Water and electrolyte disorders

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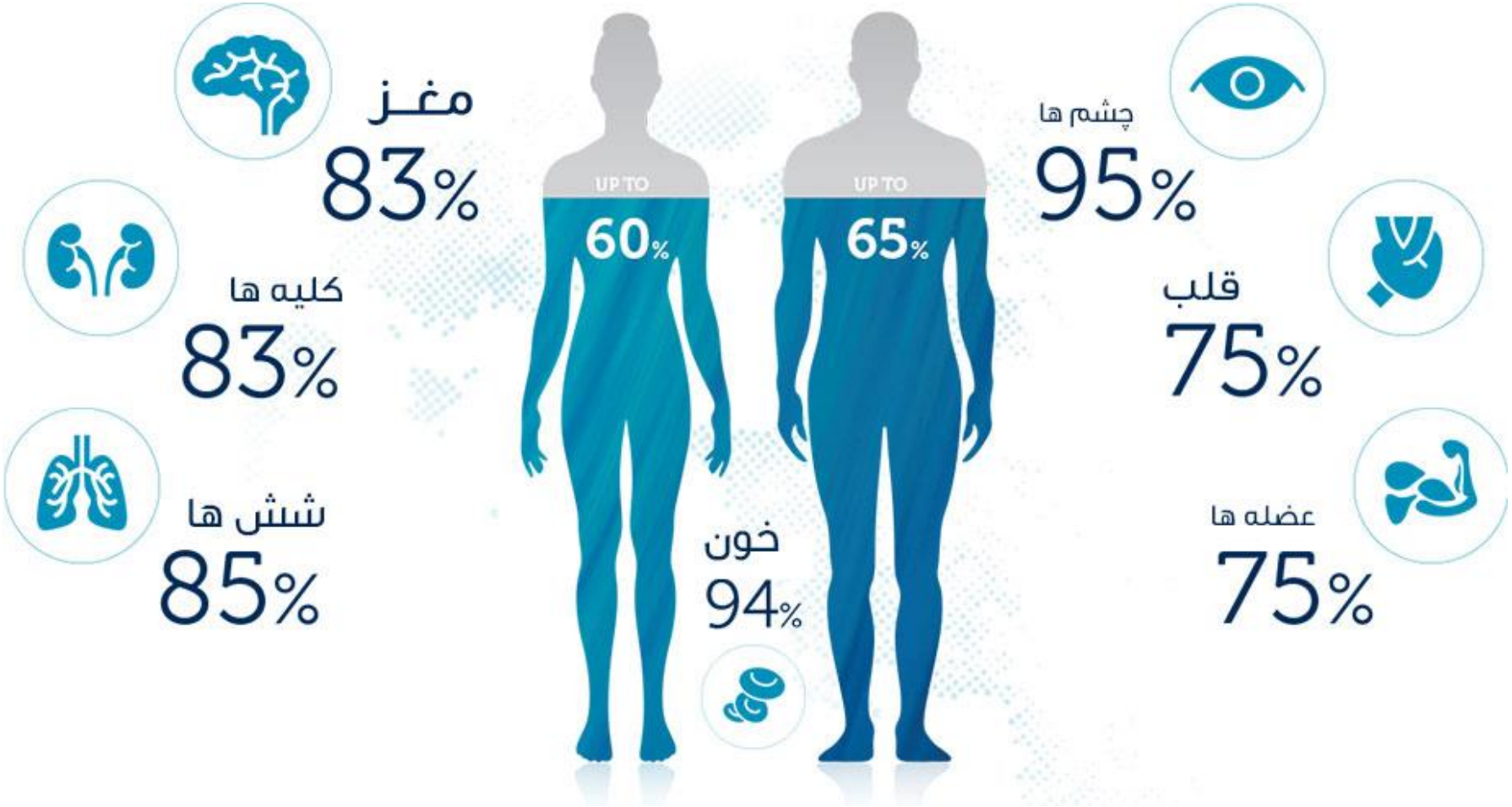
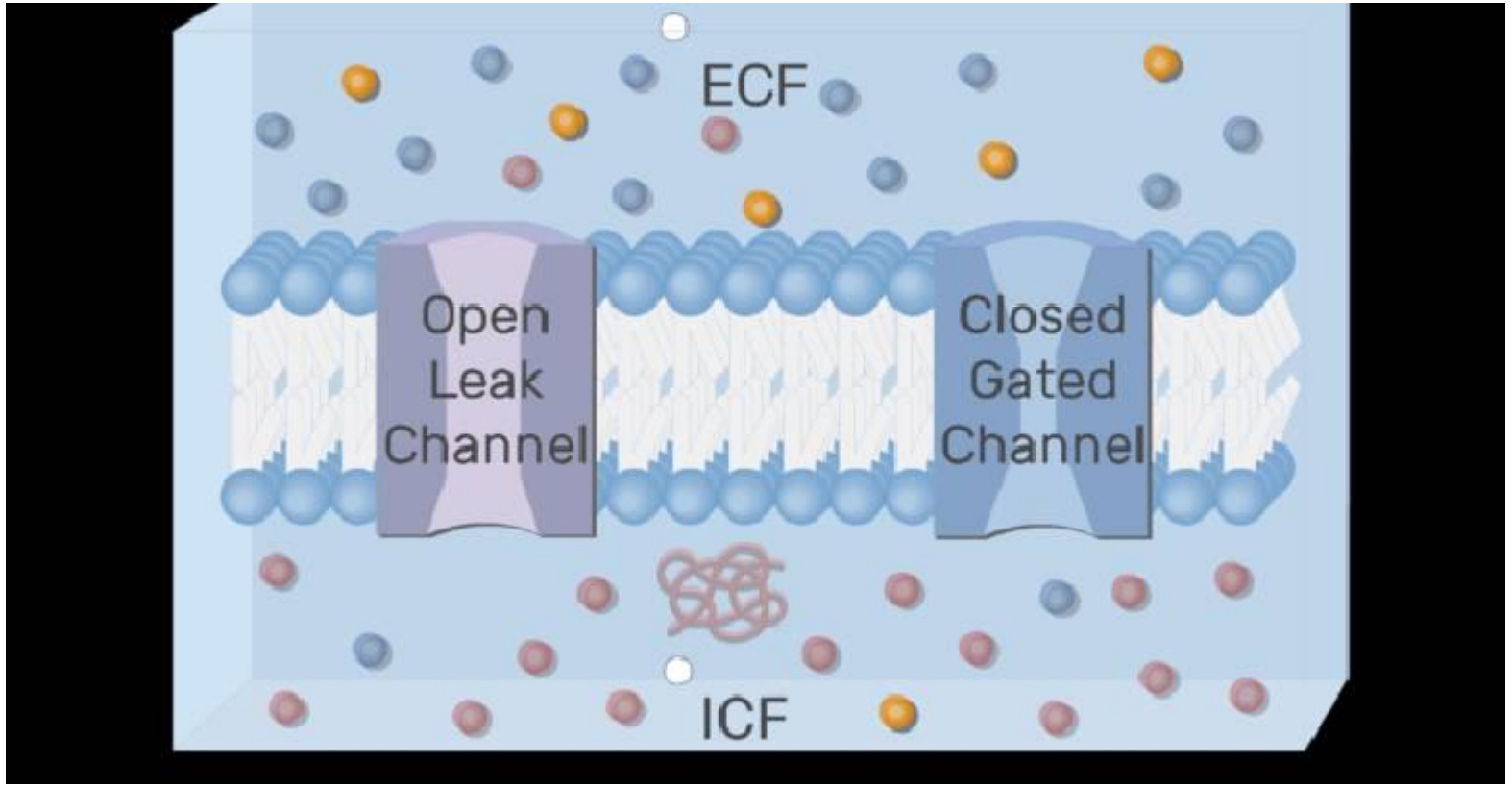
MA Special care nursing

