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FLUIDS & ELECTROLYTES

BY: MOHAMED AKABAWY , BCNSP
CLINICAL PHARMACY DIPLOMA

BODY FLUIDS

Total body water(TBW)50-60%of body wt

Intracellurar fluid 2/3 of TBW 60%

Transcellular fluid 3%of TBW

Extracellular fluids(ECF) 1/3 of TBW 40%

Intravascular space ¼ of ECF 10%

Interstatial space ¾ of ECF 30%

BODY FLUIDS

- Water the most abundant substance of body 50-60% of body WT.
- Adipose tissue least hydrated 10% only.
- Transcellular fluids (specialized fluid) such as: CSF,GIT secretions, aquos humor in eye.
- ECF the most clinically important : plasma 3L ,lymph.
- Osmosis: only solvent move through semipermeable membrane.
- Diffusion: both solvent,solute move to equalize conc. no semipermeable membrane.
- Water deficit (L) = 0.5 * body weight (kg)*(Plasma sodium (mEq/L)/140 mEq/L-1)

BODY FLUIDS

- **Third space:** result in volume depletion , common during critical illness due to capillary permeability increase.
- excess fluids in interstices **(edema).**
- in potential fluid spaces **(effusion).**
- **Fluid balance=** fluid gain (2200-2700ml) --fluid loss(2200-2700ml).

1-Gain : sensible (oral fluid 1100-1400ml + solid food 800-1000ml) + **insensible** (oxidative metabolism 300ml).

2-Loss : sensible (urine 1200-1500 ml + stool 100-200ml) + **insensible** (lung vapors 400ml + skin sweat 500-600ml).

Intravenous Fluid	Infused Volume (mL)	Equivalent Intravascular Volume Expansion (mL)
Normal saline	1000	250
Lactated Ringer solution	1000	250
5% Dextrose	1000	100
Albumin 5%	500	500
Albumin 25%	100	500
Hydroxyethyl starch 6%	500	500

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FLUID RESUSCITATION

- Intravascular fluid depletion can occur because of shock (hypovolemic or septic shock), and it is associated with reduced cardiac function and organ hypo perfusion. usually occur when about 15% (750 mL) of blood volume is lost (e.g., hemorrhage) or shifts out of the intravascular space (e.g., septic shock).

Signs and Symptoms of Intravascular Volume Depletion

- Tachycardia (HR > 100 beats/minute)
- Hypotension (SBP < 80 mm Hg)
- Orthostatic changes in HR or BP
- Increased BUN/SCr ratio > 20:1
- Dry mucous membranes
- Decreased skin turgor
- Reduced urine output
- Dizziness
- Improvement in HR and BP after a 500- to 1000-mL fluid bolus

The goal of fluid resuscitation is to restore intravascular volume and to prevent organ hypo perfusion Because intravascular volume depletion can cause organ dysfunction and death, prompt resuscitation is necessary.

a. Intravenous fluids are infused rapidly, preferably through a large-bore catheter.

b. Intravenous fluids are administered as a 500- to 1000-mL bolus, (~30 mL/kg in septic patients) after which the patient is reevaluated. this process is continued as long as signs and symptoms of intravascular volume depletion are improving.

There is no difference between crystalloids and colloids in the time to achieve fluid resuscitation or in patient outcomes. Colloids have not been shown to be superior to crystalloids, and they are associated with higher cost and some adverse effects.

Colloids can be considered after fluid resuscitation with crystalloid has failed to achieve hemodynamic goals or after clinically significant edema limits the further administration of crystalloid.

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MAINTENANCE INTRAVENOUS FLUIDS

- indicated in patients who are unable to tolerate oral fluids.
- The goal of maintenance intravenous fluids is to prevent dehydration and to maintain a normal fluid and electrolyte balance.

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Calculation of Daily Water Requirements:

Method 1: Body Weight Water Requirement.

- 1st 10 kg 100 mL/kg.
- 2nd 10 kg 50 mL/kg.
- Each additional kg 20 mL/kg (≤50 yrs).
- 15 mL/kg (>50 yrs).

Method 2: Age Water Requirement.

- Young athletic adult 40 mL/kg.
- Most adults 35 mL/kg.
- Elderly adults 30 mL/kg.

Method 3:

- 1 mL/kcal energy expenditure.

