د.وليد نصار الجفال

Lecture- 3

Fluid and Electrolyte Management of the Surgical Patient

Objectives

1. To understand the percentage and composition of body fluids.
2. To understand the function of body fluids.
3. To understand the types and manifestations of electrolytes disturbances.

Fluid and electrolyte management is paramount to the care of the surgical patient.

Body Fluids

Total Body Water

- 50-70% of total body weight is water. It varies according to age, sex, and lean body mass, 60% 0f body weight is water in males and 50% of body weight is water in females because females have more fat than male and fat contain little water.

Fluid Compartments

- Total body water (TBW) is divided into three functional fluid compartments: plasma, extravascular interstitial fluid, and intracellular fluid.
- "Rule of thirds"
    - 2 thirds being intracellular
    - 1 third is extracellular.

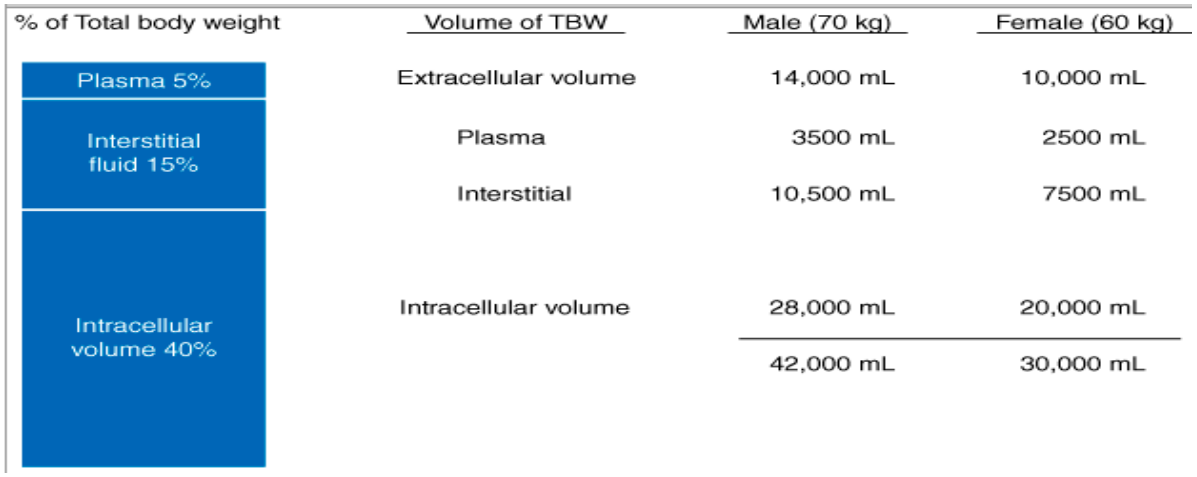
    The ECC is further subdivided into thirds by capillary membrane :

    - 2 thirds is extravascular (interstitial)
    - 1 third is intravascular,

The extracellular water comprises 20% of the total body weight and is divided between plasma (5% of body weight) and interstitial fluid (15% of body weight).

➢ For 70 kg man total body water is 60%

So: 60% × 70kg =42L →28Litres being intracellular and14 litres is extracellular.



Volume of TBW	Male (70 kg)	Female (60 kg)
Extracellular volume	14,000 mL	10,000 mL
Plasma	3500 mL	2500 mL
Interstitial	10,500 mL	7500 mL
Intracellular volume	28,000 mL	20,000 mL
	42,000 mL	30,000 mL

Composition of Fluid Compartments

The extracellular fluid is high in Na and has low K concentrations and is balanced between sodium, the principal cation, and chloride and bicarbonate, the principal anions.

The intracellular fluid is high in K and has low in Na concentration and is comprised primarily of the cations potassium and magnesium, and the anions phosphate and proteins.

FLUID EXCHANGE BETWEEN BODY FLUID COMPARTMENTS

The movement of water across a cell membrane depends primarily on osmosis. To achieve osmotic equilibrium, water moves across a semipermeable membrane to equalize the concentration on both sides. This movement is determined by the concentration of the solutes on each side of the membrane. Osmotic pressure is measured in units of osmoles (osm) or milliosmoles (mOsm) that refer to the actual number of osmotically active particles. For example, 1 mmol of sodium chloride contributes to 2 mOsm (one from sodium and one from chloride). The principal determinants of osmolality are the concentrations of sodium, glucose, and urea (blood urea nitrogen, or BUN).

