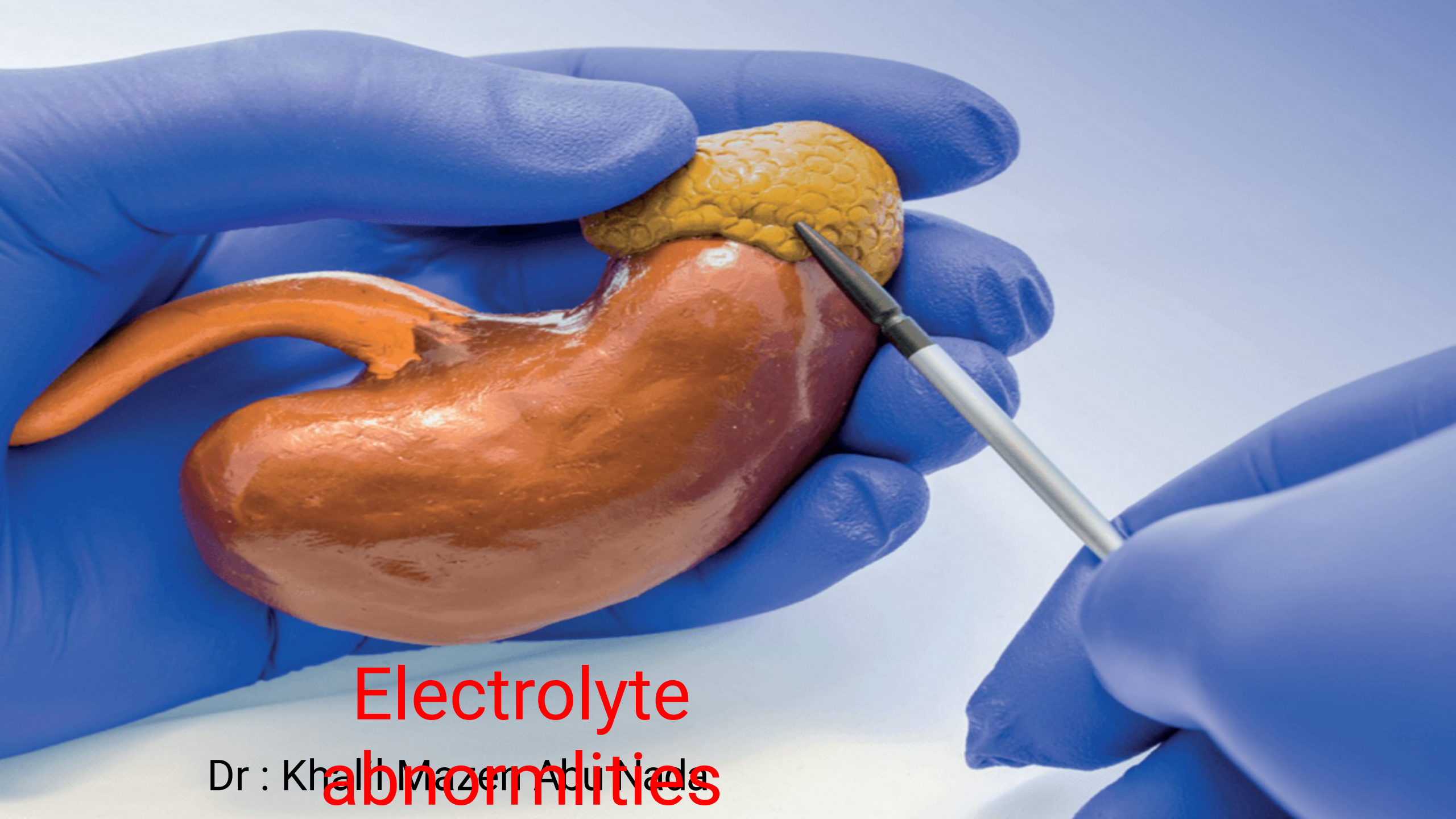


Electrolyte abnormalities

Dr : Khalil Mazen Abu Naeda

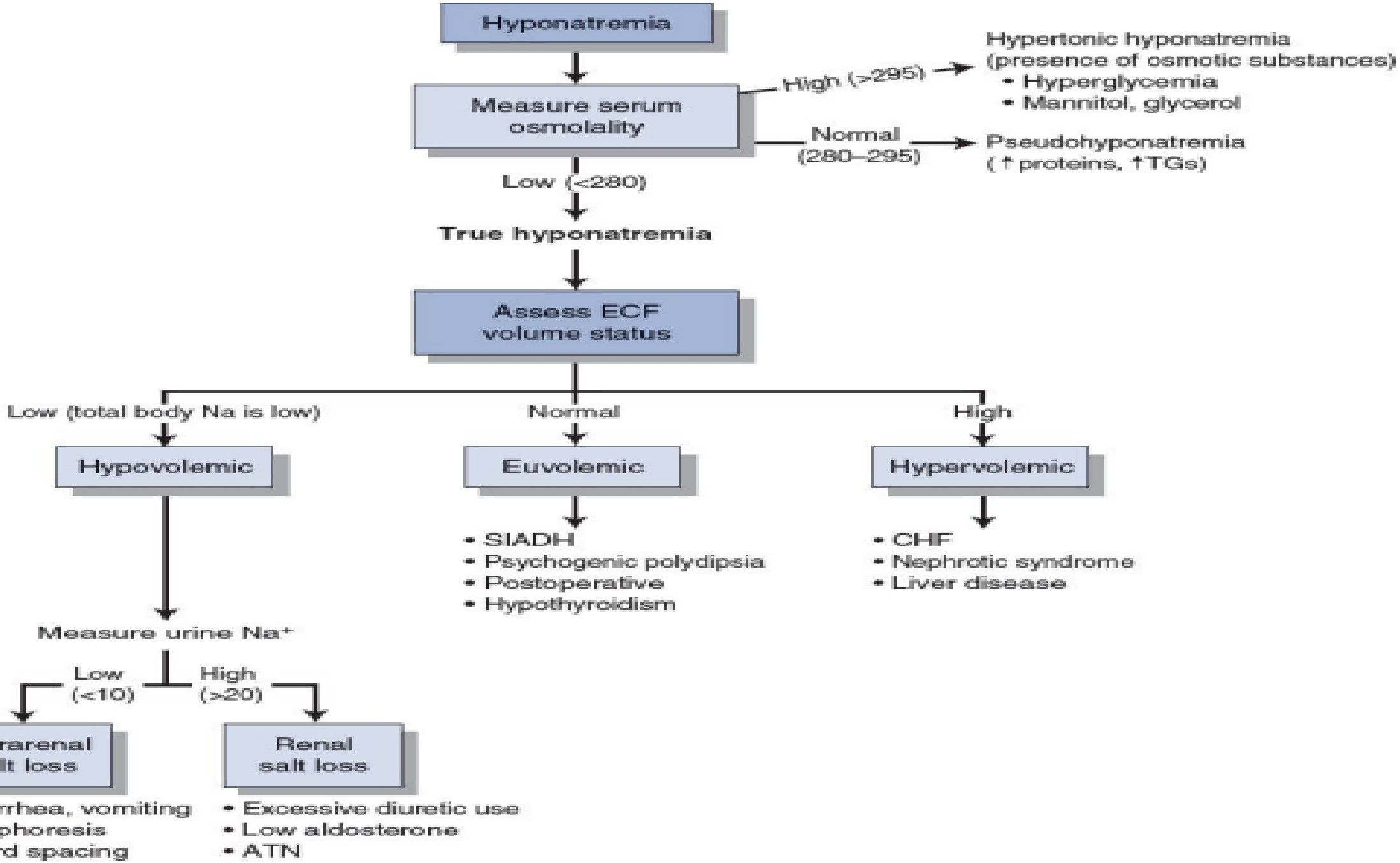
Hyponatremia

General Characteristics

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

2. It is typically defined as a plasma Na+ concentration <135 mmol/L.

3. Symptoms usually begin when the Na+ level falls to <120 mEq/L). As ECF osmolality decreases, water shifts into brain cells, further increasing ICP. (Therefore, it is critical to keep serum sodium normal or slightly high in such patients.)