C=A 65-year-old man (weight 80 kg) with a 3-day history of a body temperature of 102°F (38.9°C), lethargy, and productive cough is hospitalized for community-acquired pneumonia. His medical history includes uncontrolled hypertension and coronary artery disease. His vital signs include heart rate 104 beats/minute, blood pressure 112/68 mm Hg, and body temperature 101.4°F (38.6°C). His urine output is 10 mL/hour, K+ is 4 mEq/L, BUN is 46 mg/dL, SCr is 1.7 mg/dL, and WBC is 10.4 × 103 cells/mm3 . Other laboratory values are normal.

1. Which is most appropriate at this time?

A. Furosemide 40 mg intravenously.

B. Albumin 25% 100 mL intravenously over 60 minutes.

C. Lactated Ringer solution 1000 mL intravenously over 60 minutes.

D. D5 W/0.45% sodium chloride plus potassium chloride 20 mEq/L to infuse at 110 mL/hour.

C=2. After 2 days of appropriate antibiotic treatment, the patient has a WBC of 9 × 103 cells/mm3 , and he is afebrile. His blood pressure is 135/85 mm Hg, and his urine output is 45 mL/hour. His albumin is 3.2 g/dL, BUN is 14 mg/dL, K+ is 3.9 mEq/L, and SCr is 1.4 mg/dL. All other laboratory values are normal. His appetite is still poor, and he is not taking adequate fluids. He has peripheral intravenous access.

Which option is most appropriate to initiate?

A. Peripheral PN to infuse at 110 mL/hour.

B. Albumin 5% 500 mL intravenously over 60 minutes.

C. D5 W/0.45% sodium chloride plus potassium chloride 20 mEq/L to infuse at 110 mL/hour.

D. Lactated Ringer solution to infuse at 75 mL/hour

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D=1. The administration of 1 L of 0.9% sodium chloride to a normo-natremic patient will increase the intravascular and interstitial fluid compartments by:

- A. 1000 mL and 0 mL, respectively
- B. 0 mL and 1000 mL, respectively
- C. 750 mL and 250 mL, respectively
- D. 250 mL and 750 mL, respectively

A=2. Assuming the same weight and serum sodium concentration, which of the following patients has the greatest free water deficit ?

A. A 35-year-old male

B. A 75-year-old male

C. A 35-year-old female

D. A 75-year-old female

OSMOLARITY

1. Plasma osmolality is normally 275–290 mOsm/kg.
2. Plasma osmolality is maintained within a normal range by thirst and secretion of antidiuretic hormone [ADH] from the posterior pituitary.
3. Sodium salts are the primary determinant of plasma osmolality, and they regulate fluid shifts between the IC and EC fluid compartments.
4. Plasma osmolality (mOsm/kg) can be estimated: (2 × Na mEq/L) + (glucose mg/dL/18) + [(BUN mg/ dL) ÷ 2.8].
5. Increases in plasma osmolality cause an osmotic shift of fluid into the plasma, resulting in cellular dehydration and shrinkage.
6. Decreases in plasma osmolality cause an osmotic shift of fluid into cells, resulting in cellular overhydration and swelling

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ELECTROLYTES

- Mainly electrolytes in body : Na , k , Mg , Po4 , Ca .
- need to know :

1. normal level.
2. Symptoms of deficiency or excess.
3. Causes of deficiency or excess.
4. Treatment of deficiency or excess.
5. Precautions .

POTASSIUM

- Major intracellular cation.
- Potassium conc. (ECF 3.5-5 meq /L).
- Determine resting membrane potential.
- Normal daily potassium requirements range from(0.5 to1.5 meq /kg / day).

Regulation of K-level:

1. K-intracellular shifting : catecholamine's (β2- agonist),insulin , alkaline PH.
2. Kidney excretion.

• Glucose . • insulin. • Catechol amines (β2-agonist). • Alkalosis.	IC shifts of potassium.
• Cellular destruction. • Acidosis.	Release in ECF.

