Team Medicine

Electrolyte imbalance

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Definitions:

Osmosis: movement of water between two compartments based on osmotic pressure.

Diffusion: movement of solutes between two compartments based on concentration gradient.

Osmolality: number of osmoles of solute in a kilogram of solvent, measured as mOsm/kg water.

we add alcohol to the equation in case of alcohol toxicity.

-Calc Posm(calculated plasma osmolality) =
(2 x serum Na^+) + blood urea + glucose

-Normal osmolality of body fluids: **283-292** mOsm/kg water

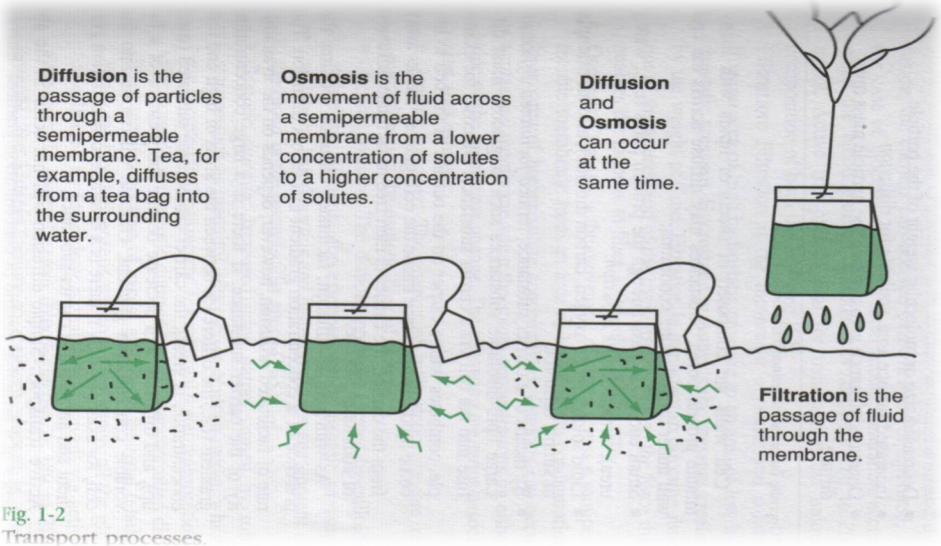


Fig. 1-2
Transport processes

The serum sodium is multiplied by two to account for accompanying anions (chloride and bicarbonate)

Homeostasis: A relative constancy in the internal environment of the body, naturally maintained by adaptive responses that promote cell function and survival. **(the body must maintain the level of fluid and electrolyte otherwise it will affect the metabolism.)**

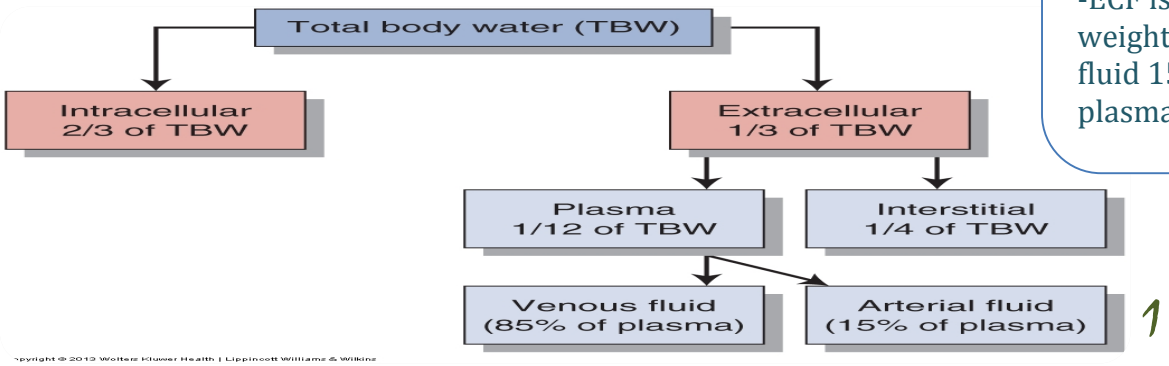
60-40-20 rule:
- TBW is 60% of body weight.

- ICF is 40% of body weight

-ECF is 20% of body weight(interstitial fluid 15% and plasma 5%)

Body fluid compartments: fluctuate based on age & gender

- Men : Total body water(TBW) = 60% of body weight .
- Women: TBW= 50% of body weight.**(more fat)**
- TBW% decreases with age and increasing obesity.
- Distribution of water:



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- Elderly: TBW = 50%
- Pediatrics(infants) TBW= 70%

Compartments are separated by thin semi-permeable membrane with pores that allow fluid movement& molecules of specific size to pass, while preventing others.
The body fluid is composed of water and dissolved substances known as *solutes* (electrolytes or non-electrolytes).

Electrolytes are substances that dissolved in solutions and dissociated into particles called ions

Cations: Positively charged ions.(e.g. Na^+ ,k^+)
Anions: Negatively charged ions.(cl)

ECF and ICF are in **osmotic equilibrium(there is no difference in osmolality, i.e. in steady state osmosis inside the cells= osmosis outside.)**
ICF osm = ECF osm = Posm
The main cation in ECF is Na+. ====➔
The main cation in ICF is k+.======➔ **Equal in concentration 140**

Tonicity:

- To compare the osmolality of a solution to that of another solution (body fluid compartments)

- Used to compare the osmolality of intravenous solutions to that of the serum:
- **Hypotonic**: Solutions have more water than solutes than ECF**(i.e. osmolality inside the cells will be higher than osmolality outside ➔water moves inside the cells and they will burst.)**
  Water will move from ECF into ICF.
  e.g. Distilled Water 0.45% NaCl (1/2)0.33% NaCl (1/3)

- **Isotonic:** Solutions have the same solute concentration as the ECF.
  It will remain in the ECF.
  e.g. NS (0.9% NaCl), Ringers Lactate, 2/3 DW-1/3 NS, 5% Dextrose in Water (D5W)

- **Hypertonic:** Solutions have more solutes than water than ECF. **(i.e. osmolality outside the cells will be higher than osmolality inside ➔water moves outside the cells and they will shrink.)**
  Water will move from ICF to ECF
  3% NaCl,10%-50% Dextrose,D5W-1/2 NS,D5NS,Amino acid solution.