Hyponatremia
Measure serum osmolality
High (>295)
Hypertonic hyponatremia (presence of osmotic substances)
• Hyperglycemia
• Mannitol, glycerol
Normal (280–295)
Pseudohyponatremia (↑ proteins, ↑ TGs)
Low (<280)
True hyponatremia
Assess ECF volume status
Low (total body Na is low)
Normal
High
Hypovolemic
Euvolemic
Hypervolemic
• SIADH
• Psychogenic polydipsia
• Postoperative
• Hypothyroidism
• CHF
• Nephrotic syndrome
• Liver disease
Measure urine Na+
Low (<10)
High (>20)
Extrarenal salt loss
Renal salt loss
• Diarrhea, vomiting
• Diaphoresis
• Third spacing
• Excessive diuretic use
• Low aldosterone
• ATN

Causes and Classification (based on serum osmolality)

1. Hypertonic hyponatremia

a. Caused by the presence of osmotic substances that cause an osmotic shift of water out of cells. These substances cannot cross the cell membrane and therefore create osmotic gradients.

b. These substances include:

_ **Glucose**—Hyperglycemia increases osmotic pressure, and water shifts from cells into ECF leading to a dilutional hyponatremia. For every 100 mg/dL increase in blood glucose level above normal, the serum sodium level decreases about 3 mEq/L.

Note that the actual sodium content in the ECF is unchanged.

_ **Mannitol, sorbitol, glycerol, maltose**

_ **Radiocontrast agents**

2. Isotonic hyponatremia (pseudohyponatremia)

A . An increase in **plasma solids (e.g., proteins or lipids)** artificially lowers the plasma sodium concentration measurement via laboratory artifact, but the amount of sodium in plasma is **normal** (hence, pseudohyponatremia).

b. This can be caused by any condition that leads to elevated protein or lipid levels

3. Hypotonic hyponatremia—"true hyponatremia"— serum osmolality <280 mOsm/kg

a. Hypovolemic

_ **Low urine sodium (<10 mEq/L)—**implies increased sodium retention by the kidneys to compensate for extrarenal losses (e.g., diarrhea, vomiting, nasogastric suction, diaphoresis, pancreatitis) of sodium containing fluid.

_ **High urine sodium (>20 mEq/L)—**renal salt loss is likely due to tubular dysfunction—for example, diuretic excess, decreased aldosterone (causes include ACE inhibitors and ARBs), ATN.

b. Euvolemic —no evidence of ECF expansion or contraction on clinical grounds

_ SIADH:

_ Hypothyroidism: mechanism not fully understood

_ Adrenal insufficiency :

_ Psychogenic polydipsia

C_ Hypervolemic (low urine sodium)— This is due to water-retaining states.

_ The relative excess of water in relation to sodium results in hyponatremia.

_ CHF

_ Nephrotic syndrome (renal failure)

_ Liver disease

Clinical Features

1. Neurologic symptoms predominate—caused by “water intoxication”—osmotic water shifts, which leads to increased ICF volume, specifically brain cell swelling or cerebral edema
    a. Headache, delirium, irritability
    b. Muscle twitching, weakness
    c. Hyperactive deep tendon reflexes

2. Increased ICP, seizures, coma

3. GI—nausea, vomiting, watery diarrhea

4. Cardiovascular—hypertension due to increased ICP

5. Increased salivation and lacrimation

6. Oliguria progressing to anuria—may not be reversible if therapy is delayed and acute renal failure develops

Diagnosis

1. Plasma osmolality—low in a patient with true hyponatremia

2 . Urine sodium concentration

Treatment

1. Isotonic and hypertonic hyponatremias—diagnose and treat the underlying disorder.

2. Hypotonic hyponatremia.

a. Mild (Na+ 120 to 135 mmol/L)

—withhold free water, and allow the patient to reequilibrate spontaneously.