	Hypokalemia	Hyperkalemia
Level	< 3.5 mEq/L	> 5.0 mEq/L
Symptoms	1. Asymptomatic 3.0–3.5 mEq/L. Nonspecific symptoms (M.weakness , lethargy , constipation). 2. Severe symptoms:(muscle necrosis, Rhabdomyolysis, ascending paralysis, Arrhythmias , Death). 3. ECG changes (flattened T waves or elevated U wave)	1. Asymptomatic if (< 5.5 mEq/L) , Nonspecific symptoms. 2. (> 6 meq/L) : a)Muscle (Cramping,Weakness,Paralysis) b) ECG changes (narrowed T waves and widening of the QRS, progress to ventricular fibrillation and asystole). c) Arrhythmias (Brady arrhythmias (cardia relax) , Ventricular fibrillation).
Causes	1.GIT Losses(vomiting , diarrhea , fistula). 2.Shift into cell (alkalosis, insulin , hypothermia , β2- agonist). 3.Low intake . 4.Urinary losses (diuretic , mineralocorticoid) 5.hypomagnesemia	1.Renal insufficiency. 2.High Intake. 3.Shifts out cell (Acidosis , hyperthermia). 4.Drug(ACE,ARB, K-sparing diuretics) . 5. Pseudo hyperkalemia (cell damage). Venipuncture ,Leukocytosis ,Thrombocytosis

goals	1.Symptoms relief. 2.Restoring K to normal . 3. preventing hyperkalemia.	1.Antagonizing cardiac effects (relaxation). 2.Symptoms relief. 3.Normal serum K.
treatment	1.Correct the cause. 2.Administration of K. Oral - 40 to 100 mEq daily on 2-4 doses. -Safer & ↓ overcorrection. 1. liquid(Less expensive, Unpleasant taste). 2. Slow-release tablets (Tolerated, Ulceration& bleeding GIT). IV in Severe cases (Kcl ,K –acetate).	-Discontinue: 1.Exogenous potassium. 2.Medications cause hyperkalemia. -Cardiac muscle protection: IV calcium gluconate →with ECG changes to restore normal membrane excitability ; dose is 10 mL of 10% calcium gluconate administered over 2–10 minutes, repeated in 5 minutes if no improvement in ECG.. - K- intracellular shift: i. Insulin and dextrose. ii. Sodium bicarbonate. iii. β2-adrenergic agonists. -Removing excess K: I. Loop and thiazide diuretics. ii. Cation-exchange resins (sodium polystyrene sulfonate, , patiromer, Sodium zirconium cyclosilicate). iii. Dialysis.

	Hypokalemia	Hyperkalemia
Precautions	**Infusion rates:** • ≤ 10 - 20 mEq/h • 40 mEq/h only in:((Emergency , Severe symptomatic)). • > 10-20 mEq/h require Cardiac monitoring ECG. • Central venous avoiding phlebitis. • Dextrose solutions avoided because worsen the hypokalemia by stimulating insulin release that promotes an intracellular shift of potassium. • A magnesium deficit should be corrected because hypomagnesaemia may result in refractory hypokalemia due to the impairment of Na-K-ATPase pump activity.	• Upon resolution continued monitoring every 4 to 12 hours until levels normal .

B =A 61-year-old man comes to the emergency department with shortness of breath and bilateral lower leg edema. Pertinent vital signs and laboratory values include heart rate 30 beats/minute, blood pressure 102/57 mm Hg, K+ 7.9 mEq/L, Na+ 139 mEq/L, glucose 278 mg/dL, Ca2+ 8.8 mg/dL, digoxin 2.2 ng/mL, BUN 49 mg/dL, and SCr 2.4 mg/dL. His ECG shows wide QRS and peaked T waves. His medical history includes heart failure, atrial fibrillation, coronary artery disease, peripheral arterial disease, and diabetes. The patient has peripheral intravenous access and an external pacemaker. Which treatment is most appropriate?

A. Calcium gluconate 10 mL intravenously over 2 minutes.

B. Insulin 10 units intravenously.

C. Sodium bicarbonate 50 mEq intravenously over 10 minutes.

D. Albuterol 10 mg nebulized over 10 minutes.

MAGNESIUM

TOTAL CONTENT 25 G IN BODY

I. ICF

- 50% to 60% in bone.
- Remainder in:

A. Cardiac.

B. skeletal.

C. Liver.

II. 2% ECF

- 1.33% protein bound.
- 2.61% active.
- 3.5% complexed.

Activation of enzymatic reactions
e.g metabolism of:
1. CHO.
2. FAT.
3. PROTEIN.