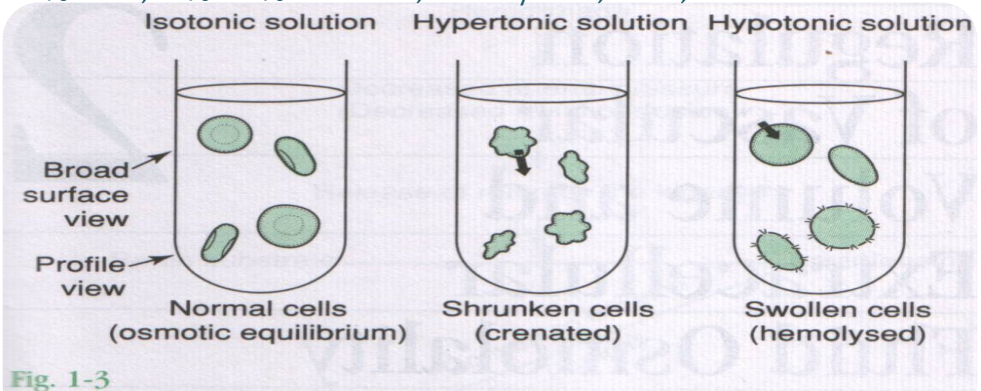


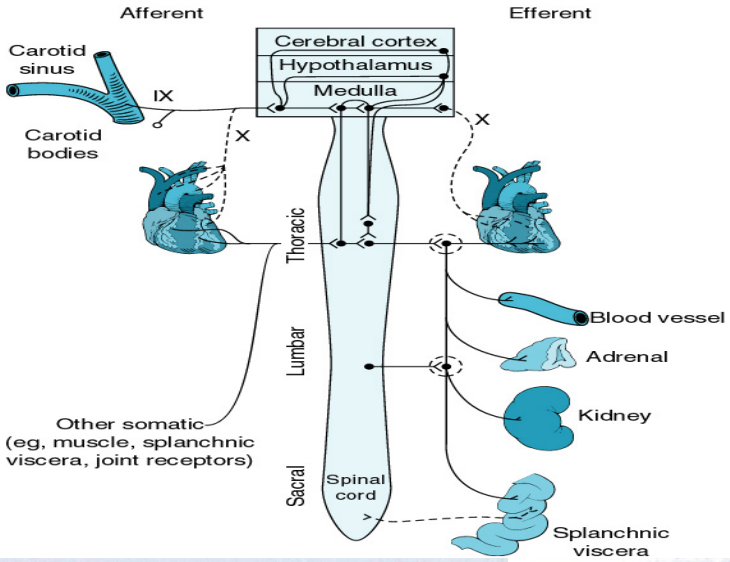
Fig. 1-3

Regulation Mechanisms of Fluid and Electrolytes:

Two important systems:

- **Thirst center** & **osmoreceptor-antidiuretic hormone system** (vasopressin).
  - They regulate **osmolality and volume.**

- **Renin – Angiotensin-Aldosterone system(aldosterone holds water inside the body while Atrial natriuretic peptide ANP do the opposite by reducing the water inside the body.)** & **stretch receptors (baroreceptors)**
  - They regulate **volume only.**



Receptors exist on Afferent pathway ,e.g. Aortic Arch& heart & other organs.

Efferent controls Cardiac output, Renin system, and Kidney absorption.

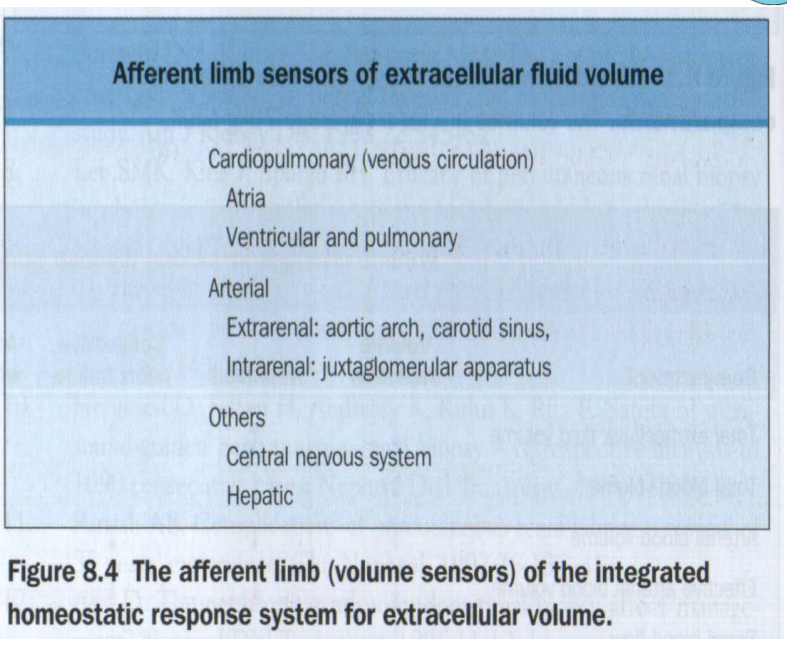


Figure 8.4 The afferent limb (volume sensors) of the integrated homeostatic response system for extracellular volume.

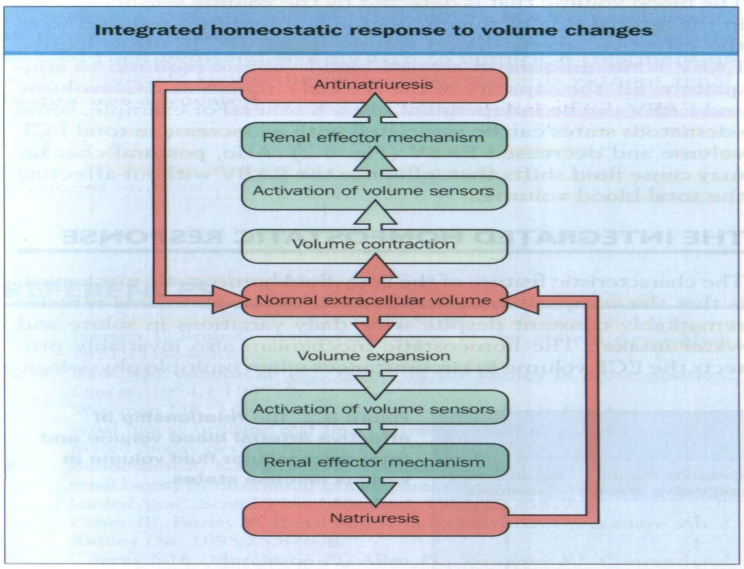
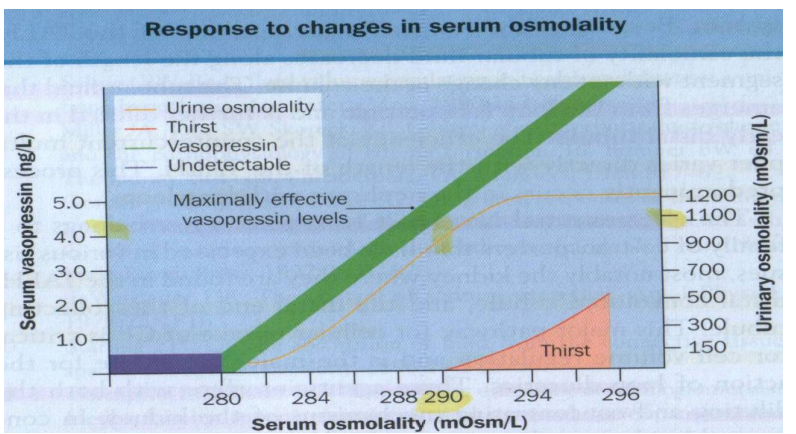