__ In SIADH, salt tablets can also be used.

b. Moderate (Na+ 110 to 120 mmol/L)—loop diuretics

c. Severe (Na+ <110 mmol/L) or if symptomatic —give hypertonic saline to increase serum sodium by 1 to 2 mEq/L/hr until symptoms improve.

_ Rate of correction: Maximum 8 mEq/L in 24 hours to prevent overcorrection and osmotic demyelination syndrome, however in symptomatic patients aim for correcting 4 to 6 mEq/L in first 6 hours.

Hypernatremia

General Characteristics

1. Defined as a plasma Na+ concentration >145 mmol/L

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

3. Assess ECF volume clinically, as follows :

a. Hypovolemic hypernatremia (sodium stores are depleted, but relatively more water has been lost)

_ Renal loss—from diuretics, osmotic diuresis (most commonly due to glycosuria in diabetics), renal failure

_ Extrarenal loss—from diarrhea, diaphoresis, respiratory losses

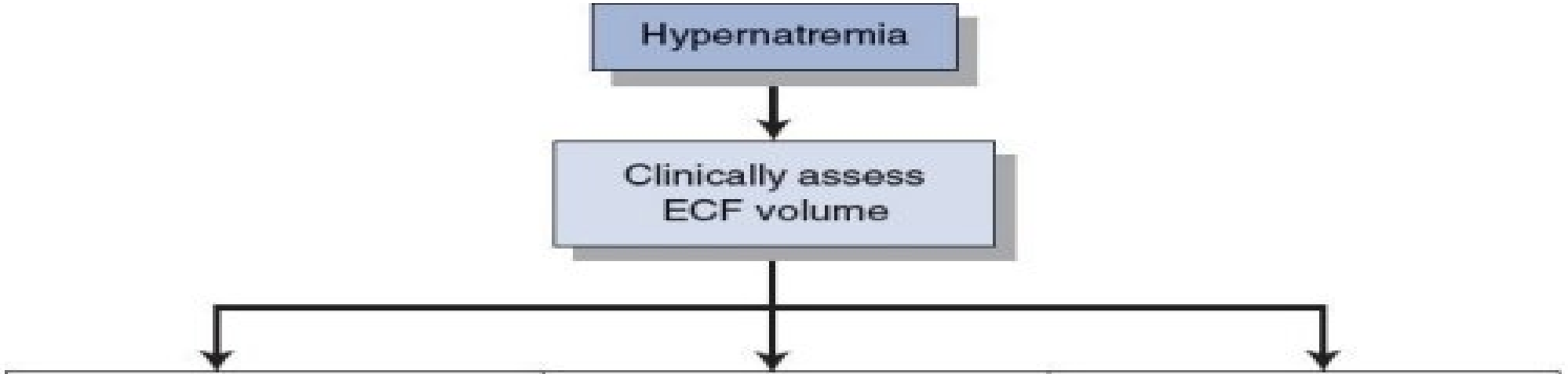
b. Isovolemic hypernatremia (sodium stores normal, water lost)

_ Diabetes insipidus: central (usually due to CNS pathologies) or peripheral (causes include lithium toxicity, hypercalcemia, hypokalemia, renal disease, drugs)

c. Hypervolemic hypernatremia (sodium excess)—occurs infrequently

_ Iatrogenic—most common cause of hypervolemic hypernatremia

_ Cushing syndrome _ Primary hyperaldosteronism



Depleted	Normal	Expanded
Hypovolemic Hypernatremia	Isovolemic Hypernatremia	Hypervolemic Hypernatremia
Loss of Na^+ and water, but more water loss than Na^+ loss	Loss of water only Na^+ stores normal	Gain of Na^+ and water, but more Na^+ gain than water gain
Causes	Causes	Causes