Basic concepts



ICF @ ECF



Fluid Compartments

Intracellular fluid (**ICF**)

- Fluid inside the cell
- **Most** (2/3) of the body's **H20** is in the **ICF**.

Extracellular Fluid (ECF)

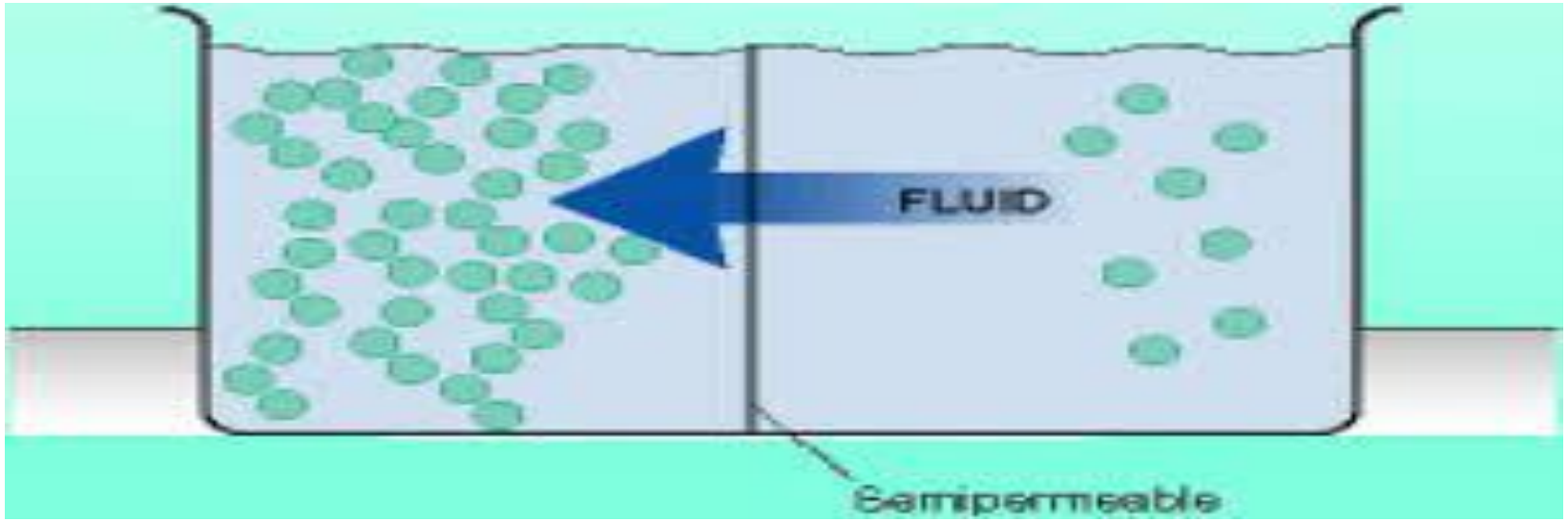
- Fluid outside the cell.
- **1/3** of body's **H20**
- More prone to loss
- 3 types:

Interstitial- fluid around/between cells

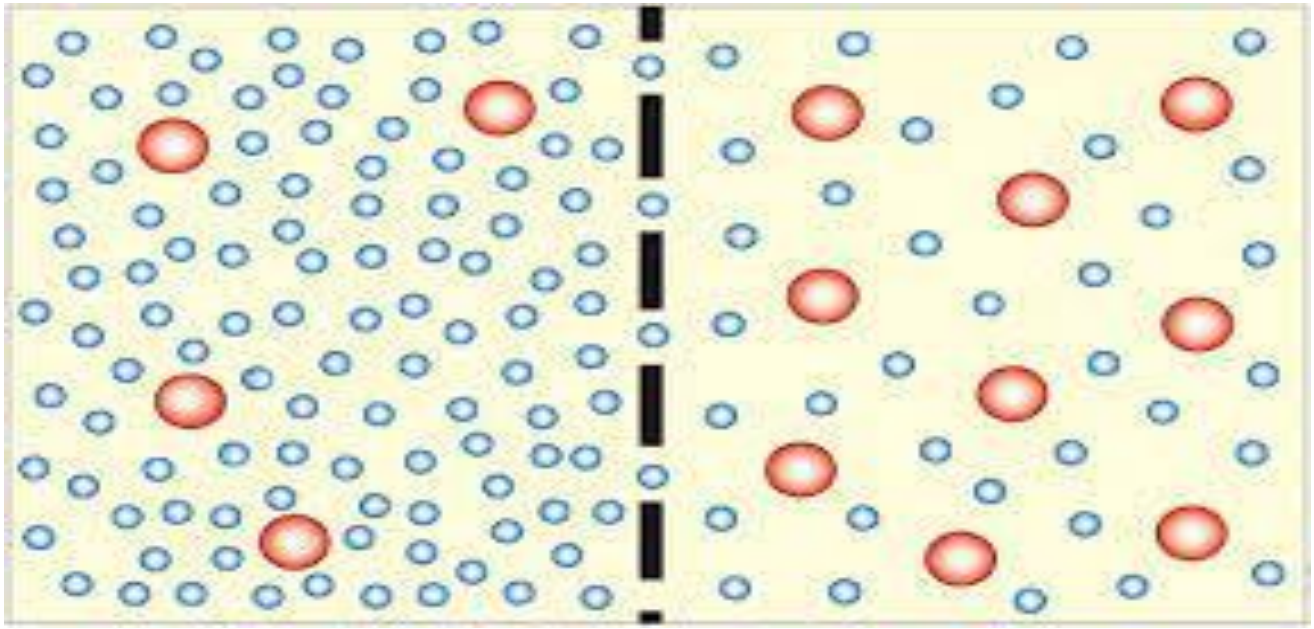
Intravascular- (plasma) fluid in blood vessels

Transcellular –CSF, Synovial fluid etc

osmolarity



The phenomenon of osmosis

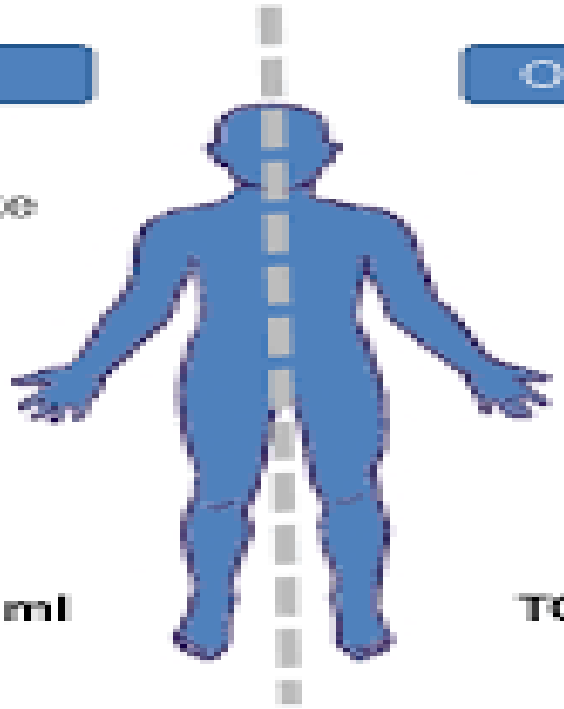


Average fluids consumed and excreted in adults on a daily basis

Table 19–1 WATER INTAKE AND OUTPUT

Form	Average Amount per 24 Hours (mL)
Intake	
Liquids	1600
Food	700
Metabolic water	200
Output	
Urine	1500
Sweat (and insensible water loss)	500
Exhaled air (water vapor)	300
Feces	200

Approximate daily values of fluid input and output in health



INPUTS	OUTPUTS
Oral fluid intake (~1500 ml)	Urine (~1500 ml)
Food (~500 ml)	Insensible loss - sweat - lungs - faeces (~1000 ml)
Metabolism (~500 ml)	
TOTAL ~ 2500 ml	**TOTAL ~ 2500 ml**

Ways of removing electrolytes from the body

- kidneys: General rule: 1ml/kg/H
- Skin: the actual amount of sweat 0-1000ML (imperceptible excretion 600ML)
- Lungs: 300 ML in unconscious state
- Digestion: 100-200 ML

Homeostatic mechanisms

- kidneys : Normally, in adults, 180 liters of plasma are filtered per day, of which 1 to 2 liters are converted into urine.
- Main functions:
  - Regulation of ECF volume and osmolality
  - Regulation of ECF electrolyte levels
  - PH adjustment

Cardiovascular

- It helps in the formation of urine by creating proper pressure in the kidneys
- Any weakness and heart failure causes disruption in blood supply to the kidneys and as a result, disruption in the regulation of water and electrolytes in the body.

lungs

- During daily exhalation, 300 ml of water is expelled from the lungs.

- Help regulate acid and base in the body

Pituitary gland

- The hypothalamus produces ADH, which is stored in the posterior part of the pituitary gland and released when needed.