Figure 8.3 A general overview of the integrated homeostatic response system regulating extracellular fluid volume during volume contraction and expansion.



If there is an increase in serum osmolality, ADH will be released and thirst will be stimulated.
Range of urine osmo : 50-1200 mmol

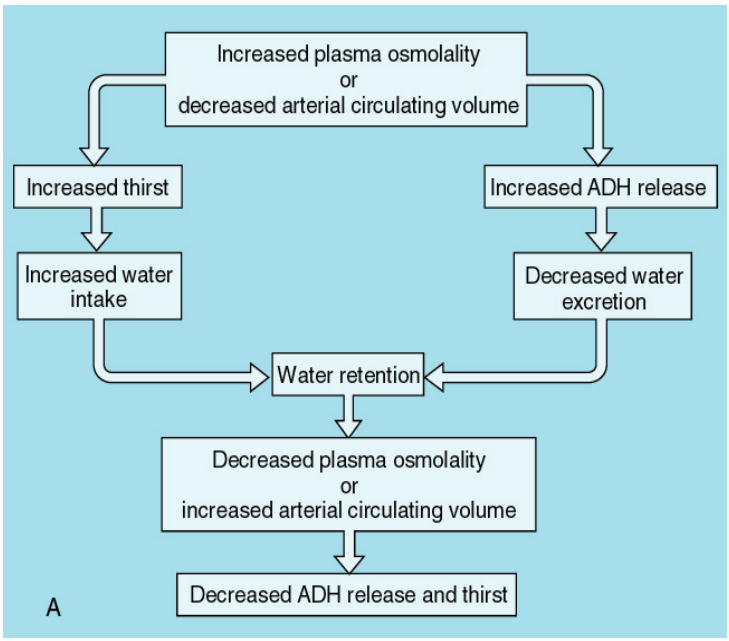
The Linear Relationship Between Urine Specific Gravity and Uosm:

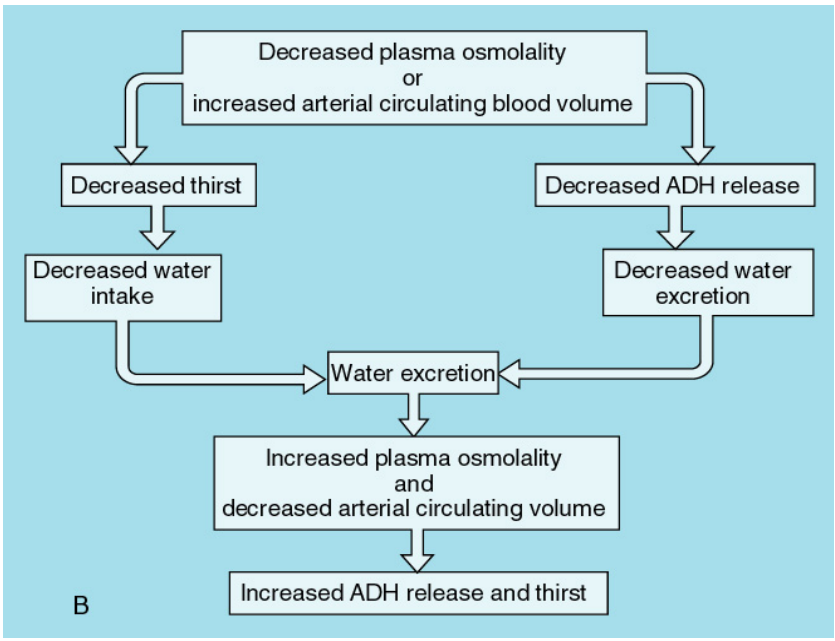
Laboratory investigation might take time to check on urine osmolality, so we estimate it by using a urine dipstick to know the specific gravity.

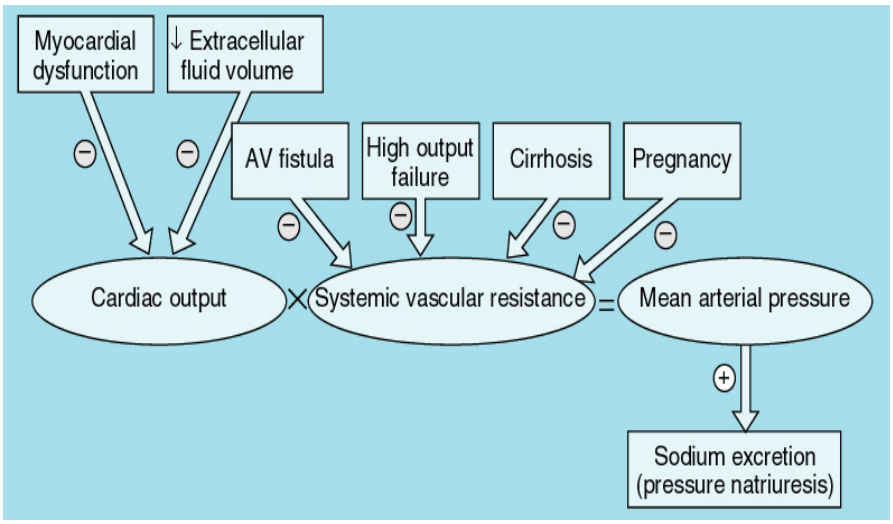
specific gravity: how dense the solution compared to pure water.

Plasma is 1.008 , since it contain solutes it is heavier than pure water.

SG	Osmolality (mOsm/Kg H2O)
1.010	**300 – 400**
1.020	**700 – 800**
1.030	**1000 – 1200**







In case of decrease in the blood pressure the kidneys will start to reserve Sodium which causes an increase in Sodium and water levels beyond what we need leading to edema.