• Renal loss
  • Diuretics
  • Osmotic diuresis (glycosuria)
  • Renal failure
• Extrarenal loss
  • Diarrhea (GI losses)
  • Diaphoresis (skin losses)
  • Respiratory losses
	• Diabetes insipidus
	• Insensible respiratory (tachypnea)
		• Iatrogenic
		  • $NaHCO_3$ therapy
		  • TPN
		• Exogenous glucocorticoids
		• Cushing's syndrome
		• Saltwater drowning
		• Primary hyperaldosteronism

Clinical Features

1. Neurologic symptoms predominate
    a. Altered mental status, restlessness, weakness, focal neurologic deficits
    b. Can lead to confusion, seizures, coma

2. Tissues and mucous membranes are dry; salivation decreases

Treatment

1. Hypovolemic hypernatremia—Give isotonic NaCl to achieve euvolemia and restore hemodynamics initially. 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 ,low sodium diet, and thiazide diuretics.

3. Hypervolemic hypernatremia—Give diuretics (such as furosemide, to correct volume status)

Hypokalemia

Causes

1. GI losses
    a. Vomiting and nasogastric drainage (volume depletion and metabolic alkalosis also result)
    b. Diarrhea
    c. Laxatives and enemas
    d. Intestinal fistulas—particularly after inflammatory bowel disorders such as Crohn's or diverticulitis
    e. Decreased potassium absorption in intestinal disorders

2. Renal losses

a. Diuretics

b. Renal tubular or parenchymal disease

c. Primary and secondary hyperaldosteronism

d. Excessive glucocorticoids—due to mineralocorticoid action at high serum levels

e. Magnesium deficiency

f. Bartter syndrome—chronic volume depletion secondary to an autosomal recessive defect in salt reabsorption in the thick ascending limb of the loop of Henle leads to hyperplasia of juxtaglomerular apparatus, which leads to increased renin levels and secondary aldosterone elevations.

3. Other causes

a. Insufficient dietary intake
b. Insulin administration
c. Certain antibiotics especially amphotericin B
d. Profuse sweating
e. Epinephrine (β2-agonists)—hypokalemia occurs in 50% to 60% of trauma patients, perhaps due to increased epinephrine levels