- The osmolarity of plasma is 290-310mOsm/kg.
- Serum Osmolality = [2 x Na] + [BUN/2.8] + [glucose/18]
- The effective osmotic pressure depend on osmotically acive particles that do not freely pass between the smipermeable membranes of the body
- Proteins are responsible for the effective osmotic pressure between plasma and interstitial compartment(colloid osmotic pressure)

Normal Exchange of Fluid and Electrolytes

The healthy person consumes an average of 2000 mL of water per day, approximately 75% from oral intake and the rest extracted from solid foods. Daily water losses include 800 to 1200 mL in urine, 250 mL in stool, and 600 mL in insensible losses. Insensible losses of water occur through both the skin (75%) and lungs (25%), and can be increased by such factors as fever, hypermetabolism, and hyperventilation. Sensible water losses such as sweating or pathologic loss of GI fluids vary widely, but these include the loss of electrolytes as well as water.

Classification of Body Fluid Changes

Disorders in fluid balance may be classified into three general categories: disturbances in **(A) volume**, **(B) concentration**, and **(C) composition**.

A)- Volume Disturbances

Disorders of volume can be classified into volume deficit and volume excess
Extracellular volume deficit is the most common fluid disorder in surgical patients and can be either acute or chronic. Acute volume deficit is associated with cardiovascular and central nervous system signs, whereas chronic deficits display tissue signs, such as a decrease in skin turgor and sunken eyes, in addition to cardiovascular and central nervous system signs. The most common cause of volume deficit in surgical patients is a loss of GI fluids from nasogastric suction, vomiting, diarrhea, or enterocutaneous fistula. In addition, sequestration secondary to soft tissue injuries, burns, and intra-abdominal processes such as peritonitis, obstruction, or prolonged surgery can also lead to massive volume deficits. Extracellular volume excess may be iatrogenic or secondary to renal dysfunction, congestive heart failure, or cirrhosis. Both plasma and interstitial volumes usually are increased.

Signs and Symptoms of Volume Disturbances		
System	**Volume Deficit**	**Volume Excess**
Generalized	Weight loss	Weight gain
	Decreased skin turgor	Peripheral edema
Cardiac	Tachycardia	Increased cardiac output
	Orthostasis/hypotension	Increased central venous pressure
	Collapsed neck veins	Distended neck veins
		Murmur
Renal	Oliguria	—
	Azotemia	
GI	Ileus	Bowel edema
Pulmonary	—	Pulmonary edema

Volume Control

Volume changes are sensed by both osmoreceptors and baroreceptors.

Osmoreceptors are specialized sensors that detect even small changes in fluid osmolality and drive changes in thirst and diuresis through the kidneys.

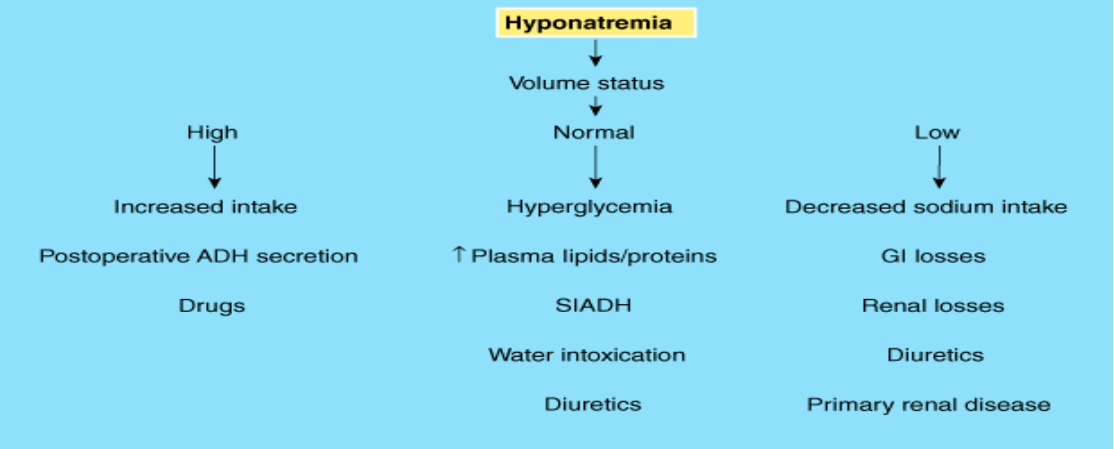
Baroreceptors also modulate volume in response to changes in pressure and circulating volume through specialized pressure sensors located in the aortic arch and carotid sinuses.