Seen in CHF, and cirrhosis

Effective Arterial Blood Volume (EABV):

is the amount of arterial blood volume required to adequately 'fill' the capacity of the arterial circulation. **(How much blood we have in the arterial side to tell the brain we have enough fullness, enough volume filling that part of circulation.)**

- is determined by cardiac output and systemic vascular resistance.
- ↓ EABV:
  - ↑ CO
  - ↑ SVR
  - ↑ Renal Na retention
- ↑ EABV:
  - ↓ CO
  - ↓ SVR
  - ↓ Renal Na retention
- **ECF volume and EABV can be independent of each other**
  - Edematous states: increase in total ECF volume and decreased EABV.**(Heart failure)**
  - Postural changes: may cause shifts that influence the EABV without affecting the total blood volume.

Clinical features of Hypovolaemia & Hypervolaemia:

Symptoms:

Hypovolemia:

- Thirst
- Dizziness on standing
- Weakness

Hypervolemia:

- Ankle swelling

	Hypovolemia	Hypervolemia
Signs	Low JVP	Raised JVP
	Postural hypotension	Peripheral oedema
	Tachycardia	Pulmonary crepitations
	Dry mouth	Pleural effusion
	Reduced skin turgor	Ascites
	Reduced urine output	Hypertension (sometimes)

(no single sign will lead to a definitive diagnosis)

Sodium and Water:

ECF volume= **absolute** amounts of Sodium and water.

Plasma Na^+ = ***ratio*** between the amounts of Sodium and water (Concentration)

- Disturbance of **Na+** balance may lead to **hypovolemia or hypervolemia**.
- Disturbance of **water** balance may lead to **hyponatremia or hypernatremia.**

- Hyponatremia = Water Excess
- Hypernatremia = Water Deficit

- Hypervolemia (Edema) = Sodium Excess
- Hypovolemia (Dehydration) = Sodium Deficit

	Hyponatremia	Hypernatremia
Hypovolemia	Hemorrhagic Shock with good oral water intake but not enough salt.(**somebody with hemorrhagic shock and able to drink water)**	Diarrhea and people who don't have access to water like children and seniors.
Hypervolemia	Advanced Congestive Heart Failure. Patients will have edema	Hemodialysis Patient after 3% Saline infusion.

Intravenous Solutions:

- Crystalloids vs Colloids. **(both have the same effect on mortality.)**
- Crystalloids: are intravenous solutions that contain solutes that readily cross the capillary membrane
  Dextrose and electrolyte solutions. They are used in practice
- Colloids are intravenous solutions that DO NOT readily cross the capillary membrane.**(faster volume expansion, since it stays only inside blood vessel)**
  Blood, albumin, plasma. Mainly used by surgeons.

Solution	Gluc	Na^+	K^+	Ca^{+2}	Cl^-	Lact	mOsm/L
D_5W	50	0	0	0	0	0	278
$D_{10}W$	100	0	0	0	0	0	556
NS	0	154	0	0	154	0	308
½ NS	0	77	0	0	77	0	154
D_5 NS	50	154	0	0	154	0	293
D_5 ½ NS	50	77	0	0	77	0	216
2/3-1/3	33	50	0	0	50	0	285
Ringer's Lactate	0	130	4	3	109	28	274

D5W: 5 g dextrose/100 mL (50 g/L) **Lytes:** mEq/L
D10W: 10 g dextrose/100 mL (100 g/L) **Gluc:** g/L
NS (0.9% NS): 0.9 g NaCl/100 mL (9 g/L)
½ NS (0.45% NS): 0.45 g NaCl/100 mL (45 g/L)
2/3-1/3: 2/3 D5W (33 g /L) + 1/3 NS (0.33 g NaCl/100mL or 33 g NaCl/L)

Nov 2013 A R Tarakji, MD 39

Important to know the D5w and the D51/2 NS

D5W: at the begging it is considered as an isotonic(278mOsm/L) but after a period (minutes to hours) the glucose will be utilized by the insulin inside the body and only water will be left making it a hypotonic solution.

If we want to use a solution as a volume expansion we use saline, it will stay in the ECF.(Normal saline (NS)—often used to increase intravascular volume if the patient is dehydrated or has lost blood; usually not the best option in patients with CHF
unless the patient needs urgent resuscitation **)* step-up to medicine**

direction of fluid:

If we give 1000 ml NS it will go to : 250 ml IV 750 ml ISF 0 intracellular

Hyponatremia:

This refers to too much water in relation to sodium in the serum.

- **Normotonic or Isotonic Hyponatremia** (*Pseudohyponatremia,*

Factitious Hyponatremia):

An increase in plasma solids lowers the plasma sodium concentration. But the amount of sodium in plasma is normal (hence, **pseud**ohyponatremia). caused by any condition that leads to elevated protein or lipid levels.**(e.g. laboratory errors, b/c they measure the Na concentration in water phase of the plasma not all the plasma and factor it to the whole plasma)**

- **Hypertonic hyponatremia:**

presence of osmotic substances that cause an osmotic shift of water out of cells. These substances include:

- **Glucose—Hyperglycemia:** increases osmotic pressure, and water shifts from cells into ECF leading to a dilutional hyponatremia. [Na^+] declines by 1.6 mEq/L for each 100 mg/dL [5.6 mmol/L] increase in serum glucose.
- **Mannitol.**

- **Hypotonic Hyponatremia** (true hyponatremia):

1- Hypovolemic: **total body water decrease and even more deficit in total body sodium:**

- Low urine sodium(< 20 mEq/L): **extra renal losses** (e.g., diarrhea, vomiting, nasogastric suction, diaphoresis, third-spacing, burns, pancreatitis) of sodium containing fluid.

- High urine sodium (>20 mEq/L): **renal loss** (e.g., diuretic excess, decreased aldosterone (ACE inhibitors), Acute tubular necrosis).