Adrenal function

- Aldesterone: its increase helps to maintain sodium and as a result water retention and potassium excretion

- reducing Aldesterone  sodium and water excretion and maintaining potassium

Parathyroid gland function

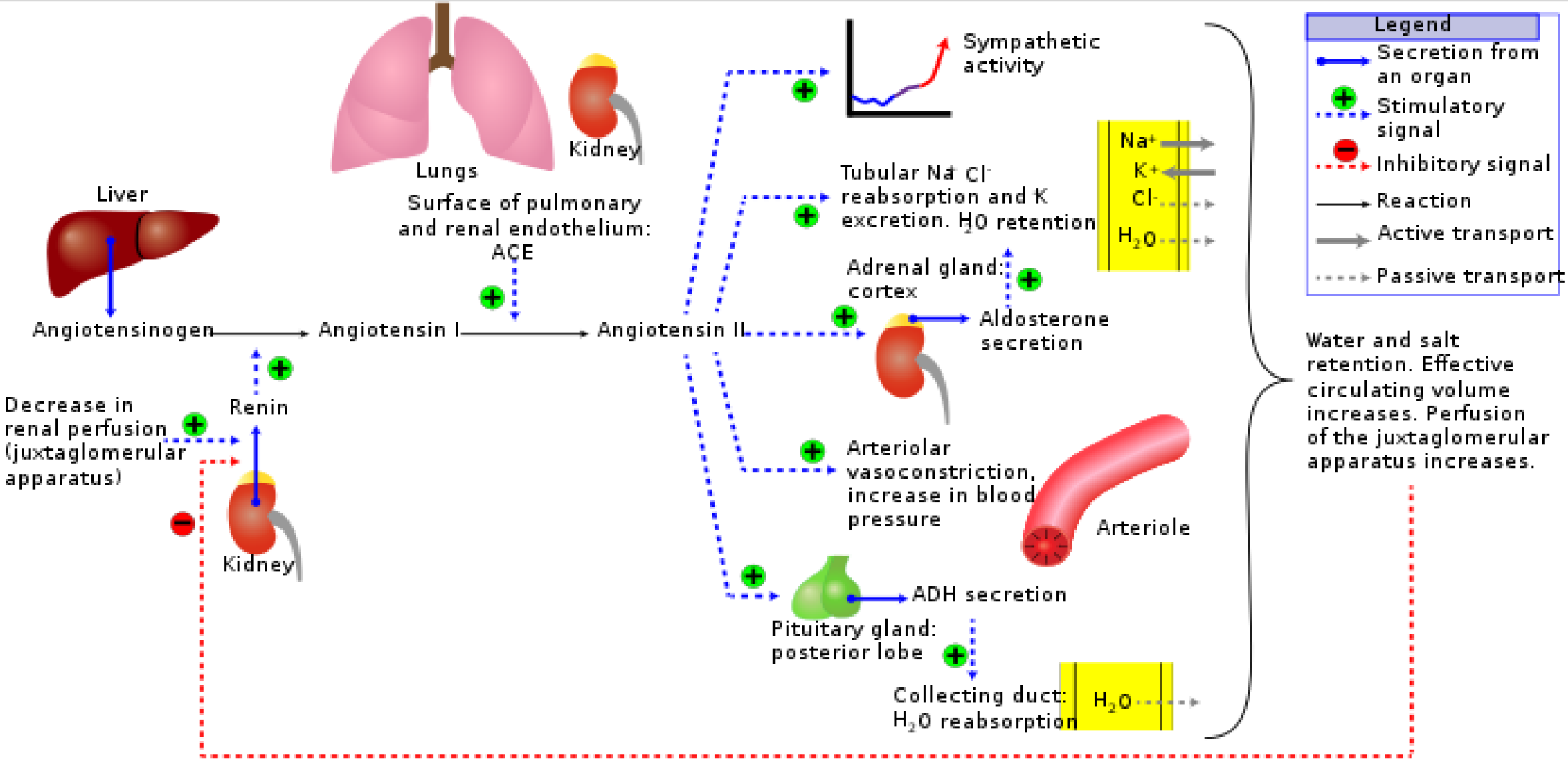
- The hormone PTH together with the thyroid regulates the balance of

calcium and phosphate

other mechanisms

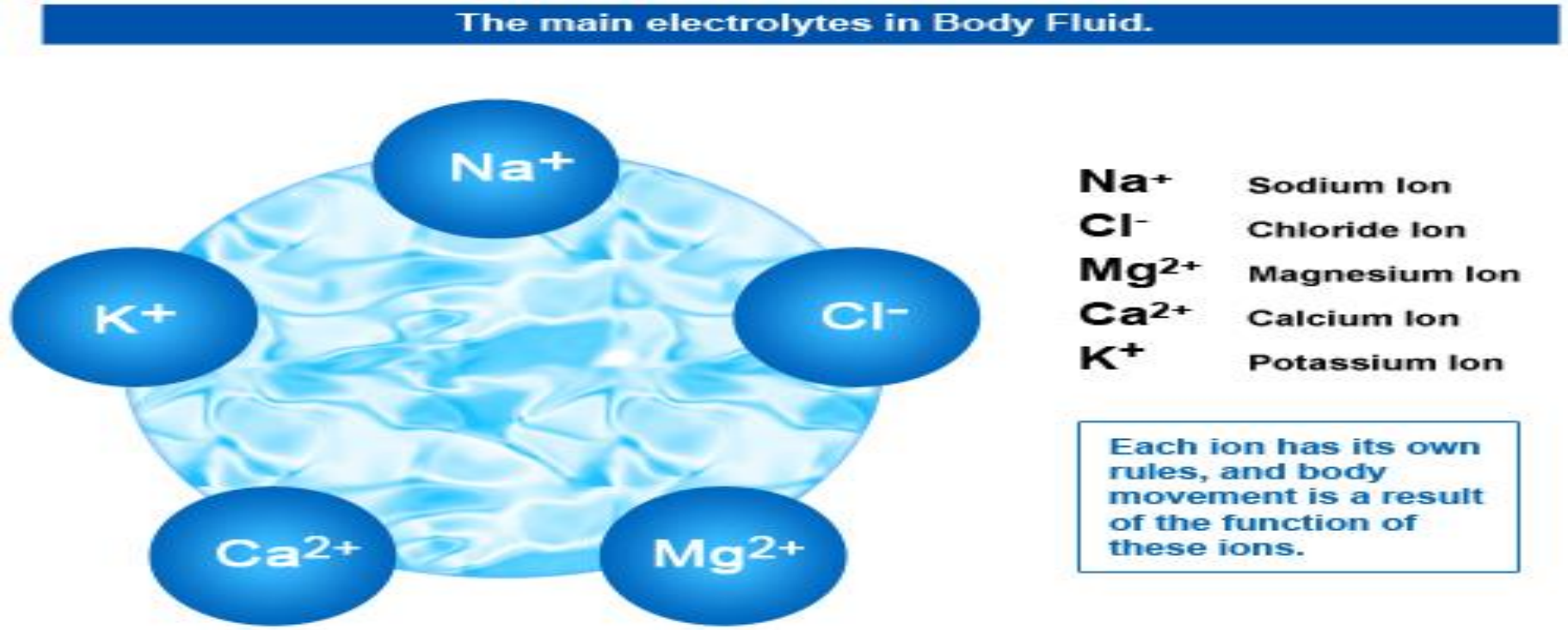
- Pressure receptors in the left atrium, carotid and aortic branches
- Renin-angiotensin-aldosterone system

