ELECTROLYTE DISORDERS

BY

ASMAA HASSAN MOHAMED

Fluids & Electrolytes Imbalances

40
Ca
CALCIUM
20

39
K
POTASSIUM
19

24
Mg
MAGNESIUM
12

23
Na
SODIUM
11

Intracellular vs Extracellular:

- **Electrolyte composition is different:**

1. **Intracellular** K+, Mg+, PO4 , SO4 and proteins
2. **Extracellular** Na+, Ca+, Mg+, Cl- , HCO3 and lactate

- **Compositions of ions are maintained:**

  - selective permeability of cell membranes.
  - active ion pumps.

- **Movement of water is passive:**

  - colloid osmotic gradients intravascular v interstitial spaces (extracellularly).

HYPERNATREMIA

➢ Sodium (Na+):

- Range 135 - 145 meq/L in serum .
- Total body volume estimated at 40 meq/kg .
- 1/3 fixed to bone, 2/3 extracellular and available for trans trans-membrane exchange .
- Normal daily requirement 1-2 meq/kg/day .
- Chief extracellular cation .

- Hypernatremia occurs when plasma sodium concentration exceeds 145 mEq/L, reflecting a deficit of water for the amount of sodium in the extracellular fluid (Water moves from ICF → ECF).

- In most cases, hypernatremia results from a negative water balance. Two categories are observed according to the volume and the aspect of the urine:

1. *Water intake is decreased*
2. *Urinary excretion of water is increased* in patients with central or nephrogenic diabetes insipidus.

CLINICAL FEATURES

- Patients may complain of polyuria or polydipsia or have obvious causes of extrarenal fluid losses; others may have no complaints at all.

- Hypernatremia should be considered in any patient presenting with altered mental status and head injury, as well as in bed-ridden patients who have no access to water.

DIAGNOSTIC STRATEGIES

- The degree of hypernatremia almost always equals the total body water deficit in adults. The patient's total body water (TBW) deficit can be estimated by the formula :

$$\text{TBW deficit} = \text{TBW} \times (\text{serum Na} / 140) - 1$$

- A patient's TBW is usually calculated by multiplying the patient's body weight in kilograms times 0.6.

MANAGEMENT

- **Has three interdependent goals:**

✓ **First, to quickly correct underlying shock, hypoperfusion, or significant hypovolemia with normal saline.**

✓ **Second, to treat the underlying cause of hypernatremia, such as fever, vomiting, or diabetes insipidus.**

✓ **Third, to carefully lower the serum sodium level, usually by replacement of the body's total water deficit.**

HYPONATREMIA

- **Defined as serum sodium concentration of less than 135 meq/L, is the second most common electrolyte abnormality encountered in clinical practice.**
- **Although hyponatremia has many causes, they fall into four general categories:** pseudohyponatremia, hyponatremia with dehydration and decreased extracellular volume, hyponatremia with increased extracellular volume, and euvolemic hyponatremia with increased total body water.

PSEUDOHYPONATREMIA

- A falsely low sodium reading caused by the presence of other osmolar particles in the serum. This is explained by the increased percentage of large molecular particles relative to sodium.

- These large molecules do not contribute to plasma osmolality, resulting in a state in which the relative sodium concentration is decreased but the overall osmolality remains unchanged. Severe hypertriglyceridemia and hyperproteinemia are two common causes of this condition.

HYPOVOLEMIC HYPONATREMIA

- Occurs when there is decreased extracellular volume combined with an even greater loss of sodium.

- Hyponatremia with dehydration due to body fluid losses includes sweating, vomiting, diarrhea, and gastrointestinal suction, burns, and intra-abdominal sepsis.

- Hypovolemic hyponatremia due to renal causes includes diuretic use (thiazides), mineralocorticoid deficiency and renal tubular acidosis.

HYPERVOLEMIC HYPONATREMIA

- Hyponatremia with increased extracellular volume, occurs when sodium and water are retained but water retention exceeds sodium retention.
- Most of these patients present with edema. Occurs in patients with heart failure, chronic renal failure, and hepatic failure.
- The fluid retention in these states is secondary to renal hypoperfusion, resulting in increased aldosterone secretion and a decrease in free water excretion.