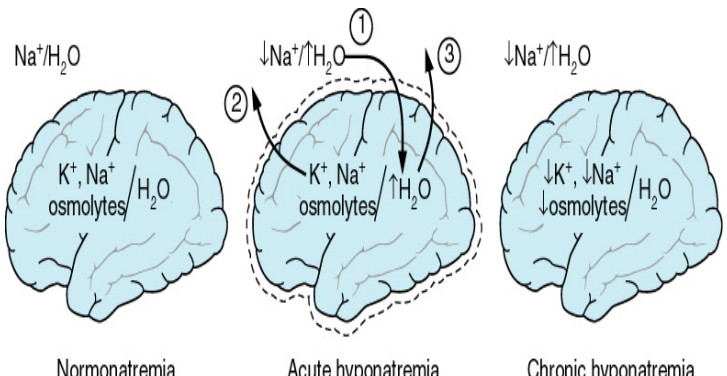
2- Euvolemic (no edema): no evidence of ECF expansion:

- SIADH(Syndrome of inappropriate antidiuretic hormone secretion): **excessive release of antidiuretic hormone result in dilutional hyponatremia in which the sodium remains normal but total body fluid increases.**
diagnosis of SIADH include: HIVE

- **H:** Hypoosmolar Hyponatremia (Posm <275 mOsm/Kg H2O)
- **I:** Inappropriate urine concentration (Uosm >100 mOsm/Kg H2O)
- **V:** Euvolemia, No diuretic use.
- **E:** Endocrine = normal Thyroid, adrenal and renal function.

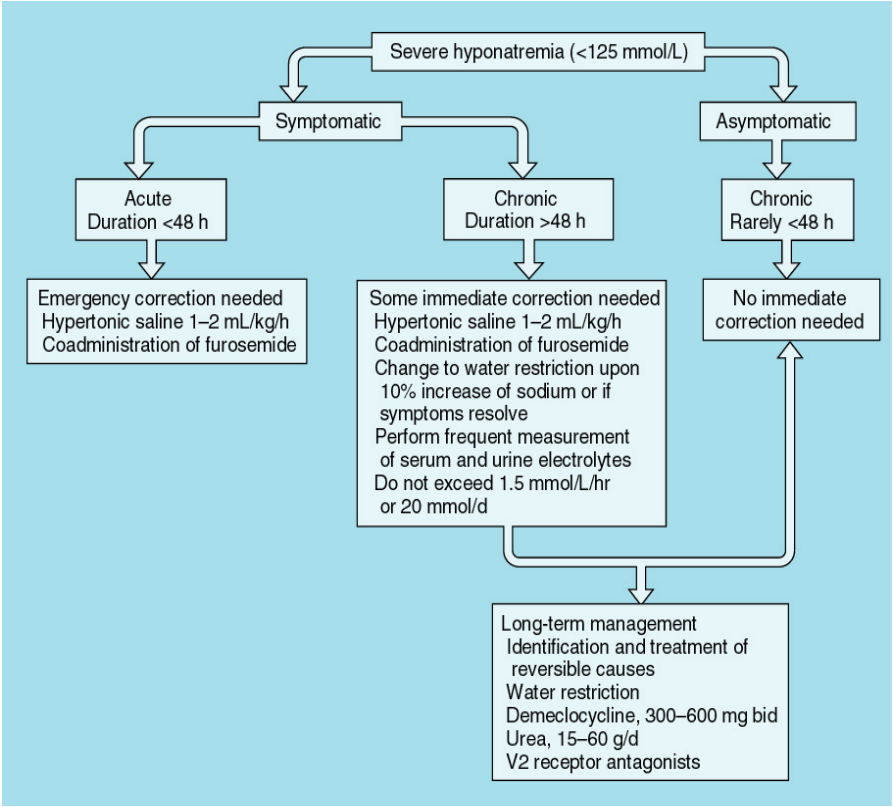
- Hypothyroidism.

3- Hypervolemic (low urine sodium): due to water-retaining states. The relative excess of water in relation to sodium results in hyponatremia.(e.g., CHF, Nephrotic syndrome, Liver disease).



Symptoms usually begin when the Na+ level falls to <120 mEq/L.
As ECF osmolality decreases, water shifts into brain cells, further increasing ICP resulting in a swollen brain causing seizures and coma.
In chronic hyponatremia(>48 h) the brain release some osmolytes. No sowllening happen.

Hyponatremia: Treatment:



Isotonic and hypertonic hyponatremias—Diagnose and treat the underlying disorder.

Hypotonic hyponatremia:

- Mild (Na+ 120 to -130 mmol/L)—Withhold free water, and allow the patient to re-equilibrate spontaneously.
- Moderate (Na+ 110 to 120 mmol/L)—loop diuretics (given with saline to prevent renal concentration of urine due to high ADH)
- Severe (Na+ < 110 mmol/L or if symptomatic)—Give hypertonic saline to increase serum sodium by 1 to 2 mEq/

step to medicine*

Hypernatremia:

Refers to excess sodium in relation to water; can result from water loss or sodium infusion.

1- Hypovolemic hypernatremia (sodium stores are depleted, but more water loss than sodium loss):

- Low urine sodium(< 20 mEq/L): **extra renal losses**(e.g., diarrhea, diaphoresis, respiratory losses).
- Low urine sodium(> 20 mEq/L): **renal loss**(e.g., diuretics, osmotic diuresis).

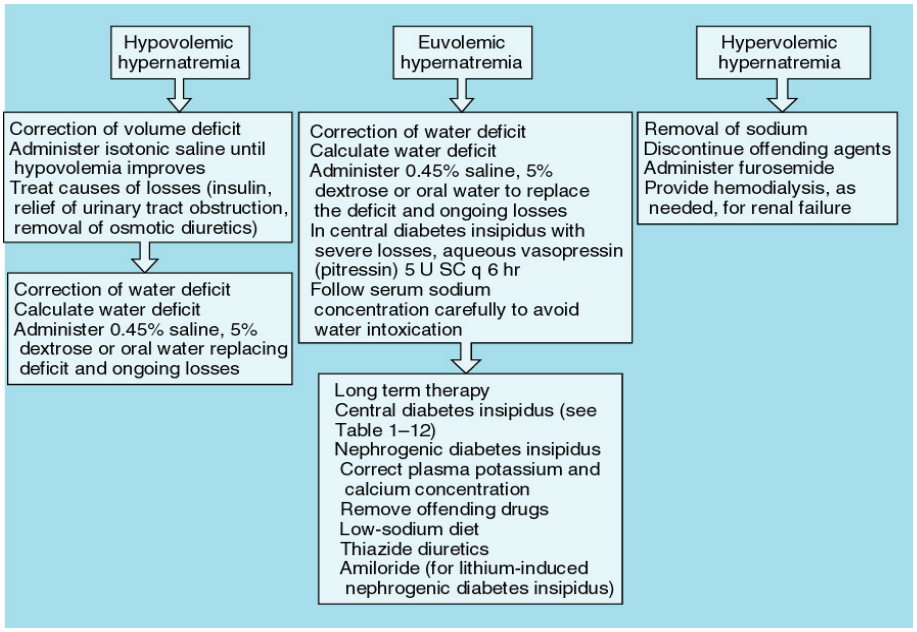
2- Euovolemic hypernatremia (sodium stores normal, water lost):

- **Diabetes insipidus .**
- Insensible respiratory (tachypnea).