Component of:
- **Bone & factor in PTH secretion.**
- **Neuromuscular transmission.**
- **Cardiovascular excitability.**
- **Muscle contractility.**

Regulation of Mg level by:

1. GIT: Absorb in distal jejunum & ileum.

2. Renal: Elimination of one third of intake maintaining (Mg=1.8 to 2.3 mg/dL) .

3. Bone

	Hypomagnesemia	Hypermagnesemia
	< 1.8 mg/dL	> 2.8 mg/dL
Symptom	1. *Neuromuscular hyper excitability* (Tetany , spasm, seizures). 2. *Cardiac complications :* (Arrhythmias, sudden cardiac death, and hypertension).	(Mg > 4.8 mg/dL). Nausea, vomiting, Muscular weakness , Hypotension , Bradycardia , heart block, asystole, respiratory failure, and death.
Causes	*1.↓ intake or absorption* (Mg-free PN , GIT necrosis). *2. Renal losses* (Acute tubular necrosis) . 3.Drug-induced (diuretics). *4. Intracellular shifts of magnesium* e.g. Refeeding . 5. hypokalemia and hypocalcemia	1.Renal insufficiency 2.Increased Intake e.g. antacids

	Hypomagnesemia	Hypermagnesemia
Treatment	• Oral isn't preferred because of : a)Slow onset b)GI intolerance. • Symptomatic patients should initially be treated with (8–32 mEq) of MG-so4 by slow IV. infusion (8meq/hr) to avoid hypotension or increased renal excretion because of rapid administration). followed by about 0.5 mEq/kg/day added to intravenous fluid and administered as a continuous infusion. • For emergency treatment (e.g., torsades), Mg can be administered by intravenous push. • Reduce the dose by half in patients with kidney insufficiency. • About half of administered Mg is excreted in the urine; therefore, magnesium replacement can occur over 3–5 days.	1. Stop all Mg containing medications. 2. Normal saline with *Loop diuretics for asymptomatic.* 3. *IV calcium gluconate:* For symptomatic & severe cases to maintain the cardiac stability. 4. *hemodialysis* In Renal insufficiency.

PHOSPHORUS

- Majority in bones & soft tissues.
- Main intracellular anion.
- serum conc. (2.7 to 4.5 mg/dL).
- **Functions:**

1. Bone and cell membrane 2. Maintenance pH 3. ATP
4. 2,3-diphosphoglycerate synthesis for oxygen release from Hg to tissues.
5. Neurological 6. Muscular

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PHOSPHORUS

- **Factors affecting serum levels:**

1. Intake.
2. Intestinal absorption.
3. Renal excretion.
4. Shift.

• Glucose . • insulin. • Catechol amines . • Alkalosis.	IC shifts of PO4.
• Cellular destruction. • Acidosis.	Release in ECF.

	Hypophosphatemia	Hyperphosphatemia
	< 2.5 mg/dL	> 4.5 mg/dL
Symptom	1. Neurologic:(confusion, seizures, and coma). 2. Neuromuscular:(weakness, or rhabdomyolysis) 3. cardiac and respiratory failure. 4. Tissue hypoxia (↓2,3-diphos.glyc. conc.)	1.Soft tissues and vascular calcification. 2. Hypocalcaemia symptoms (Anorexia, nausea, vomiting , dehydration ,Neuromuscular irritability).
Causes	1. *Renal elimination*. 2. *IC shifting*. 3. TTT of diabetic *ketoacidosis.* 4. *Po4-binders,* Al-containing antacids or sucralfate. 5. *Administration of CHO loads* in malnourished causing refeeding syndrome.	1. *Renal insufficiency*. 2. *Cellular destruction* e.g. (Trauma , hyper catabolism ,Cytotoxic agents Hemolysis , rhabdomyolysis) 3. *Transcellular shifts* to the ECF as respiratory or metabolic acidosis. 4. *Use of phosphate-containing laxatives* or enemas, especially in the elderly.