B)- Concentration disturbances

Changes in serum sodium concentration are inversely proportional to total body water (TBW). Therefore, abnormalities in TBW are reflected by abnormalities in serum sodium levels.

Hyponatremia

A low serum sodium level occurs when there is an excess of extracellular water relative to sodium. Extracellular volume can be high, normal, or low.



Hyponatremia also can be seen with an excess of solute relative to free water, such as with untreated hyperglycemia or mannitol administration. Glucose exerts an osmotic force in the extracellular compartment, causing a shift of water from the intracellular to the extracellular space. Hyponatremia therefore can be seen when the effective osmotic pressure of the extracellular compartment is normal or even high. When hyponatremia in the presence of hyperglycemia is being evaluated, the corrected sodium concentration should be calculated as follows:

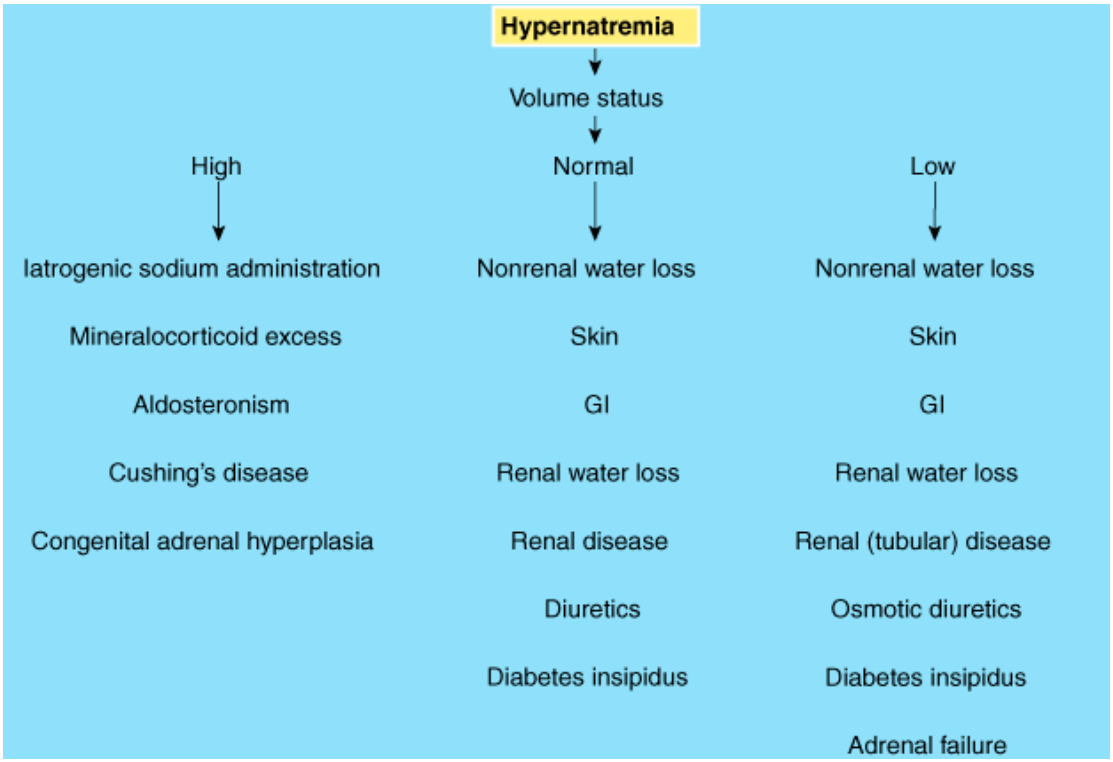
Lastly, extreme elevations in plasma lipids and proteins can cause pseudohyponatremia, because there is no true decrease in extracellular sodium relative to water.