3- Hypervolemic hypernatremia (sodium excess): occurs infrequently

- Iatrogenic—**most common cause.**(large amount of parenteral NaHCO3)
- Hyperaldosteronism
- Exogenous glucocorticoids

Hypernatremia Treatment:



1- Hypovolemic hypernatremia: isotonic NaCl to restore hemodynamics.

Correction of hypernatremia can wait until the patient is hemodynamically stable, then replace the free water deficit

2- Isovolemic hypernatremia: Patients with diabetes insipidus require vasopressin.

3- Hypervolemic hypernatremia: Give diuretics (furosemide) and D5W to remove excess sodium.

Potassium:

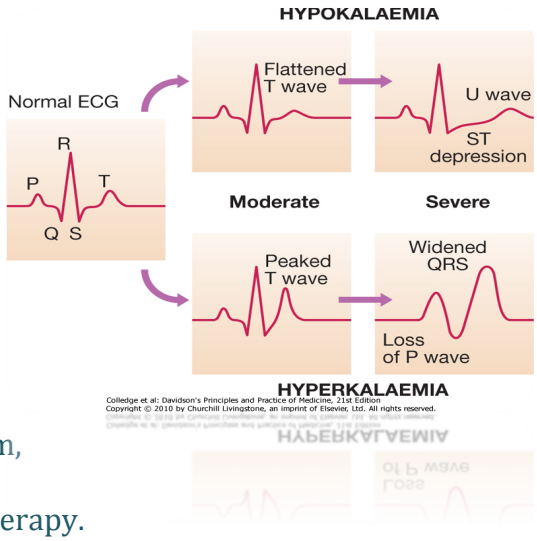
- 98% of the total potassium is in **ICF**
- Serum potassium is 3.5 –5.5 mmol/L
- Hypokalemia—Alkalosis and insulin administration may cause hypokalemia
- because they cause a shift of potassium into the cells.
- Hyperkalemia—Acidosis and anything resulting in cell lysis increase serum K+
    - (both force K+ out of cells into the ECF).

Function:

- Conduction of impulses
- Acid base balance
- Protein synthesis and carbohydrate metabolism

Potassium secretion is under multiple controls:

- Sodium load delivered to the kidneys
- Acid base status
- Potassium intake
- Aldosterone levels



Hypokalemia:

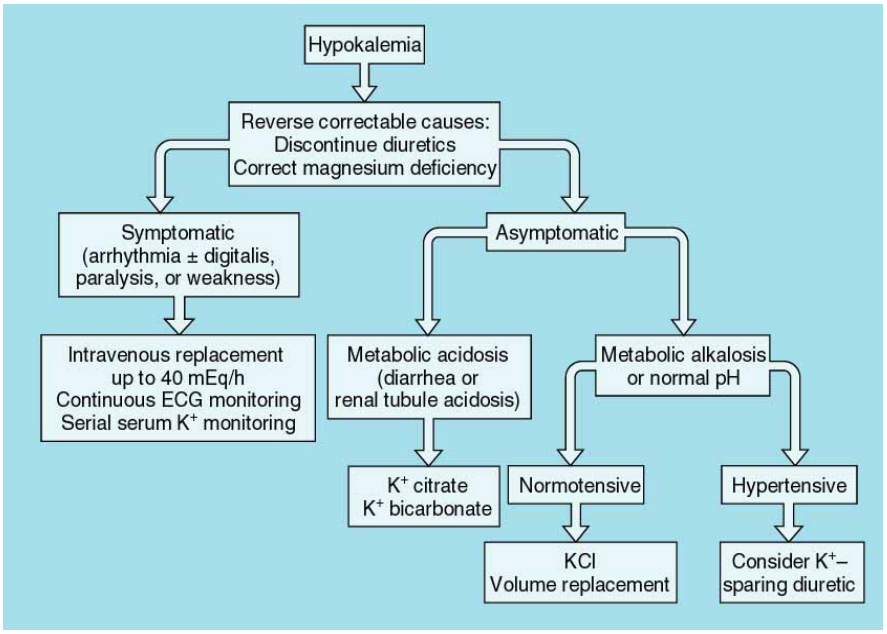
1- GI losses: with alkalosis , vomiting and nasogasteric Aspiration.
With acidosis, Diarrhea, Laxatives and enemas.
2- Renal losses: with hypertension, hyperaldosteronism, Excessive glucocorticoids.
3- Decrease k^+ intake : Insufficient dietary intake, i.v therapy.

4- Redistribution into cells: Insulin administration, alkalosis.

Hypokalemia: Clinical manifestations:

1. Cardiovascular: Arrhythmias, Digitalis toxicity
2. Neuromuscular:
    1. Smooth muscle: Ileus
    2. Skeletal muscle: Weakness, Paralysis, Rhabdomyolysis
    3. Endocrine: Glucose intolerance
3. Renal/electrolyte:
    1. Vasopressin resistance
    2. Increased ammonia production
    3. Metabolic alkalosis
    4. Structural changes: Renal cysts, Interstitial changes

Treatment of hypokalemia:



1- Identify and treat the underlying cause.
2-Discontinue any medications that can aggravate hypokalemia.
3-Oral KCl is the preferred (safest) method of replacement and is appropriate in
4-most instances. Always retest the K+ levels after administration:.
a. Using 10 mEq of KCl increases K+ levels by 0.1 mEq/L.
b. It comes in slow-acting and fast-acting forms.

Step-up to medicine

Hyperkalemia(the most dangerous and can be the most fatal):

Pseudohyperkalemia (spurious): **artificially elevated plasma K+ concentration due to K+ movement out of cells immediately before or after venipuncture.**

1. sample is not processed quickly, some red blood cells will **hemolyze** and cause spillage of K+ leading to a falsely elevated result.
2. leukocytosis and thrombocytosis.

Redistribution:

1. Acidosis
2. Insulin deficiency.(Insulin stimulates the Na+-K+-ATPase and causes K+ to shift into cells)
3. Rapid administration of β-blocker.
4. Sever hyperglycemia.