Renin–angiotensin–aldosterone system



Body electrolytes

- Active chemical elements in the body
- Main cations: sodium, potassium, calcium and magnesium



Na

- The most abundant electrolyte of ECF
- Its concentration is 135-145.Meq/lit
- The first determinant of ECF osmolality and volumeIts
- regulation by thirst, ADH and renin-angiotensin-aldosterone system
- Sodium plays a role in stabilizing the electrical-chemical state necessary for muscle contraction or transmission of nerve waves

Hyponatremia

- Na < 135 meq/lit
- Hyponatremia is a secondary cause of osteoporosis.

- When evaluating patients, clinicians should categorize them according to their fluid volume status (hypovolemic hyponatremia, euvolemich hyponatremia, or hypervolemic hyponatremia).

- Our first question? What is the cause of sodium reduction?

- What makes hyponatremia scary?

- Management of hyponatremia varies depending on the severity of symptoms

Hypovolemic hyponatremia

- Sodium excretion is more than water excretion.
- Etiology: Losing too much sodium (diarrhea, vomiting, sweating)
- Prescribing diuretic drugs along with a low-salt diet.
- Aldosterone deficiency.

Hypervolemic hyponatremia

- Total body water is much more than body sodium. High ECV, (cirrhosis ,CHF)
- Peripheral edema and prominent JVP are its symptoms.

Isovolemic hyponatremia

- Total body water increases moderately, but total body sodium remains at normal levels.
- Etiology: Excessive water absorption, Excessive administration of dextrose water for injection, Inappropriate discharge of ADH, Lung tumors, Head trauma, Endocrine disorders

pathophysiology

- The changes that occur due to hyponatremia are caused by a decrease in the excitability of the membranes.
- When the level of sodium outside the cell decreases, this space will become hypoosmolar. Therefore, the fluid moves into the cell and causes cellular edema, which causes seizures.
- A 5% increase in cerebral cell volume causes an increase in ICP and a decrease in cerebral blood flow

Clinical manifestions

- A gradual decrease in sodium to 120 Meq/lit may not cause symptoms, but a sudden drop to this level can be life-threatening.
- Decreased skin turgor.
- dry mucus.
- Headache
- Decreased salivation.
- Decreased standing BP
- Nausea
- Abdominal cramps
- Neurological disorders (change in mental status, stable epilepsy and coma)
- Anorexia, muscle cramps, thirst

- When sodium falls below 115 mEq, symptoms of increased ICP include drowsiness, confusion, unilateral numbness, papilledema, convulsions, and death.

treatment

- Cautious administration of sodium (PO, NGT, IV)
- Type of IV fluid received (normal saline, ringer lactate)

- If there are neurological symptoms
- Administration of 3 to 5% sodium chloride hypertonic solution only in special care conditions and very cautiously and with small amounts
- The main goal in severe hyponatremia is to increase sodium only to the extent that neurological symptoms disappear

- US and European guidelines recommend treating severely symptomatic hyponatremia with bolus hypertonic saline to reverse hyponatremic encephalopathy by increasing the serum sodium level by 4 mEq/L to 6 mEq/L within 1 to 2 hours but by no more than 10 mEq/L (correction limit) within the first 24 hours. This treatment approach exceeds the correction limit in about 4.5% to 28% of people.
- Overly rapid correction of chronic hyponatremia may cause osmotic demyelination syndrome, a rare but severe neurological condition, which can result in parkinsonism, quadriparesis, or even death.

Hypernatremia

- Na > 145Meq/lit

Etiology:

- Unconscious patients
- heat stroke
- diabetes insipidus
- Hemodialysis
- Drowning in sea water
- Administration of hypertonic food with NGT without proper administration of water

Signs And symptoms

- Neurological symptoms due to cell dehydration include restlessness, weakness and delirium,
- thirst as a first symptom in alert patient
- dry tongue
- increased temperature.

treatment

- Gradual reduction of serum sodium level by hypotonic solutions
- Gradual reduction of serum sodium level prevents brain edema.
- If the cause of hypernatremia is diabetes insipidus, desmopressin spray is used.