Clinical Features

1. Arrhythmias—prolongs normal cardiac conduction
    a. S_T depression . U waves appear if severe

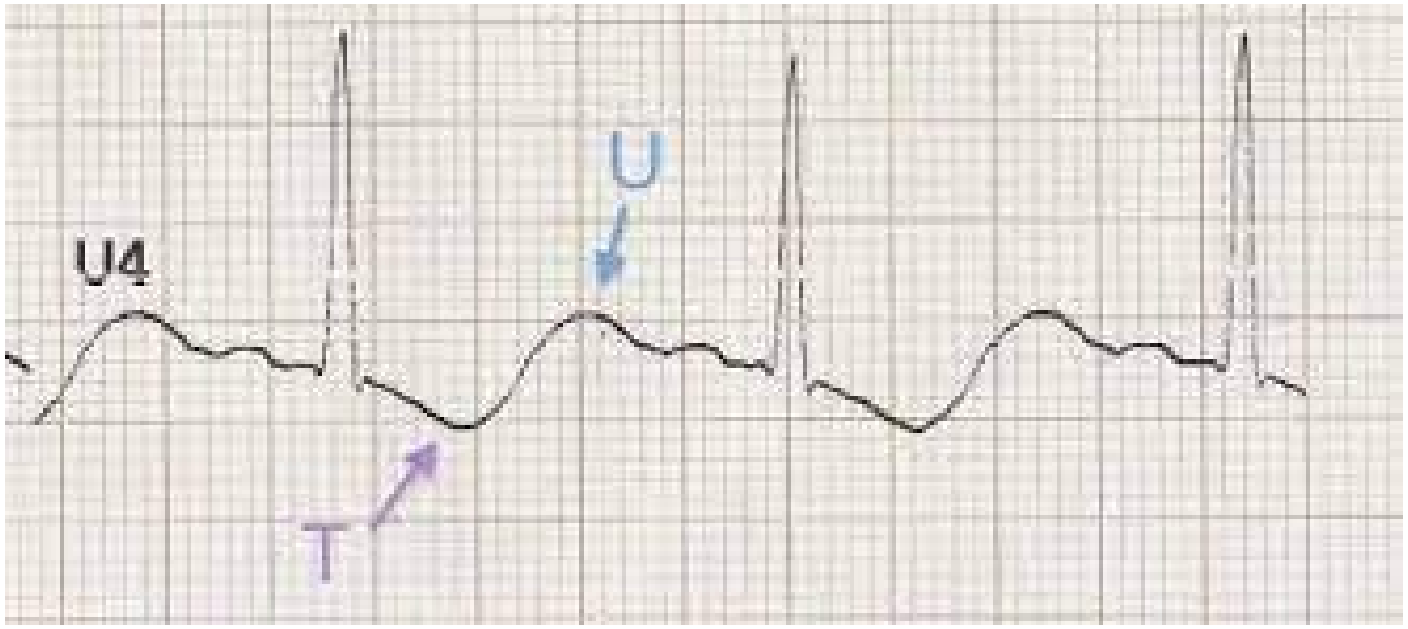
2. Muscular weakness, fatigue, paralysis, and muscle cramps

3. Decreased deep tendon reflexes

4. Paralytic ileus

5. Polyuria and polydipsia

6. Nausea/vomiting



Treatment

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 most instances.

4. IV KCl can be given if hypokalemia is severe (<2.5)

Hyperkalemia

Causes

1. Increased total-body potassium
    a. Renal failure (acute or chronic)
    b. Hypoaldosterone states: Addison disease, hyporeninemic hypoaldosteronism, ACE inhibitors, potassium-sparing diuretics (spironolactone)
    c. Iatrogenic–excessive doses of potassium
    d. Blood transfusion—usually due to lysed cells

2. Redistribution—translocation of potassium from intracellular to extracellular space

a. Acidosis (notably not seen in lactic or ketoacidosis)
b. Tissue/cell breakdown—rhabdomyolysis (muscle breakdown), chemotherapy, hemolysis, burns
c. Insulin deficiency—insulin stimulates the Na+-K+-ATPase and causes K+ to shift into cells.
d. Rapid administration of β-blocker

3. Pseudohyperkalemia (spurious)

a. This refers to an artificially elevated plasma K+ concentration due to K+ movement out of cells immediately before or after venipuncture.

_ Contributing factors include prolonged use of a tourniquet with or without repeated fist clenching. This can cause acidosis and subsequent K+ loss from cells.

Clinical Features

1. Arrhythmias—The most important effect of hyperkalemia is on the heart.
   _ With increasing potassium, ECG changes progress through tall, peaked T waves, QRS widening, PR interval prolongation, loss of P waves, and finally a sine-wave pattern.

