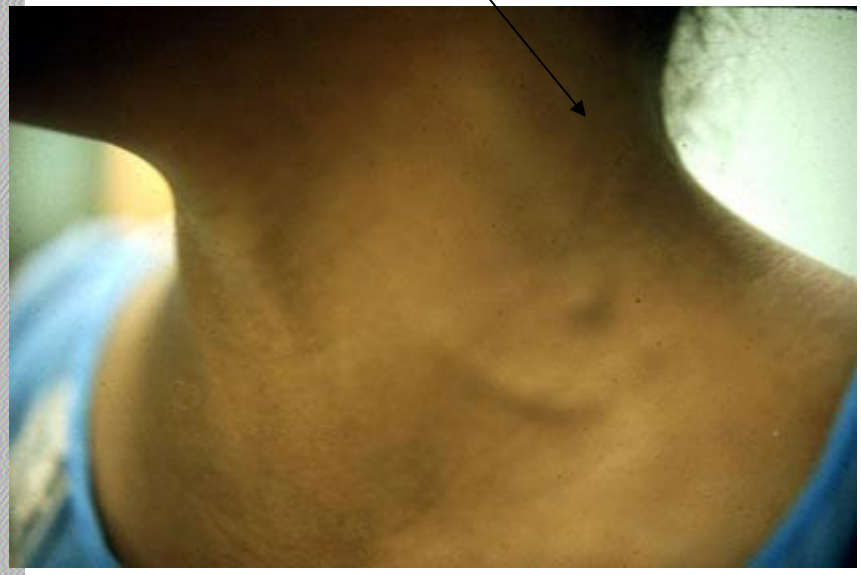
INPUT = OUTPUT = EUVOLEMIA

- **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 , PCWP
- Lactic acid
- CXR findings