	Hypophosphatemia	Hyperphosphatemia
Treatment	1. *Asymptomatic with functioning GIT. (Oral PO4),but* poorly absorbed. 2. Symptomatic patients receive (0.5–0.75 mmol/kg of IBW) of phosphorus administered IV over 3–6 hours (maximum rate is 7.5 mmol/hour) *as faster rates cause:* *a)Thrombophlebitis b)Metastatic deposition.* IV K3 PO4. or Na3 PO4. , IV K preferred over Na *unless* *a)K is > 4 mEq/L* b)Renal insufficiency exists. 1.Dosing is empirical because serum not reflect stores. *2.Reduced doses in renal patient .*	1.Stop Exogenous sources. 2.In normal renal function, (Volume repletion + diuretics is sufficient) . 3.Dialysis with renal insufficiency.

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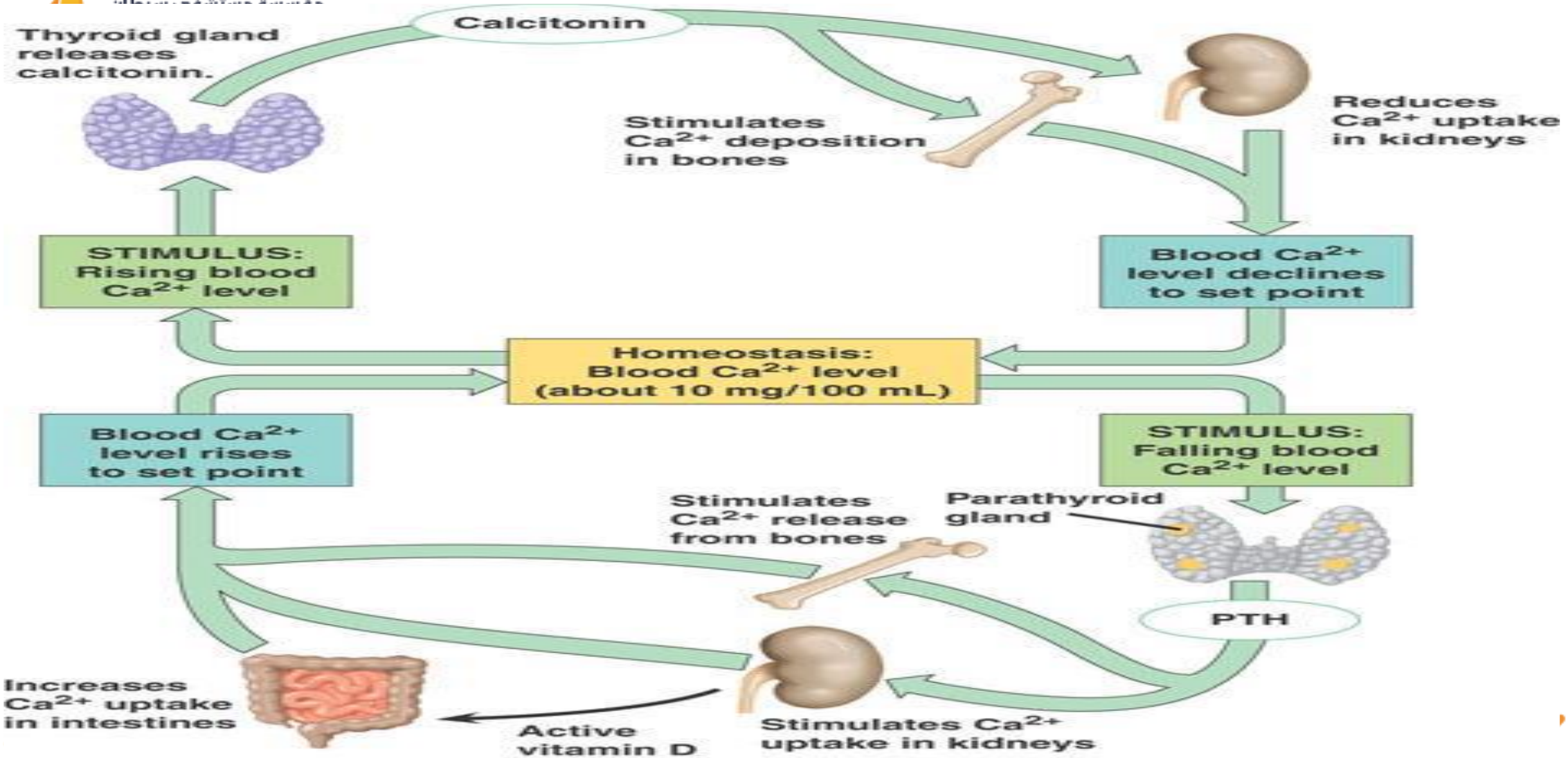
CALCIUM