Renal retention of k+:

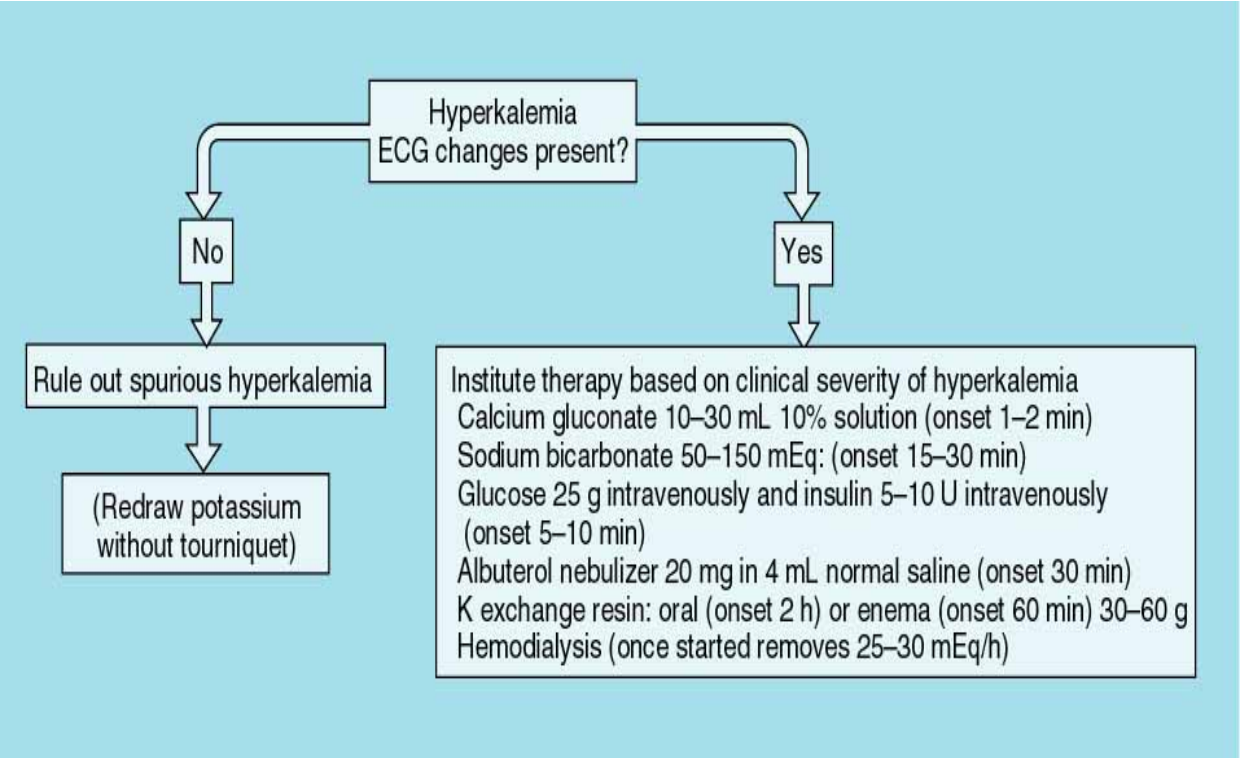
1- Renal failure (acute or chronic).
2- Tubular secretory failure:

3- With low aldosterone: Addison's disease, Hyporeninemic hypoaldosteronism, ACE inhibitors.
4- With normal- or high aldosterone: Potassium-sparing diuretics (spironolactone),pesduohypoaldosteronism, tubulointerstitial disease (SLE, renal transplant).

Hyperkalemia: Clinical manifestations:

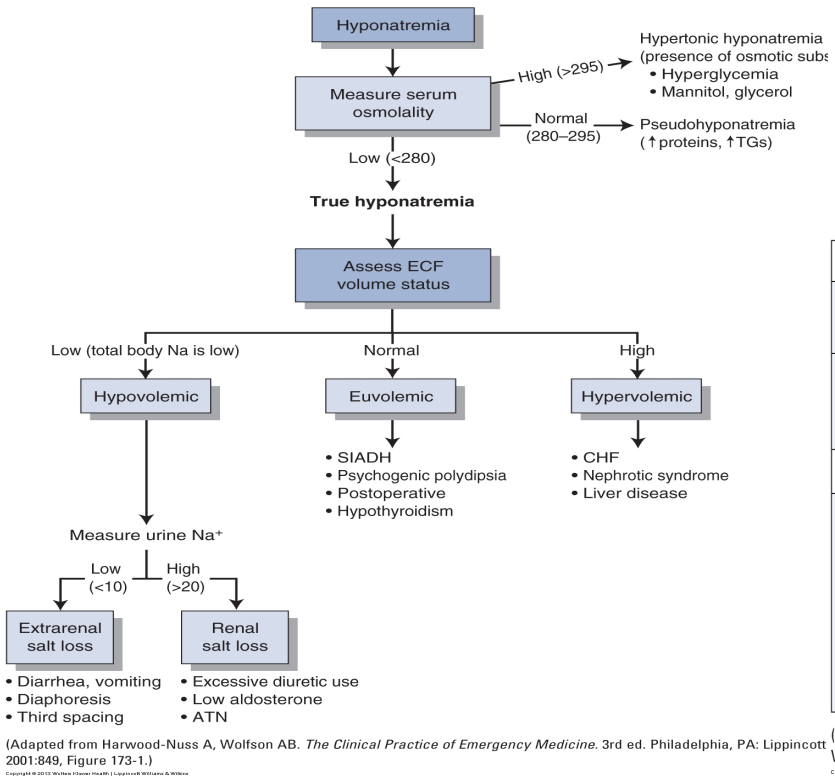
1- Cardiovascular:
T-wave abnormalities
Lengthened segments
Brady-arrhythmias
2- Neuromuscular:
Ileus
Paresthesias
Weakness
Paralysis
3- Renal/electrolyte:
Decreased ammonia production
Metabolic acidosis

Treatment of hyperkalemia:

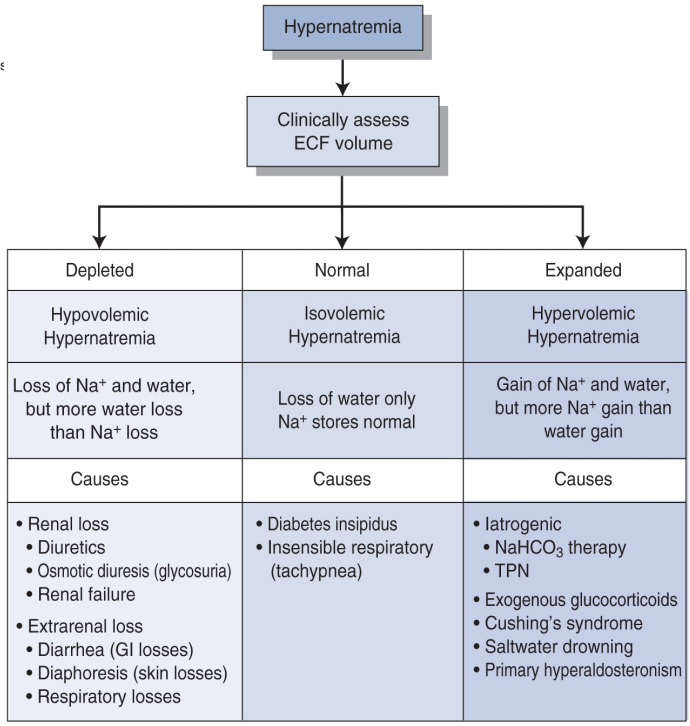


- calcium gluconate(stabilize the heart)
- Insulin(to shift the k+ inside the cells)
- Albuterol nublizer(beta agonist, shift k+ inside the cells)
- Sodium bicarbonate(to shift the k+ inside the cells)
- The main treatment is to get rid of k+:
  Through stool by resonium A
  Through urine by diuretics, or dialysis for patient does not have any urine output.

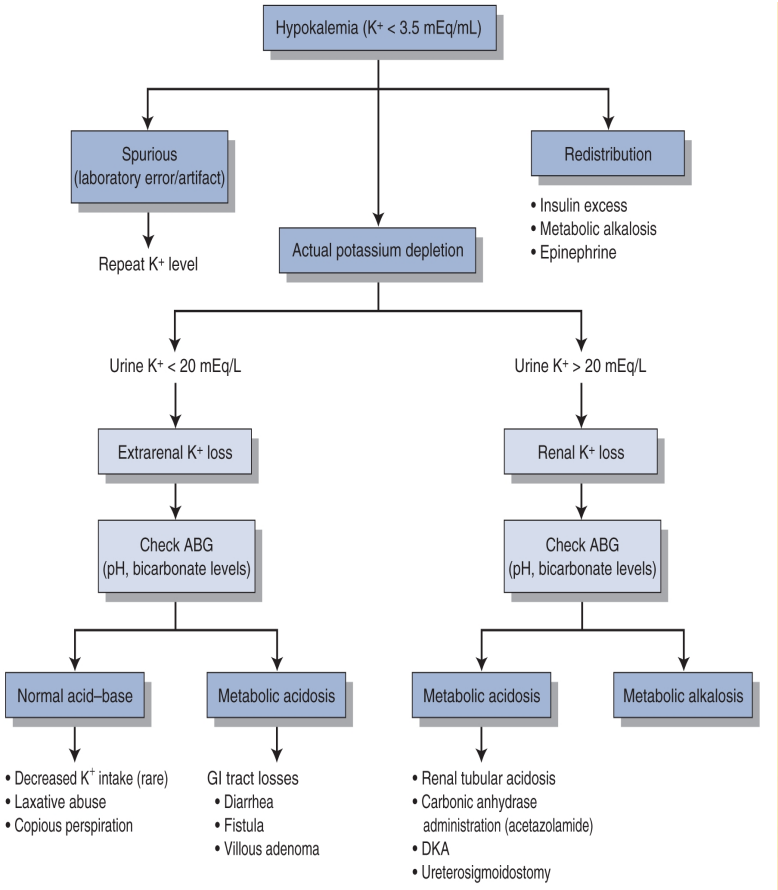
Summary :

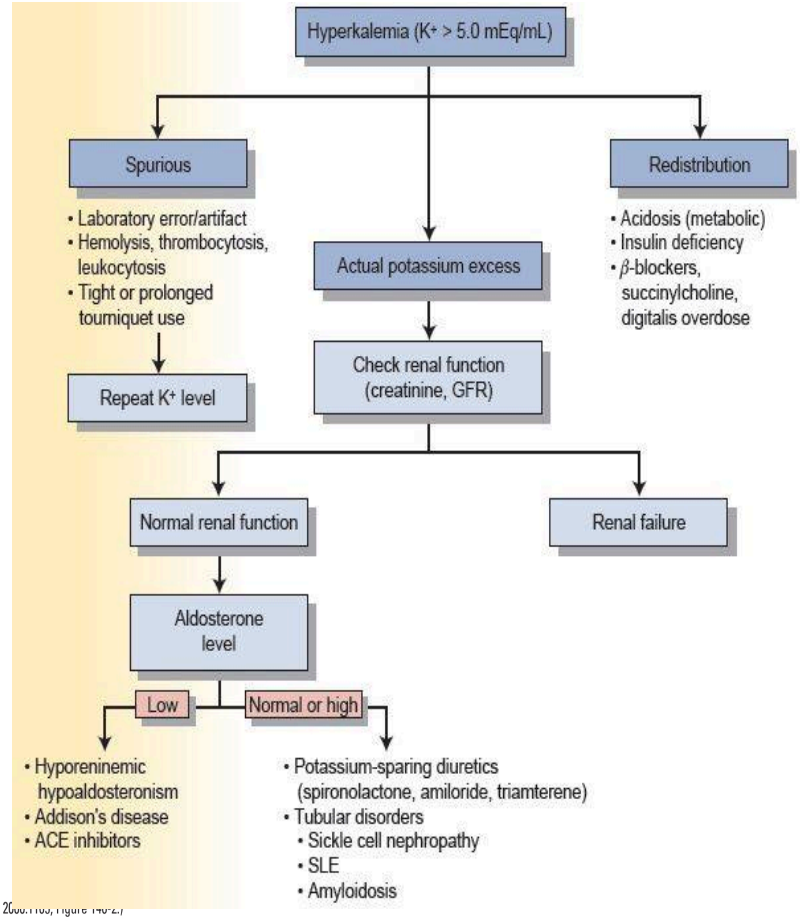


(Adapted from Harwood-Nuss A, Wolfson AB. *The Clinical Practice of Emergency Medicine.* 3rd ed. Philadelphia, PA: Lippincott 2001:849, Figure 173-1.)



(Adapted from Glasscock RJ, Massry SG. *Massry and Glassock's Textbook of Nephrology.* 4th ed. Philadelphia, PA: Lipp Williams & Wilkins, 2000.)





(Adapted from Humes DH, DuPont HL, Gardner LB, et al. *Kelley's Textbook of Internal Medicine.* 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2