K

- The main electrolyte inside the cell.
- It plays a role in nervous functions and the activity of skeletal and cardiac muscles.
- The kidney is the first regulator of potassium balance (80% of its excretion is renal and 20% through the intestines and sweat glands).
- Help balance acid and base

Hypokalemia

- K < 3.5 meq/lit

Etiology

- Medications such as diuretics and corticosteroids, ,salbutamol, epinephrine, insulin
- Increased secretion of aldesterone such as in cushings syndrome
- Vomiting and diarrhea
- Wound drainage particulary GI prolonged suctions
- Excessive diaphoresis
- Kidney disease
- Inadequate potassium intake
- Movement of k from ECF to ICF such as alkalosis,hyperinsulinism

Etiology

- Patients who are subject to a constant increase in insulin (patients who receive TPN).
- Water intoxication
- Iv therapy with potassium deficient solutions
- Magnesium deficiency

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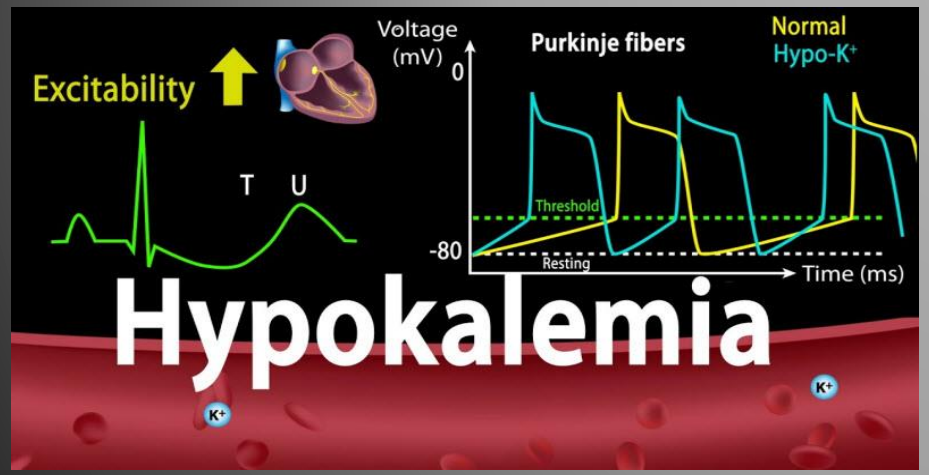
Assessment

- Cardiovascular
- irregular Pulse rate
- Peripheral pulse
- Orthostatic hypotension
- Electrocardiogram changes



- Respiratory
- Ineffective respiration
- Diminished breath sounds

- Neuromuscular
- Skeletal muscle weakness and leg cramps
- Deep tendon hyporeflexia
- Anxiety
- Lethargy
- Confusion
- Coma

Clinical manifestions

- Tiredness
- Anorexia
- nausea and vomiting
- muscle weakness
- Leg cramps
- Reduce bowel movements
- Sensory disorders
- Heart rhythm disorder
- In the long term, potassium deficiency causes a decrease in insulin release and as a result glucose intolerance

Treatment

- Oral and injectable treatment methods
- Foods high in potassium include: most fruits, vegetables, whole grains, milk and meats.

Nursing considerations

- Timely prediction (fatigue, anorexia, muscle weakness, reduced bowel movements, sensory disorders and heart rhythm disorders)
- Patients who take digitalis are at risk to potassium deficiency.
- Encouraging high-risk patients to consume foods containing potassium.
- Accurate control of I/O

- The most important complication of rapid potassium injection is ventricular fibrillation and cardiac arrest.
- Oral potassium supplements can cause small lesions in the intestine and even gastrointestinal bleeding, so the patient should be carefully examined.
- Examination of urinary status during intravenous administration of potassium. If urine decreases to less than 20 cc per hour in two consecutive hours, intravenous potassium should be stopped

Hyperkalemia

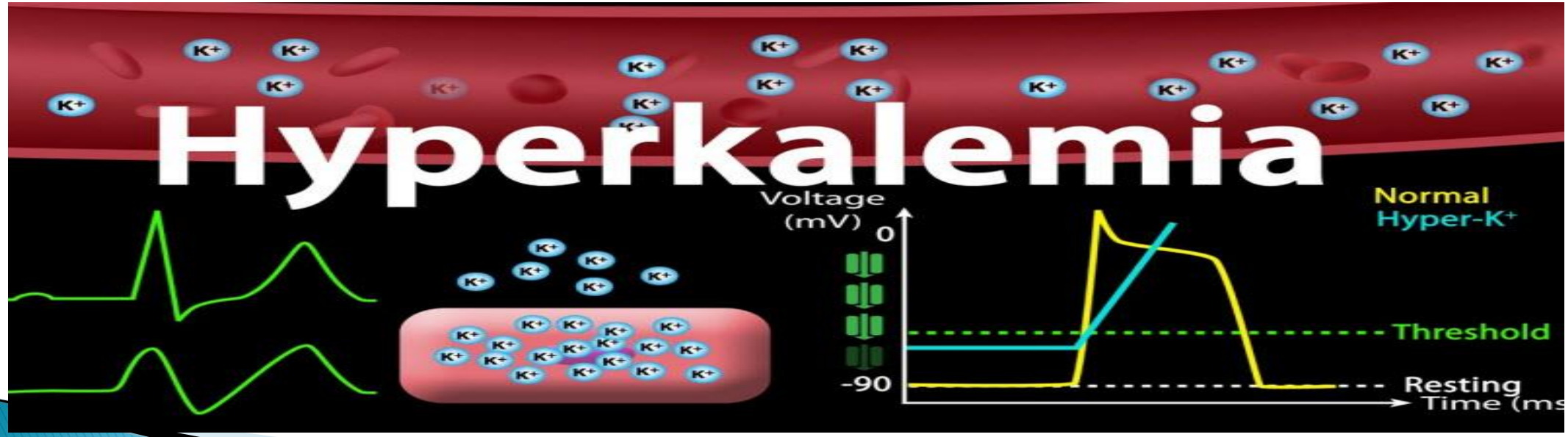
- K > 5MEq/l

Etiology

- Decreased renal excretion of potassium
- Rapid potassium infusion and movement of potassium from ICF to ECF
- Hypo aldosteronism and Addison's disease.
- Medications include (potassium chloride, heparin, ACE inhibitors, NSAIDS, potassium-sparing diuretics)
- Metabolic Acidosis
- Tissue damage
- Tighten the tourniquet
- Leukocytosis and thrombocytosis
- potassium shift outside the cell, severe infections, chemotherapy (due to the breakdown of malignant cells)

Clinical manifestations

- The most important consequence is the effect on the myocardium.
- Symptoms of muscle weakness and arrhythmia.
- k > 6 (beginning of changes in the cardiogram).