- One of the most abundant ions in the body, accounts for 1% to 2% of adult human body weight.
- Serum calcium concentrations are under hormonal control primarily mediated by PTH, vitamin D, and calcitonin.
- Low serum calcium concentrations stimulate the release of PTH, which increases bone resorption , augments renal conservation of calcium, and activates vitamin D, which increases intestinal calcium absorption.
- Calcitonin is released by the thyroid gland in response to elevated serum calcium concentrations and acts to inhibit bone resorption and increase urinary calcium excretion.

Functions:

•Cell membranes. •Neuromuscular. •Endocrine activites. •Coagulation. •Bone.

- Corrected total calcium(mg/dl) =measure total calcium (mg/dl)+0.8(4-albumin g/L).



Total body calcium

- 99% in → Bones & Teeth
- <1% in the serum:
  - Complexed with non protein (PO4, HCO3,..).
  - 50% protein bound to (albumin).
  - The ionized is the active.

	Hypocalcaemia	Hypercalcemia
Serum	• Total calcium (< 8.5 mg/dL) . • ionized calcium (<1.1 mmol/L).	• Total calcium (> 10.5 mg/dL). • ionized calcium (>1.3 mmol/L).
Symptom	• CVS: Hypotension , decreased myocardial contractility , ECG changes. • Neuromuscular: Paresthesia , Muscle cramps, tetany, or seizures.	• Early : Fatigue , nausea, vomiting, constipation , anorexia, and confusion . • Severe may cause Arrhythmias.
Causes	1.vitamin D deficiency. 2.hypoparathyroidism. 3.Alkalosis decrease ionized Ca. 4.hyperphosphatemia. 5.Drugs induced hypocalcaemia (Bisphosphonates/Calcitonin/Lasix)	1.Hyperparathyroidism/Cancer. 2.Toxicity of vitamin A&D. 3.Chronic use of CaCO3 antacid.

	Hypocalcaemia	Hypercalcemia
Treatment	• Asymptomatic due to hypoalbuminemia ➔ no therapy. • acute symptomatic hypocalcemia: 200–300 mg of elemental Ca2+ administered IV. over 5–10 minutes. 1. 1 g CACL2= 273 mg of elemental Ca2+ administered through a central line; peripheral administration of CACL2 can result in severe limb ischemia. 2. 2–3 g of calcium gluconate= 180–270 mg of elemental Ca2+; preferred for peripheral line. 3. Do not infuse Ca2+ at a rate faster than 60 mg/min ; rapid administration is not recommended, is associated with hypotension, bradycardia, or asystole. 4. Continuous Infusion rate not exceed 0.5 to 2 mg/kg/hr. 5. In cases of hypocalcaemia due to hyperphosphatemia, use (renagel) prior to calcium replacement to reduce the risk of soft tissue calcification.	Candidates for treatment: 1.Mild hypercalcaemia: Hydration and (movement). 2. Severe hypercalcaemia: immediate treatment to avoid: a)acute renal failure. b)arrhythmias. c)Coma. • Treatment tools: 1. Forced diuresis: a) IV hydration At 200 to 300 mL/h to reverse volume depletion due to hypercalcemia. b) Furosemide 40 to 100 mg IV After hydration enhance renal excretion Monitoring avoid volume depletion. 2. Bisphosphonates: Delayed onset so ineffective in acute cases. 3. Hemodialysis: Life threatening hypercalcemia in renal patient.

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D=3. Which of the following electrolyte derangements is commonly seen in a patient with a high output nasogastric tube ?

A. Hyperchloremia and hyperkalemia

B. Hyperchloremia and hypokalemia

C. Hypochloremia and hyperkalemia

D. Hypochloremia and hypokalemia

SODIUM

- normal serum conc is (135-145meq/L).
- Principle cation in ECF ,function as osmotic determinant in regulating water distribution in body.
- Determinant of plasma osmolality (fluid shifts between IC&EC compartment).

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HYPONATREMIA

Hyponatremia: serum level **(<136meq/L)** ,fluid shift into cells.