EUVOLEMIC HYPONATREMIA

- Is one in which patients are euvolemic but have increased total body water. Causes of this type of hyponatremia include , the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), psychogenic polydipsia, hypothyroidism, and accidental or intentional water intoxication. These patients do not present with edema because most of the increased body water is intracellular and not intravascular.

- SIADH occurs when normal antidiuretic hormone secretion is lost and antidiuretic hormone is secreted independently of the body's need to conserve water. That causes total body water to increase, diluting the body's sodium and causing the serum sodium to decrease.

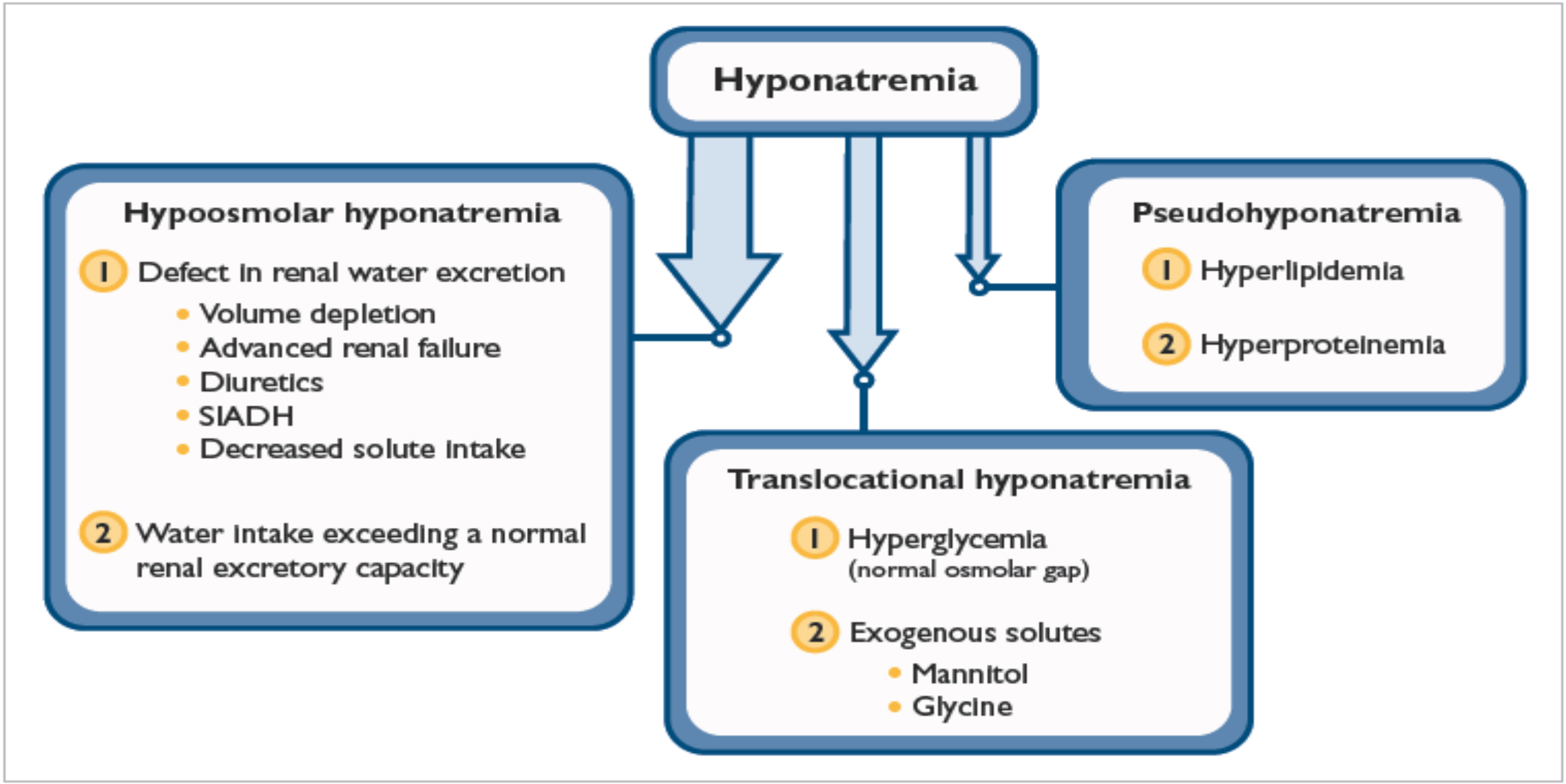


Figure 8. Low Na^+ concentration can be hypoosmolar or hyperosmolar (translocational) or can reflect pseudohyponatremia.