LEG OEDEMA TESTING

NECK VEINS DISTENTION

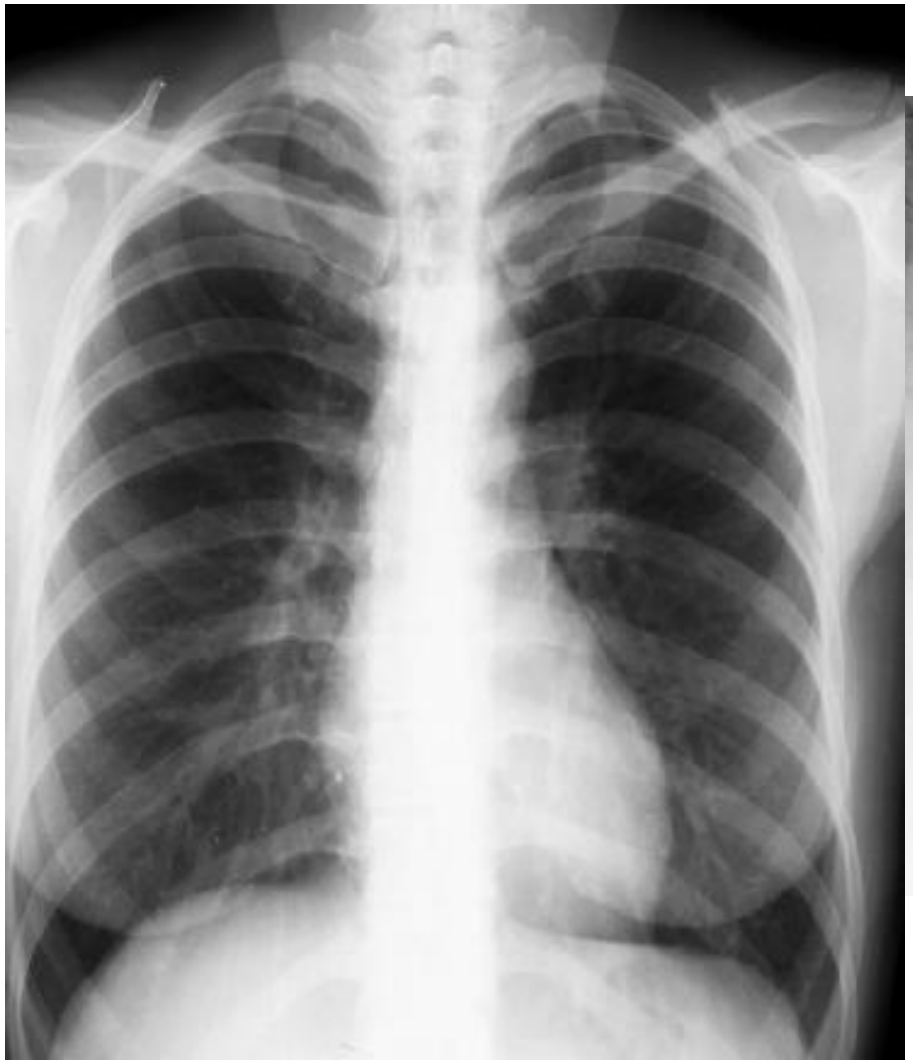


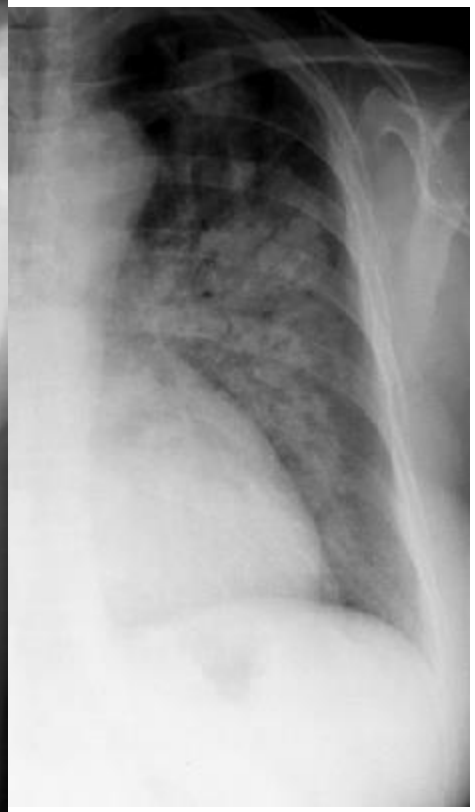


CHEST X-RAY

NORMAL

PULMONARY OEDEMA





Electrolyte implance

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.