Treatment

- Limit potassium consumption in non-critical situations.
- In kidney disorders, the use of kayexalate.
- Bicarbonate sodium
- In cases of severe Hyperkalemia: calcium gluconate.
- Intravenous injection of regular insulin plus hypertonic dextrose.
- Types of loop diuretics.
- Hemodialysis or peritoneal dialysis

Nursing attentions

- Attention to the symptoms of muscle weakness and cardiac arrhythmia
- Paying attention to serum potassium, urea, creatinine and arterial blood gases.
- Diet restriction

- K oxalate binds with other cations in the digestive system, and in the event of hypomagnesemia and hypocalcaemia, it may cause an increase in fluid load and sodium retention. It may also cause intestinal perforation in patients with intestinal paralysis

calcium

- More than 99% of calcium is located in the skeletal system. About one percent of skeletal calcium has the ability to convert into blood calcium.
- Calcium plays a major role in the transmission of nerve impulses and helps regulate the contraction and relaxation of muscles, including the heart muscle.
- Another role in blood coagulation.
- The normal amount of serum calcium is 8.6-10.2 mg/dl
- If there is natural stomach acidity and vitamin D, calcium can be absorbed through food
- Major excretion through feces
- Regulation of serum calcium level by calcitonin and PTH

Hypocalcaemia

- Ca < 8.6 mg/dl

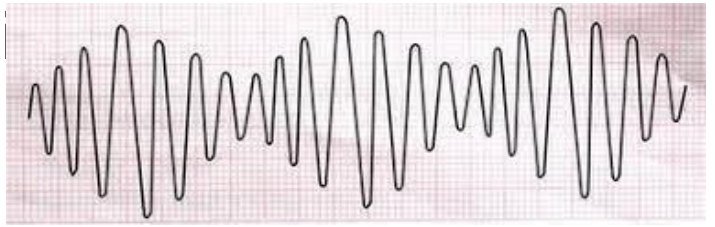
Etiology

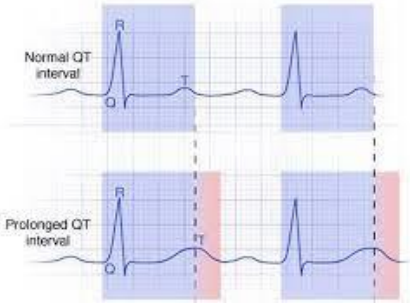
- Primary hypoparathyroidism.
- Administering too much blood containing citrate.
- Inflammation of the pancreas.
- Kidney failure
- Inadequate intake of vitamin D.
- Magnesium deficiency
- Central thyroid cancer
- Low levels of serum albumin
- Alkalosis and alcohol abuse
- Medicines (corticosteroids, phosphates, isoniazid, cyclic diuretics, aminoglycosides)

Clinical manifestions

- Tetany
- Truso's sign
- Shustok's sign
- Convulsions

- Other symptoms include mental changes, dizziness, depression, delirium and even hallucinations
- larynx spasm
- ECG changes and Torsade de Po





Signs and symptoms of chronic hypocalcaemia

- Increase in bowel movements
- Dry and brittle hair and nails
- Abnormalization of the clotting process
- Osteoporosis

Diagnosis

- Evaluation of serum calcium level (of course, serum albumin level and pH should also be considered).

treatment

- Intravenous calcium injection
- Vitamin D treatment
- Foods containing calcium (milk, green leafy vegetables, canned salmon)

Nursing attentions

- Pay attention to symptoms
- Warning to patients who use laxatives and antacids containing phosphorus

Hypercalemia

- Ca > 10.2mg/dl

Etiology

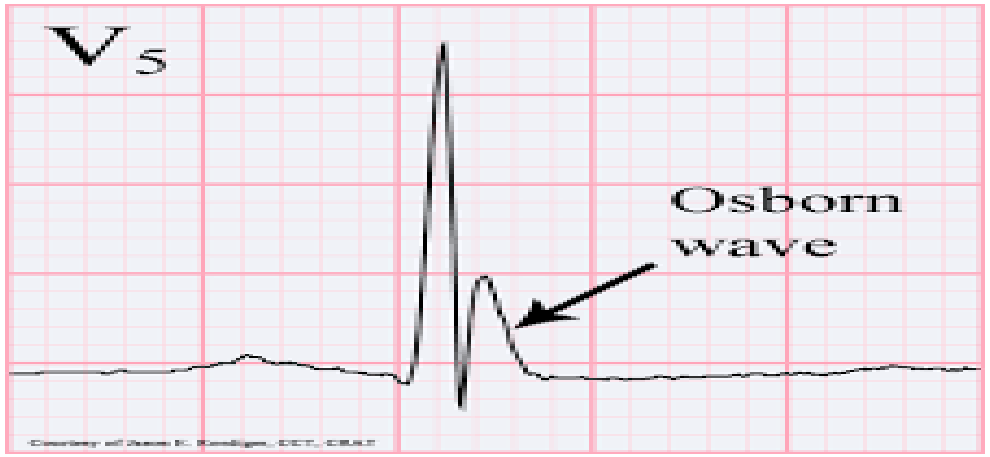
- The most common causes of Hypercalemia are:
- Malignancies
- Increased PTH
- Immobility after severe fractures
- Poisoning with vitamins A, D