1. Hypertonic saline is used in traumatic brain injury to reduce an elevated ICP and thereby increase cerebral perfusion pressure , It is also used for cerebral edema or herniation.
2. Hypertonic saline is used for symptomatic hyponatremia.
3. Asymptomatic (SIADH) is usually treated with fluid restriction of less than 1000 mL of fluid per day.
4. Hyponatremia associated with severe hyperglycemia, As hyperglycemia is corrected with insulin, the serum sodium will normalize
5. **Corrected Na** = serum Na + [1.6 (glucose – 100)/100]
6. Hyponatremia associated with hypervolemia

a. this situation is treated with fluid restriction or diuresis.

b. Symptomatic hyponatremia is uncommon in patients with heart failure.

c. Hypertonic saline could be considered in symptomatic patients; however, they may also need diuresis to prevent worsening volume overload.

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SYMPTOMS

1.are generally attributable to hypo-osmolality, with subsequent water movement into brain cells causing cerebral edema.

2. If hyponatremia occurs chronically, cerebral cell swelling is prevented by osmotic adaptation.

a. Solutes move out of brain cells to prevent the osmotic shift of water into brain cells.

b. For this reason, patients with chronic hyponatremia may show less severe or no symptoms.

3. Neurologic symptoms are related to the rate and degree of change in serum sodium.

4. Acute hyponatremia occurs over 1–3 days.

CAUSES

1. Replacement of lost solute with water

a. Loss of solute (e.g., vomiting, diarrhea) usually involves the loss of isotonic fluid; After the loss of isotonic fluid, hyponatremia can develop when the lost fluid is replaced with water.

b. A common cause of hyponatremia in hospitals is the postoperative administration of hypotonic fluid.

2. Volume depletion and organ hypo perfusion stimulate ADH secretion to increase water reabsorption in the collecting tubules, potentially causing hyponatremia.

3. SIADH and cortisol deficiency are both related to the excessive release of ADH.

4. Medications, including thiazide diuretics, antiepileptic drugs (e.g., carbamazepine, oxcarbazepine), and antidepressants (SSRI, TCA), can cause hyponatremia. Drug-induced hyponatremia is more likely to occur in older adults and in those who drink large volumes of water.

5. Renal failure impairs the ability to excrete dilute urine, predisposing to hyponatremia.

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TREATMENT

1. Treat underlying cause.
2. Raise serum sodium at a safe rate, change no greater than 10–12 mEq/L in 24 hours.
3. Treatment depends on volume status, the presence and severity of symptoms, and the onset of hyponatremia.

a. euvolemic or edematous: Fluid restriction (to less than 1000 mL/day)

b. euvolmic (i.e., SIADH) or hypervolemic (i.e., heart failure) use Vasopressin antagonists.

4. intravascular volume depletion, volume must be replaced first with intravenous crystalloids (e.g., 0.9% sodium chloride).
5. Correct hypokalemia, if present, with hyponatremia
6. estimate the Na+ deficit in the following manner: 0.5 (LBW) × (140 – Na+) for women (multiply LBW by 0.6 for men).

PSEUDO-HYPONATREMIA

- **pseudo hyponatremia because Na content in the body is not actually reduced. Instead, Na shifts from the EC compartment into the cells in an attempt to maintain plasma osmolality in a normal range.**
- **Another adaptation to increased plasma osmolality is the shift of water from inside cells to the EC compartment, which further dilutes the Na concentration.**

i. **Severe hyperlipidemia can be associated with a normal or elevated plasma osmolality.**

ii. **Severe hyperglycemia (i.e., during diabetic ketoacidosis) is associated with an elevated plasma osmolality.**

- **Once the underlying condition is corrected, Na will shift out of the cells, and hyponatremia will resolve.**

B=A 72-year-old woman (weight 60 kg) with a history of hypertension has developed hyponatremia after starting hydrochlorothiazide 3 weeks earlier. She experiences dizziness, fatigue, and nausea. Her serum sodium is 116 mEq/L. Her blood pressure is 86/50 mm Hg, and heart rate is 122 beats/minute.

3. In addition to discontinuing hydrochlorothiazide, which initial treatment regimen is best?

A. Administer 0.9% sodium chloride infused at 100 mL/hour.

B. Administer 0.9% sodium chloride 500-mL bolus.

C. Administer 3% sodium chloride infused at 60 mL/hour.

D. Administer 23.4% sodium chloride 30-mL bolus as needed.

C=Which is the best treatment goal for the first 24 hours in correcting the patient's serum sodium from her initial value of 116 mEq/L?