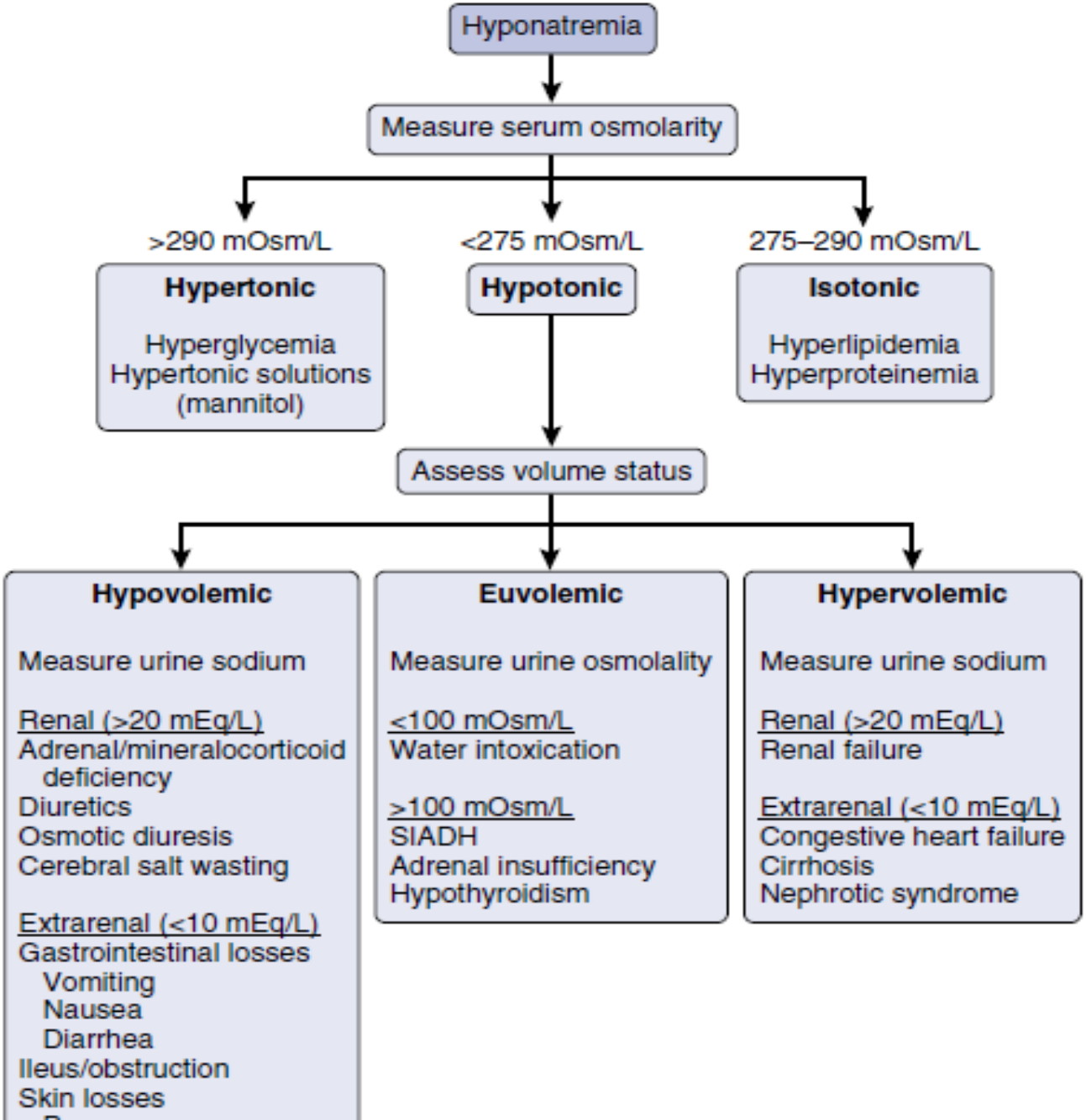
HYPONATREMIA

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

- **Hypotonic hyponatremia**
- **HYPO-VOLEMIC** : NG suction , burns , pancreatitis , diaphoresis >> **IV NS & Correct underlying cause**

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

- **HYPER-VOLEMIC :** RF, CHF , LIVER FAILURE , DILITUIONAL , FLUID **OVERLOAD >> fluid restriction and diuretics**


Hyponatremia
Measure serum osmolarity
>290 mOsm/L
<275 mOsm/L
275–290 mOsm/L
Hypertonic
Hyperglycemia
Hypertonic solutions
(mannitol)
Hypotonic
Isotonic
Hyperlipidemia
Hyperproteinemia
Assess volume status
Hypovolemic
Measure urine sodium
Renal (>20 mEq/L)
Adrenal/mineralocorticoid deficiency
Diuretics
Osmotic diuresis
Cerebral salt wasting
Extrarenal (<10 mEq/L)
Gastrointestinal losses
Vomiting
Nausea
Diarrhea
Ileus/obstruction
Skin losses
Euvolemic
Measure urine osmolality
<100 mOsm/L
Water intoxication
>100 mOsm/L
SIADH
Adrenal insufficiency
Hypothyroidism
Hypervolemic
Measure urine sodium
Renal (>20 mEq/L)
Renal failure
Extrarenal (<10 mEq/L)
Congestive heart failure
Cirrhosis
Nephrotic syndrome

HYPONATREMIA

- Clinically : seizures , confusion lethargy , coma weakness

- Always don’t forget to correct hyponatremia slowly (0.25–0.5 mEq/L per hour), to avoid central pontine myelinolysis.

HYPERNATREMIA

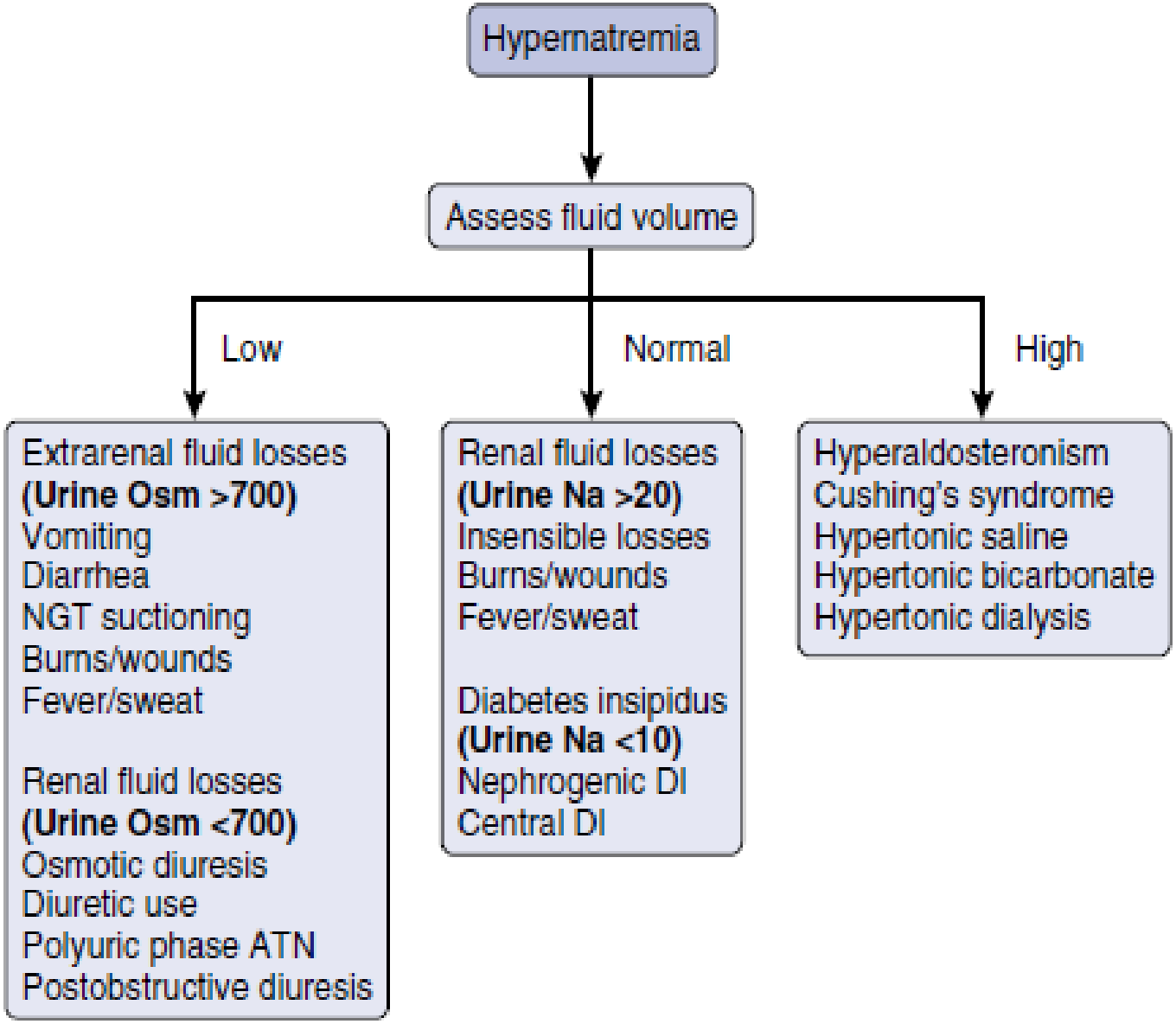
- Hypernatremia is defined as a sodium level greater than 145mEq/L
- (moderate, 146–159 mEq/L; severe, ≥160 Eq/L).
- **Causes :**