Clinical Manifestations	
Central nervous system	Headache, confusion, hyperactive or hypoactive deep tendon reflexes, seizures, coma, increased intracranial pressure
Musculoskeletal	Weakness, fatigue, muscle cramps/twitching
GI	Anorexia, nausea, vomiting, watery diarrhea
Cardiovascular	Hypertension and bradycardia if significant increases in intracranial pressure
Tissue	Lacrimation, salivation
Renal	Oliguria

Treatment: free water restriction and, if severe, the administration of sodium (3% normal saline), should be corrected slowly because rapid correction of hyponatremia can lead to pontine myelinolysis, with seizures, weakness, paresis, akinetic movements, and unresponsiveness, and may result in permanent brain damage and death.

Hypernatremia

Hypernatremia results from either a loss of free water or a gain of sodium in excess of water. Like hyponatremia, it can be associated with an increased, normal, or decreased extracellular volume



Clinical Manifestations	
Central nervous system	Restlessness, lethargy, ataxia, irritability, tonic spasms, delirium, seizures, coma
Musculoskeletal	Weakness
Cardiovascular	Tachycardia, hypotension, syncope
Tissue	Dry sticky mucous membranes, red swollen tongue, decreased saliva and tears
Renal	Oliguria
Metabolic	Fever

Treatment: Treatment of the associated water deficit. In hypovolemic patients, volume should be restored with normal saline before the concentration abnormality is addressed. Once adequate volume has been achieved, the water deficit is replaced using a hypotonic fluid such as 5% dextrose, 5% dextrose in $^{1}/_{4}$ normal saline, or enterally administered water.

The rate of fluid administration should be titrated to achieve a decrease in serum sodium concentration of no more than 1 mEq/h and 12 mEq/d for the treatment of acute symptomatic hypernatremia. because overly rapid correction can lead to cerebral edema and herniation.

C)- Composition Changes:

Hyperkalemia

Hyperkalemia is defined as a serum potassium concentration above the normal range of 3.5 to 5.0 mEq/L.

Etiology of Hyperkalemia	
Hyperkalemia	
Increased intake	
Potassium supplementation	
Blood transfusions	
Endogenous load/destruction: hemolysis, rhabdomyolysis, crush injury, gastrointestinal hemorrhage	
Increased release	
Acidosis	
Rapid rise of extracellular osmolality (hyperglycemia or mannitol)	
Impaired excretion	
Potassium-sparing diuretics	
Renal insufficiency/failure	
Clinical Manifestations	
System	
GI	Nausea/vomiting, colic, diarrhea
Neuromuscular	Weakness, paralysis, respiratory failure
Cardiovascular	Arrhythmia, arrest
Renal	—
ECG changes	high peaked T waves (early), widened QRS complex, flattened P wave, prolonged PR interval (first-degree block), sine wave formation, and ventricular fibrillation.

Treatment:

- Identify and treat cause
- 10 – 20 mL intravenous 10% calcium chloride over 10 min in patients with ECG abnormalities (reduced risk of ventricular fibrillation)
- 50 mL 50% dextrose plus 10 units short acting insulin over 2-3min (Monitor plasma glucose and K+ over next30-60 min)
- Bicarbonate 1 ampule IV
- Regular Salbutomol nebulizers
- Consider oral or rectal calcium Resonium (ion exchange resin), although this is more effective for non-acute hyperkalaemia.
- Haemodialysis for persistent hyperkalemia

Hypokalemia

Hypokalemia is much more common than hyperkalemia in the surgical patient.

Etiology of Hypokalemia
Inadequate intake
Dietary, potassium-free intravenous fluids, potassium-deficient TPN
Excessive potassium excretion
Hyperaldosteronism
Medications
GI losses
Direct loss of potassium from GI fluid (diarrhea)
Renal loss of potassium (gastric fluid, either as vomiting or high nasogastric output)
intracellular shifts from metabolic alkalosis or insulin therapy

Clinical Manifestations	
GI	Ileus, constipation
Neuromuscular	Decreased reflexes, fatigue, weakness, paralysis
Cardiovascular	Arrest
ECG changes	U waves, T-wave flattening, ST-segment changes, and arrhythmias (with digitalis therapy)

Treatment:

potassium repletion, the rate of which is determined by the symptoms. Oral repletion is adequate for mild, asymptomatic hypokalemia.

If IV repletion is required, usually no more than 10 mEq/h is advisable in an unmonitored setting. This amount can be increased to 40 mEq/h when accompanied by continuous ECG monitoring, and even more in the case of imminent cardiac arrest from a malignant arrhythmia associated hypokalemia. Caution should be exercised when oliguria or impaired renal function is coexistent.