Clinical manifestations

- Anorexia, nausea and vomiting and severe constipation
- Confusion and memory impairment and sleepiness
- Extreme thirst and excessive urination, abdominal cramps

Diagnosis

- Ca > 10.2 mg.dl
- ECG changes
- Salikovich's urine test



Treatment

- Treatment of root causes
- Drug treatment (intravenous injection of normal saline solution) Administration of intravenous phosphate
- calcitonin

Nursing attentions

- Encourage movement and drinking fluids
- Encourage the consumption of liquids containing sodium
- Investigating digital poisoning

Assessment findings: hypo and hyper Ca

Hypocalcemia	hypercalcemia
Cardiovascular: decreased HR – hypotension–	Cardiovascular: increased HR in the early phase. Brady that can lead to arrest in late phases. Increased BP
Respiratory: not directly affected.	Ineffective as result skeletal muscle weakness
Neuromuscular: irritable skeletal muscles: twitches, cramps, Tetany, seizures, painful muscle spasms. Paresthesias followed by numbness that may affect the lips, nose– trousseaus and chvosteks signs. – Hyperactive DTR– anxiety, irritability	Muscle weakness – diminished or absent DTR Disorientation, lethargy, coma
Gastrointestinal: increased gastric motility; hyperactive bowel sounds– cramping – diarrhea	decreased gastric motility; hypoactive bowel sounds– anorexia, nausea, abdominal distention. Constipation
Electrocardiographic changes: prolonged QT interval	Electrocardiographic changes: shortened QT interval widened T wave, heart block

Magnesium

- After potassium, magnesium is the most abundant intracellular cation.
- Magnesium plays an activating role in many intracellular enzyme systems and plays a role in the metabolism of proteins and carbohydrates.
- Magnesium works directly at the nerve-muscle junction.
- The role of peripheral vasodilatation
- Magnesium excretion is mostly renal
- Our normal level is between 1.3 – 2.3 mg.dl

Hypomagnesaemia

- Mg < 1.3mg.dl

Etiology

- The most important route of magnesium loss is the gastrointestinal tract Other causes include drugs (diuretics, aminoglycosides, cyclosporine, etc.), blood transfusion containing citrate) and diabetic ketoacidosis.

Clinical manifestations

- Neuromuscular changes include increased excitability with muscle weakness, tremors, and slow and involuntary twisting movements (athetosis).
- Tetany, nystagmus, vertigo, tonic–clonic convulsions, laryngeal congestion and symptoms (Shustok's and Trousseau, partly caused by hypocalcaemia)
- Apathy, depression and anxiety
- QRS widening and Torsade de pointes

Diagnosis

- In the laboratory analysis, the amount of Mg $<$ 1.3 mg.dl

Treatment

- A mild magnesium deficiency can only be compensated through a diet that includes leafy vegetables, nuts, grains, seafood, chocolate, and peanut butter.
- Intravenous injection of magnesium sulfate
- Urine output before, after and during magnesium injection is necessary, and if the volume of urine is less than 100 ml within 4 hours, the attending physician should be informed

Nursing attentions

- Pay attention to signs and symptoms
- Safety precautions in hypomagnesemia patients due to dizziness and convulsions
- Paying attention to digitalis consuming patients because magnesium deficiency in these patients provides the basis for digitalis poisoning.
- Checking patients' swallowing before giving food

Hypermagnesaemia

a rare disorder because the kidneys usually excrete magnesium in an adequate amount and it falsely rises during the lysis of the blood sample and tightening the tourniquet tightly and for a long time.

Etiology

- The most common cause of Hypermagnesaemia is kidney failure
- Excessive administration of magnesium in pregnancy hypertension

Clinical manifestions

- Stagnation of neuromuscular activities and deep tendon reflexes
- Low blood pressure
- Nausea and vomiting and tissue calcification
- Facial flushing
- Problems in speech (dysarthritis)
- lethargy
- Muscle weakness and paralysis
- In high amounts heart block and coma

Diagnosis

- MG > 2.3 mg.dl
- At the same time, there is an increase in potassium and calcium

Levels

ECG changes (prolongation of PR interval, lengthening of T waves and widening of QRS and atrio-ventricular block)

Treatment

- Avoiding magnesium administration in kidney failure patients In emergency cases such as respiratory depression and arrhythmic disorders, calcium gluconate injection
- Performing Hemodialysis with magnesium-free liquid
- In patients with adequate renal function, administration of cyclic diuretics, sodium chloride and ringer lactate

Nursing attentions

- Monitor vital signs
- DTR check
- Control I.O

Assessment findings: hypo and hyper MG

Hypomagnesemia	Hypermagnesaemia
Cardiovascular: tachycardia – hypertension	Cardiovascular: bradycardia – dysrhythmias– hypotension
Respiratory : shallow respiration	Respiratory : respiratory insufficiency when the skeletal muscles of respiration are involved.
Neuromuscular: twitches– paresthesias – positive trousseaus and chvostecks sign – Tetany– seizure	Neuromuscular: diminished or absent deep tendon reflexes – skeletal muscle wekness
Central nervous system: irritability– confusion	Central nervous system: drowsiness and lethargy hat progresses to coma
Electrocardiographic changes : tall T waves. Depressed ST segment.	Electrocardiographic changes: prolonged PR interval, widened QRS complexes.

References

- Kaplan medical. Clinical internal medicine review. 2023
- NCLEX- RN. Linda A silvestri @ Angela E silvestri. 8 edition. 2020
- Joseph G verbalis, julianna barsony and caulige.Hyponatremia – induced osteoporosis. 2010
- Sikarin u, anawin s. association between hyponatremia, osteoprosis, and fracture 2016.
- Brunner and suddarths 2018 12 edition