➢ In surgical pt. its mostly due to volume depletions with inadequate hydration , others : DI Vomiting diarrhea , diuretics

- **Clinically :**

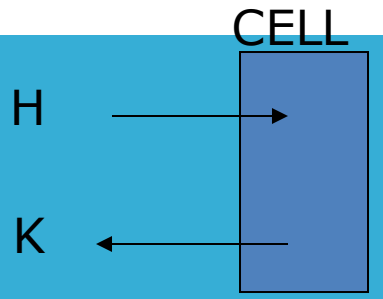
➢ seizures ,confusion, tremors .respiratory paralysis .

- **Slow lowering of serum sodium is very important less than 12 mEq/day TO AVOID SEZIURES . (brain edema)**
- Treatment by hypotonic fluids D5W ., ½ NS , ¼ NS


Hypernatremia
Assess fluid volume
Low
Normal
High
Extrarenal fluid losses
(Urine Osm >700)
Vomiting
Diarrhea
NGT suctioning
Burns/wounds
Fever/sweat
Renal fluid losses
(Urine Osm <700)
Osmotic diuresis
Diuretic use
Polyuric phase ATN
Postobstructive diuresis
Renal fluid losses
(Urine Na >20)
Insensible losses
Burns/wounds
Fever/sweat
Diabetes insipidus
(Urine Na <10)
Nephrogenic DI
Central DI
Hyperaldosteronism
Cushing's syndrome
Hypertonic saline
Hypertonic bicarbonate
Hypertonic dialysis

B

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



- 98% INTRACELLULAR
- 2 % EXTRACELLULAR

- 10% LOSS OF TOTAL BODY K
  DROPS SERUM LEVEL FROM
  4 to3 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.
- HYPERKALEMIA LEADS TO ACIDOSIS.
- ACIDOSIS LEADS TO HYPERKALEMIA