A. **Increase Na+ concentration to 140 mEq/L.**
B. **Increase Na+ concentration to 132 mEq/L.**
C. **Increase Na+ concentration to 126 mEq/L.**
D. **Maintain serum sodium of 116–120 mEq/L.**

D=One day later, the patient has somewhat improved. Her blood pressure is 122/80 mm Hg, and heart rate is 80 beats/minute. Her serum sodium is 120 mEq/L, and K+ is 3.2 mEq/L; she still feels tired. She is eating a regular diet. Her ECG is normal. Which is the best recommendation?

A. D5 W/0.9% sodium chloride plus potassium chloride 40 mEq/L to infuse at 100 mL/hour.
B. 0.9% sodium chloride infused at 100 mL/hour.
C. 3% sodium chloride infused at 60 mL/hour.
D. Potassium chloride 20 mEq by mouth every 6 hours for 4 doses

HYPERNATREMIA

- Serum Na (> 145meq/L) ,so cause hyper osmolality.
- Water move out cells into ECF space ,causing dehydration in brain cells.

Symptoms of hypernatremia: primarily neurologic related to rate of increase in plasma osmolality ,degree of this increase.

- earlier symptoms: lethargy,weakness,irritability.
- progress to seizures,coma,death if serum (Na >158meq/L) .

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CAUSES

1. Loss of water because of fever, burns, infection, renal loss (e.g., diabetes insipidus), gastrointestinal (GI) loss
2. Retention of Na because of the administration of hypertonic saline or any form of Na.
3. Hypernatremia is prevented first by the release of ADH, causing water reabsorption, also prevented by thirst.
4. Cerebral osmotic adaptation patients with chronic hypernatremia can have

a. Brain cells take up solutes, Na+ , and K+ , thus limiting the osmotic gradient between the IC and EC fluid compartments.

b. This prevents cellular dehydration, and it will increase the brain volume toward a normal value, despite hypernatremia. Because of osmotic adaptation, patients with chronic hypernatremia may be asymptomatic.

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SYMPTOMS

1. Similar to hyponatremia, the symptoms of hypernatremia are related to the rate of increase in plasma osmolality and the degree of increase in plasma osmolality.
2. Earlier symptoms include lethargy, weakness, and irritability.
3. Symptoms can progress to twitching, seizures, coma, and death if serum sodium is greater than 158 mEq/L. However, some neurologic injuries may have higher serum sodium targets.
4. Cerebral dehydration can cause cerebral vein rupture with subsequent intracerebral or subarachnoid hemorrhage.

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TREATMENT

1. Rapid correction of chronic hypernatremia can result in cerebral edema, seizure, permanent neurologic damage, and death
2. In patients with symptomatic hypernatremia, serum sodium should be reduced slowly by no more than 0.5 mEq/L/hour or 12 mEq/L/day
3. Treat hypernatremia by replacing water deficit slowly over several days to prevent overly rapid correction of serum sodium

the estimated water deficit (in liters) is (0.4 × LBW) × [(Serum sodium/140) – 1] in women (multiply LBW by 0.5 in men).

4. Administer free water orally or intravenously as D5 W.
5. If concurrent Na and water depletion occur (e.g., vomiting, diarrhea, diuretic-induced depletion), use a combination of D5 W and 0.225% sodium chloride.

D=A 74-year-old woman (weight 50 kg) has been receiving isotonic tube feedings at 60 mL/hour for the past 8 days through her gastrostomy feeding tube. She recently had an ischemic stroke; she is responsive but is not able to communicate. Her serum sodium was 142 mg/dL on the day the isotonic formula was initiated, and it has risen steadily to 149, 156, and 159 mg/dL on days 3, 4, and 8, respectively, after the start of the tube feedings. What is the best treatment for her hypernatremia?

A. Administer sterile water intravenously at 80 mL/hour.
B. Administer D5 W intravenously at 80 mL/hour.
C. Administer D5 W/0.225% sodium chloride intravenously at 80 mL/hour.
D. Administer water by enteral feeding tube 200 mL every 6 hours.