CLINICAL FEATURES

- Nonspecific signs include anorexia, nausea, vomiting, and generalized weakness. Acutely hyponatremic patients whose sodium level drops below 120 meq/L during 24 to 48 hours may present with severe neurologic findings, including confusion, seizures, cerebral edema, coma, and brainstem herniation.
- Determination of the hydration status of the patient may help establish the etiology of the hyponatremia.
- Hypovolemic hyponatremia is more likely in the patient with diminished skin turgor, increased capillary refill, and dry mucous membranes.
- Patient with jugular venous distention, peripheral edema, or pulmonary congestion is much more likely to have hypervolemic hyponatremia. Patients with SIADH will have no edema and have normal skin turgor.

- CNS damage due to hyponatremia may be caused by cerebral edema and increased intracranial pressure, by osmotic fluid shifts during overly aggressive treatment.
- When they are subjected to a hyponatremic environment, neurons become depleted of sodium and potassium in an attempt to limit their own osmolarity to prevent intracellular fluid shifts that would lead to cerebral edema.
- If fluid therapy raises extracellular sodium levels too quickly, fluids shift out of neurons and diffuse demyelination may occur, leading to flaccid paralysis and often death due to a syndrome most commonly referred to as central pontine myelinolysis, although it is more accurately labeled the osmotic demyelinating syndrome.

DIAGNOSTIC STRATEGIES

- **Hypovolemic hyponatremia** due to **nonrenal causes** typically have a low urinary sodium or chloride level **(<20 meq/L)** as they try to retain solute. Hypovolemic hyponatremia due to **renal causes** will have elevated urine sodium and chloride levels **above 20 meq/L** as their kidneys cannot retain sodium or chloride.

- **Euvolemic hyponatremia** typically have a urinary sodium concentration **greater than 20 meq/l** secondary to volume expansion caused by water retention.

- **Hypervolemic hyponatremia** secondary to CHF or cirrhosis have urine sodium levels of **less than 20 meq/l** because of renal hypoperfusion, whereas those with **renal causes** of hypervolemic hyponatremia or with SIADH have sodium levels in **excess of 20 meq/l** as their kidneys are not able to retain sodium