Hyperkalaemia due to acidosis

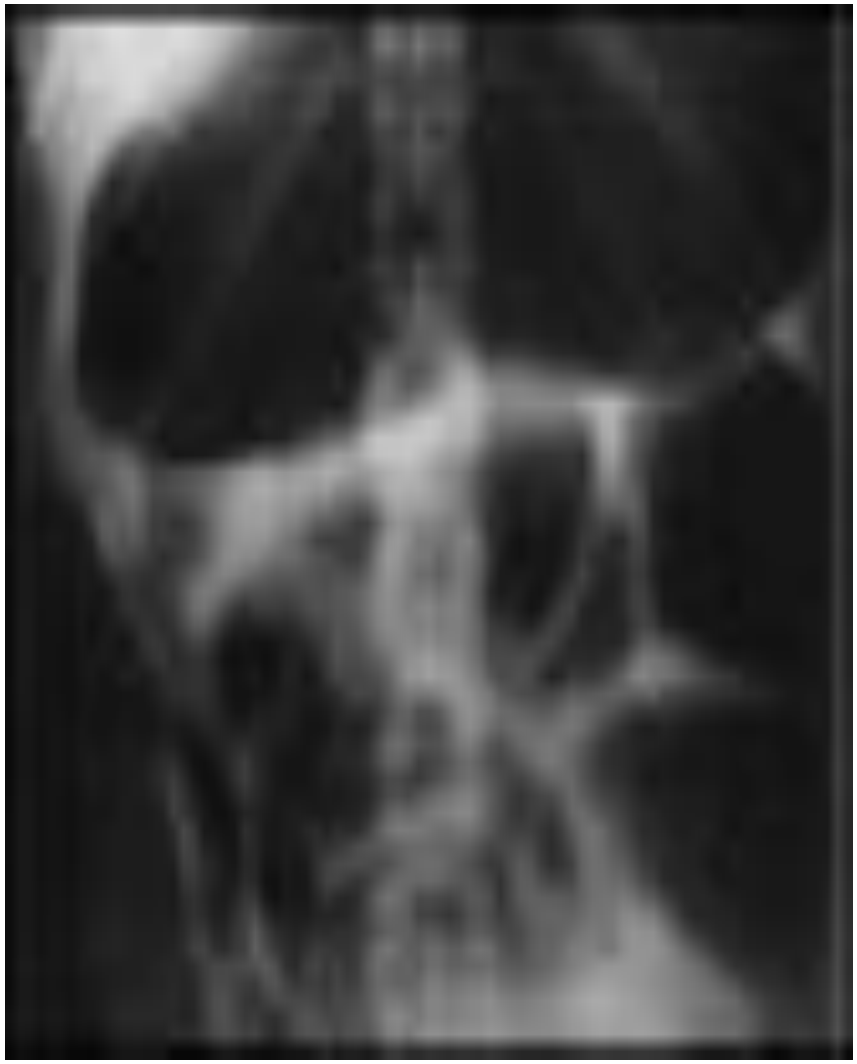
IN ACIDOSIS [H] IONS GO INTO THE CELL TRYING TO REDUCE THE ACIDOSIS .

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

HYPOKALAEMIA

- **IS THE COMMONEST INTHE SURGICAL WARD**

PARALYTIC ILEUS



HYPOKALAEMIA
LOW SERUM POTASSIUM

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

- **1-GIT.** **A-[LOW INTAKE]** **OR**
  B-[HIGH LOSS; VOMIT. N/GTUBE, DIARRHOEA]
- **2-RENAL LOSS BY A-DIURETICS OR**
  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**

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, PVC ,AF.

TREATMENT OF HYPOKALAEMIA

Treat the underlying cause if possible

- **REPLACEMENT:-**
- **KCL IV**

- **MAX RATE THROGH :**
- **PERIPHERAL IV LINE : 10 mEq/hour**
- **CENTRAL LINE : 20 mEq/ hour**

BE CAREFUL OF HYPERKALAEMIA >>RAPID CORRECTION >>> **CARDIAC ARREST IN DIASTOLE**

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**
- **BURNS**
- **CRUSH INJURY**
- **SEVERE CATABOLIC STATE [SEPSIS]**
- **RENAL FAILURE**
- **ADDISON`S DISEASE**
- **BLOOD TRANSFUION**