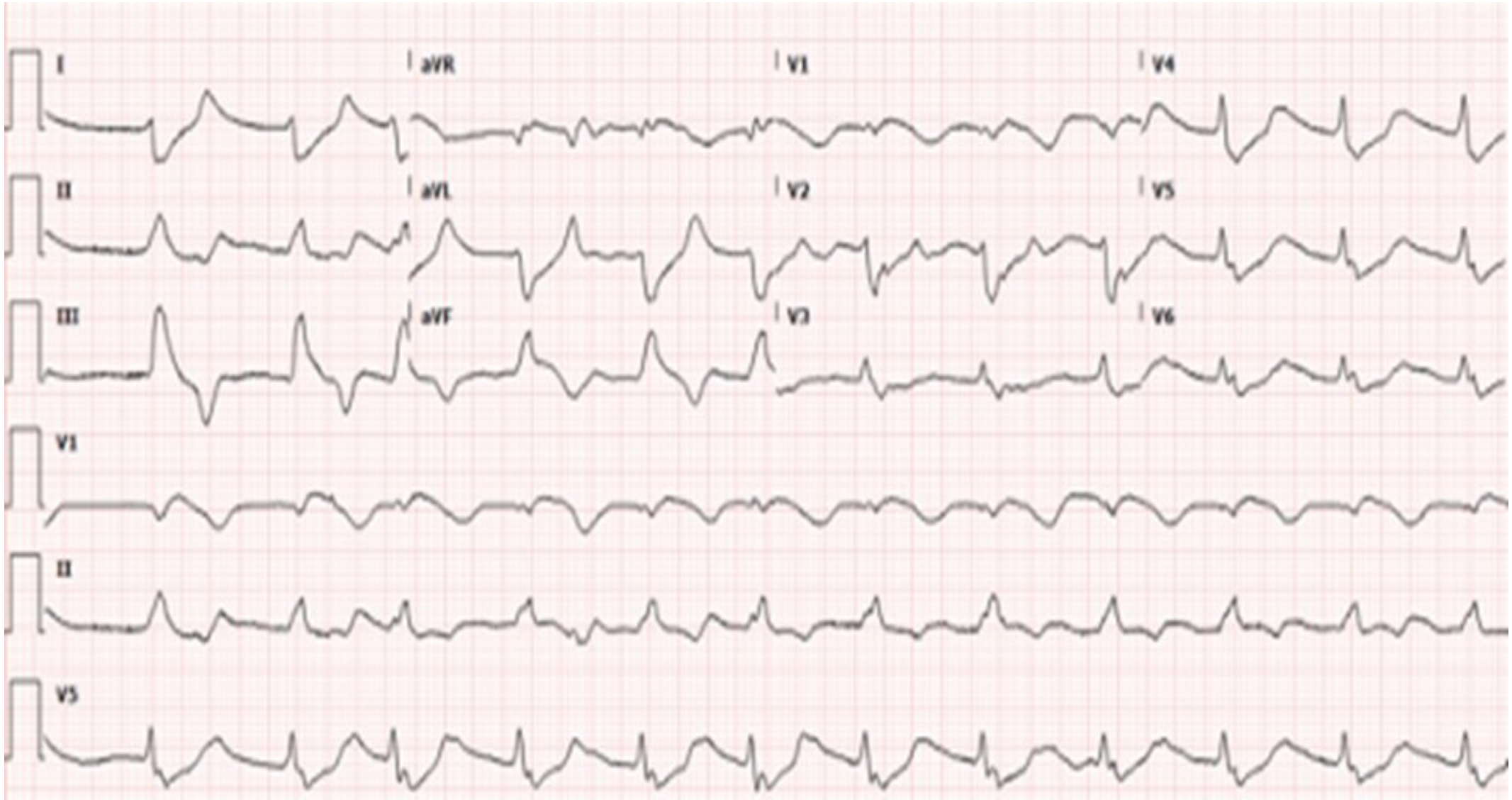
2. Muscle weakness and (rarely) flaccid paralysis

3. Decreased deep tendon reflexes

4. Respiratory failure

5. Nausea/vomiting, intestinal colic, diarrhea

6. Acidosis due to decreased ammonium formation in renal tubules


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Treatment

1. If the hyperkalemia is severe, or if ECG changes are present, first give IV calcium.
    a. Calcium stabilizes the resting membrane potential of the myocardial membrane —that is, it decreases membrane excitability.

2. Shift potassium into the intracellular compartment.
    a. Beta agonists (e.g., albuterol)
    b. Glucose and insulin
    c. Sodium bicarbonate—Increases pH level, which shifts K+ into cells via H+/K+ pump.

3. Remove potassium from the body.
    a. Kayexalate
    b. Diuretics (furosemide)
    c. Hemodialysis.