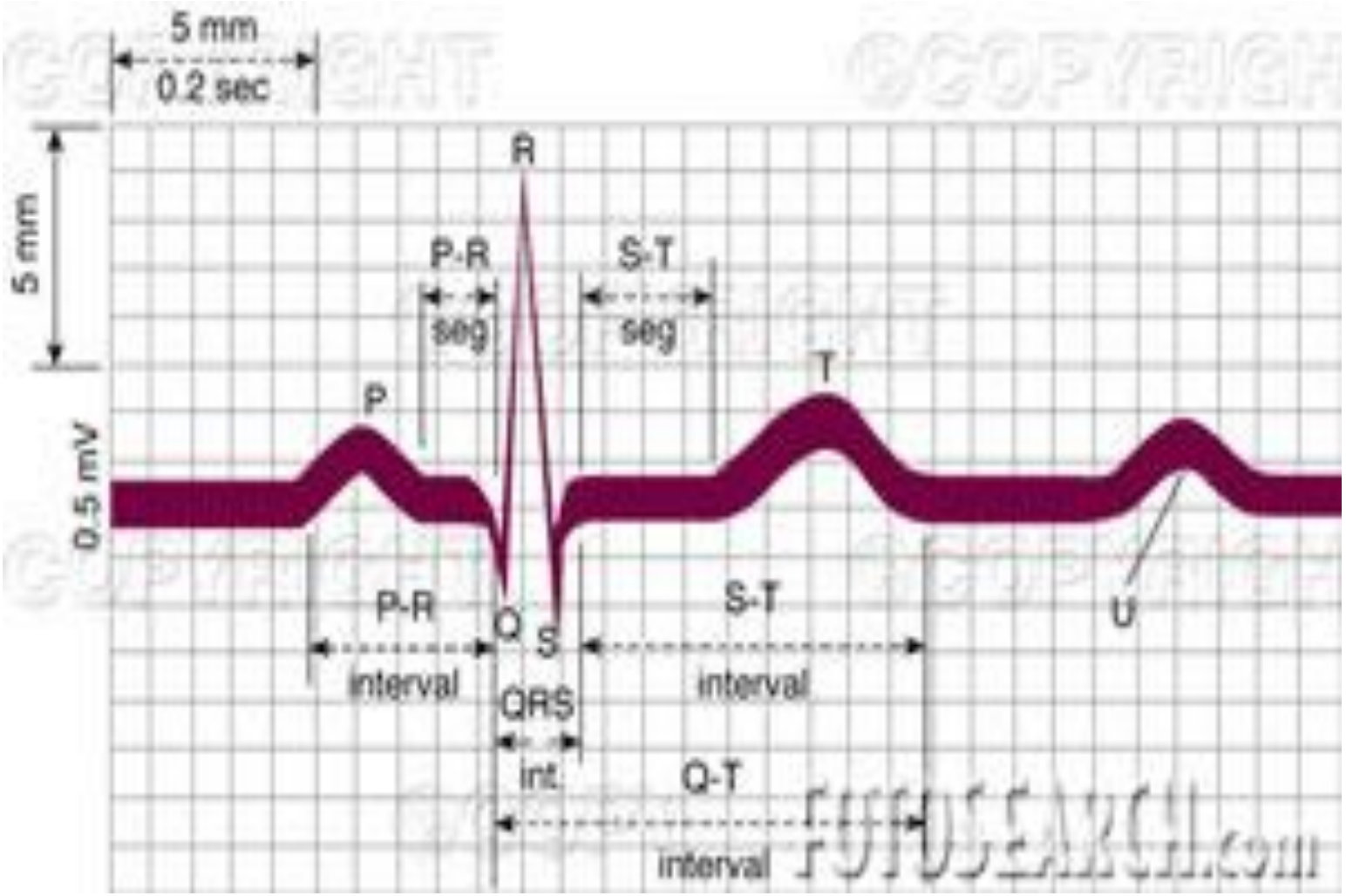
OTHER CAUSES

- **7-ACIDOSIS**
- **8-LEUCOCYTOSIS[MARKED]**
- **9-THROMBOCYTOSIS[ABOVE ONE MILLION]**
- **10-HAEMOLYSIS**
- **11-HAEMOLYSED SPECIMEN**

HYPERKALAEMIA

- **Clinically** :
- Decreased DTR
- Weakness
- Parasthesia
- Paralysis
- Respiratory failure

NORMAL ECG


5 mm
0.2 sec
5 mm
0.5 mV
R
P-R
seg
S-T
seg
T
P
P-R
interval
Q
S
S-T
interval
U
QRS
int.
Q-T
interval

ECG CHANGES in hyperkalemia :

critical value > 6.5

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

EMERGENCY TREATMENT OF HYPERKALAEMIA

- **CALCIUM GLUCONATE I.V. REVERSES** THE ACTION OF [K] ON THE HEART

- **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 COTROVERSIAL

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 , ENZYMETIC 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

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

9-RHABDOMMYOLYSIS

ALL SOFT TISSUE INJURY CAN CAUSE HYPOCALCEMIA

HYPOCALCEMIA
CLINICAL MANEFESTATION

1-CIRCUMORAL NUMBNESS,TINGLING TIPS OF FINGERS AND TOES

2-NEUROMUCULAR HYPERACTIVITY

A-EXAGERATED DEEP REFLEXES

B-POSITIVE CHVOSTEK SIGN

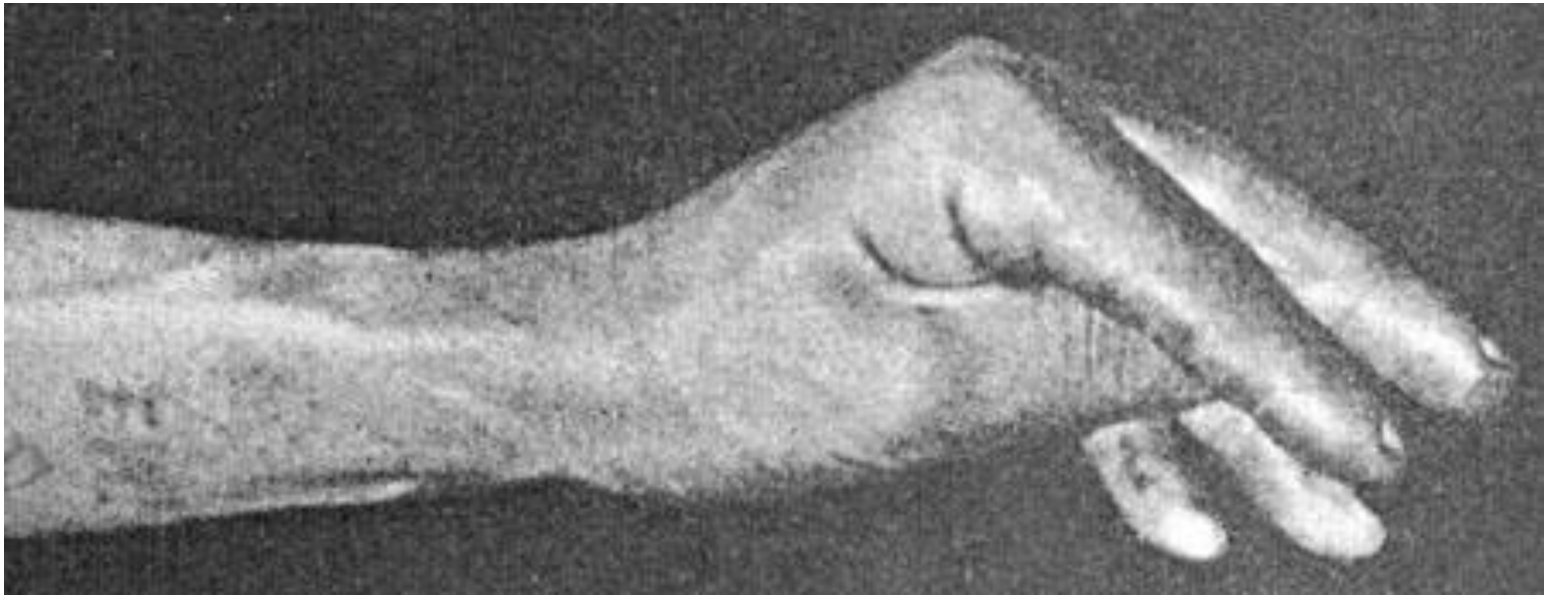
C-CARPOPEDAL SPASM

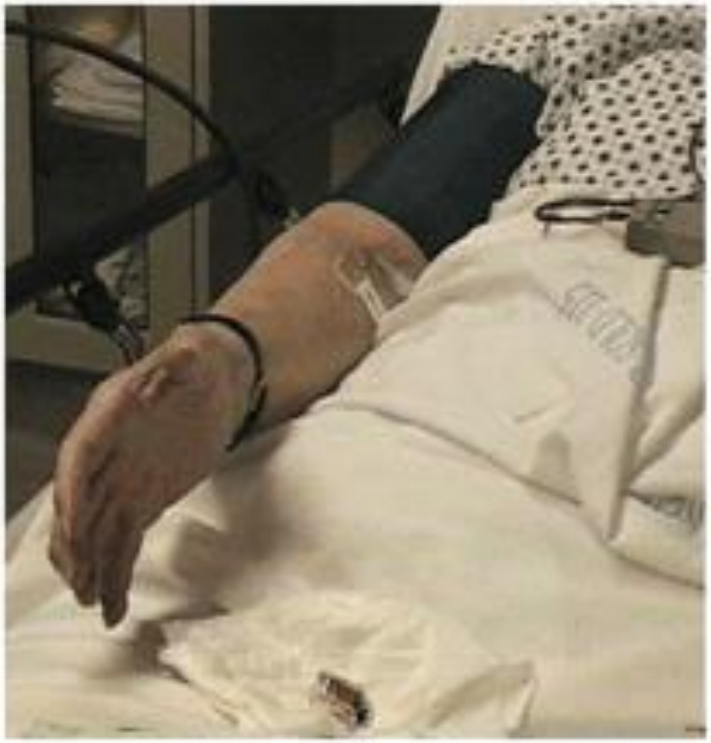
D-MUSCLE CRAMPS

E-ABDOMINAL CRAMPS

F-CONVULSIONS [RARE]

CARPOPEDAL SPASM





A

B

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

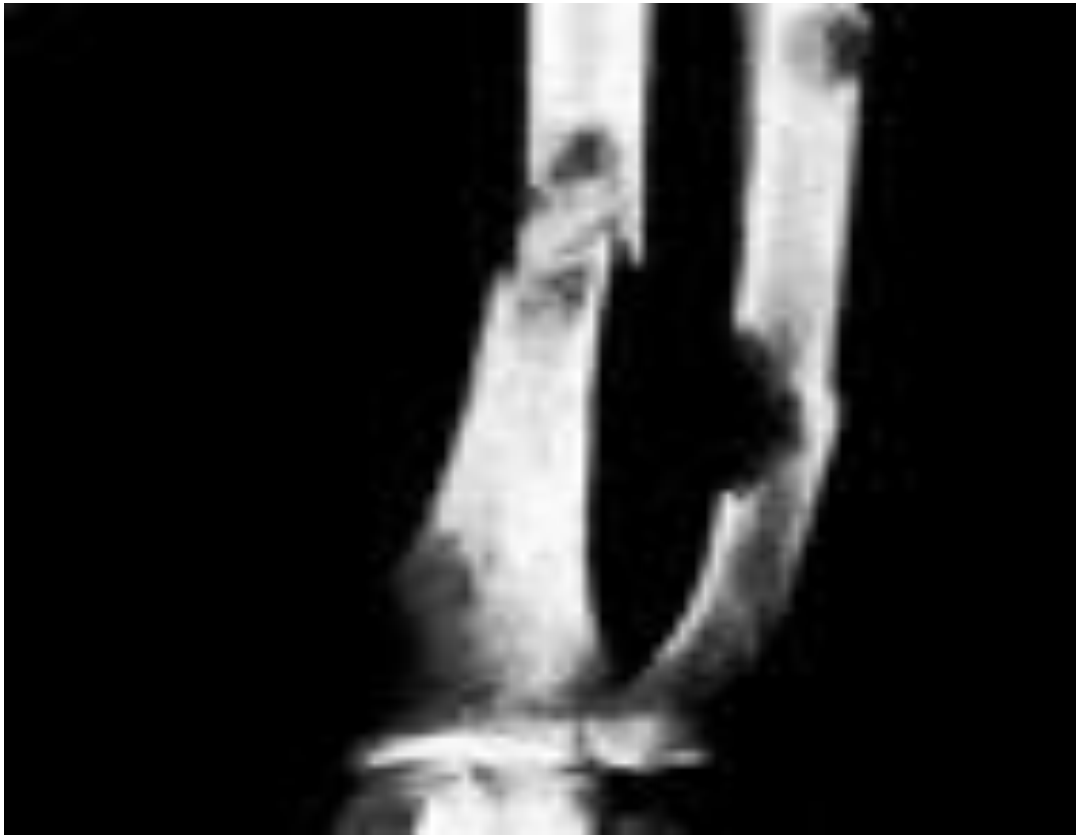
NEUROMUSCULAR LOW ACTIVITY

- 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, STONES, ABDOMINAL GROANS AND PSYCHIC MOANS IN HYPERPARATHYROIDISM

MEDICAL EMERGENCY:>15 mg/100ml

Bone cysts and fractures



SEVERE HYPERCALCEMIA

[>14.5mg/100ml]

1.CORRECT DEHYDRATION AND WASH OUT CALCIUM THROUGHTHE 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]
- NECESSARY FOR ENZYME FUNCTIONS

Hypomagnesaemia: LOW MAGNESIUM

CLINICAL PICTURE

LIKE LOW CALCIUM

1. EXAGERATED 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
- 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 pCO_2.

- ACIDOSIS : POOR MINUTE VENTILATION (COPD, CNS DEPRESSION PTX , PLEURAL EFFESION, MORHPINE), high pCO_2.

METABOLIC

- **METABOLIC ACIDSOSIS**

- **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 , 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.

- **o Calculation of maintenance:**
- **100/50/20 rule:**
  - 1st 10 kg: 100 mL/kg/day
  - 2nd 10 kg: 50 mL/kg/day
  - Rest: 20 mL/kg/day

- **4/2/1 rule (per hour)**
  - 1st 10 kg: 4mL/kg/hour
  - 2nd 10 kg 2 mL/kg/hour
  - Rest: 1mL/kg/hour

Solutions

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

Isotonic Crystalloids

- 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.
- The lactate in Ringer's lactate will be converted to bicarbonate >Patients will become alkalotic.
- 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:**
- Hypernatremia (hyperosmolarity) and hyperchloremia
- Hypokalemia
- Central pontine demylenation

Hypotonic solutions

- Examples include D5W and 0.45% NaCl.
- Should not be sued 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

Suitable for metabolic acidosis

Fluid loss associated with alkalosis: Replace with N/S

Fluid loss associated with acidosis: Replace with Ringer lactate

Colloid solutions

- 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