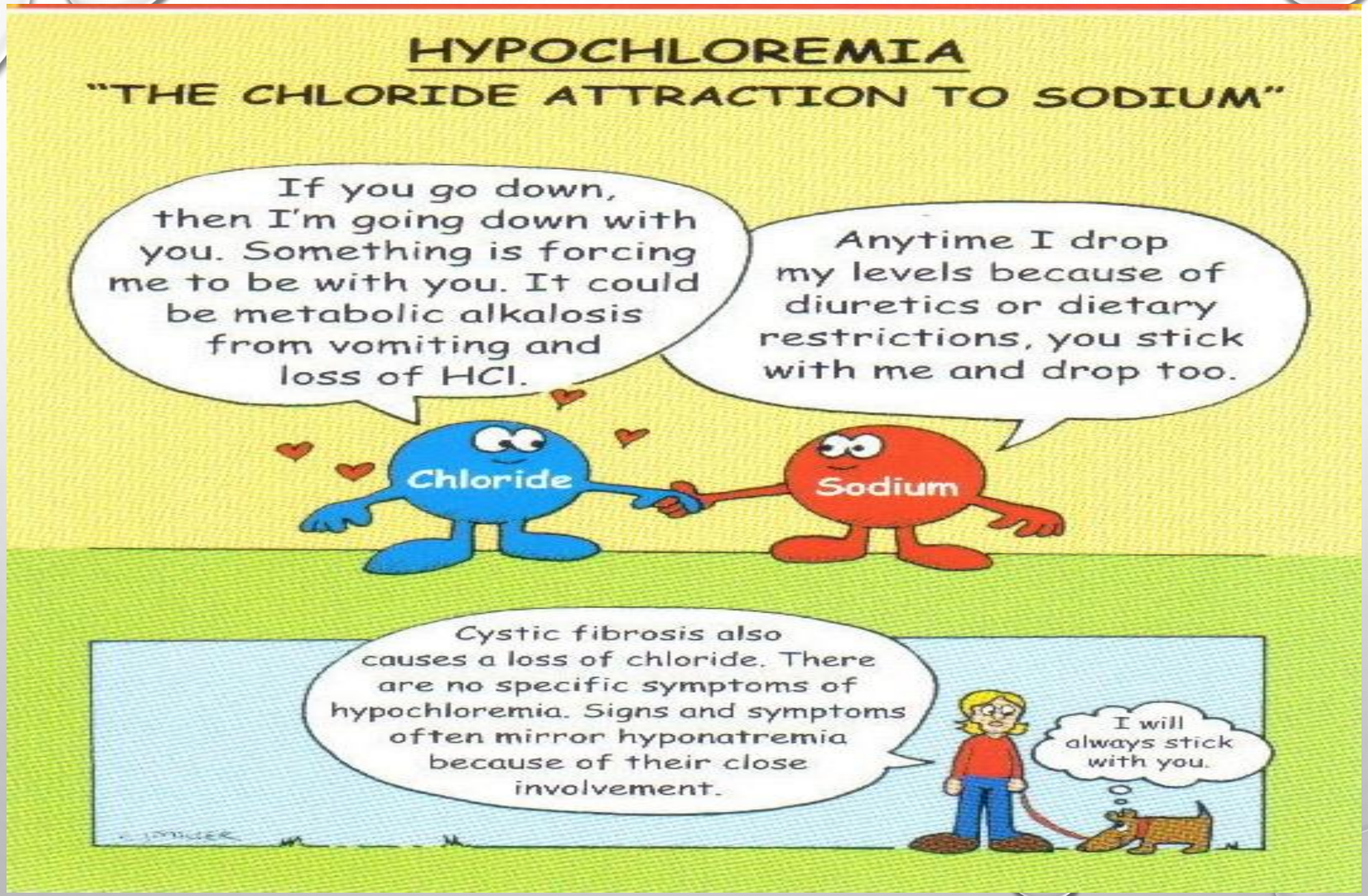
MANAGEMENT

- Most patients presenting to the ED with hyponatremia are stable and require no emergent therapy. However, patients who have serum sodium levels of significantly less than 120 meq/L and those who have acute alterations in mental status need immediate intervention.

- The following equation is helpful to estimate the effect of 1 liter of any infusate on serum sodium:

Change in serum Na = infusate Na − serum Na / total body water + 1

- Treatment of hypovolemic hyponatremia begins with rehydration. Hypotensive, dehydrated patients are volume resuscitated with normal saline.
- Restriction of fluid and sodium is the preferred treatment of patients with hypervolemic hyponatremia. Hemodialysis is an alternative in patients with renal impairment. Patients with CHF will usually benefit from diuretics.
- The mainstay of treatment of euvolemic hyponatremia is free water restriction. As the hypo-osmolality in SIADH results from a relative reduced ability to excrete water, the restriction of free oral water intake is the first recommendation. Most cases of SIADH caused by malignant disease resolve with effective antineoplastic therapy.


HYPOCHLOREMIA
"THE CHLORIDE ATTRACTION TO SODIUM"
If you go down, then I'm going down with you. Something is forcing me to be with you. It could be metabolic alkalosis from vomiting and loss of HCl.
Anytime I drop my levels because of diuretics or dietary restrictions, you stick with me and drop too.
Chloride
Sodium
Cystic fibrosis also causes a loss of chloride. There are no specific symptoms of hypochloremia. Signs and symptoms often mirror hyponatremia because of their close involvement.
I will always stick with you.

HYPERKALEMIA

➢ POTASSIUM (K+)

- RANGE 3.5 - 5.0 MEQ/L IN SERUM.
- TOTAL BODY VOLUME ESTIMATED AT 50 MEQ/KG.
- NORMAL DAILY REQUIREMENT 0.5 – 0.8 MEQ/KG/DAY.
- CHIEF INTRACELLULAR CATION (98%).

- Defined as serum potassium level greater than 5.0 mEq/L, is the most dangerous acute electrolyte abnormality.

- it is usually totally asymptomatic, with cardiac arrest as its first "symptom". Thus the diagnosis of hyperkalemia depends on paying specific attention to risk factors for impaired potassium excretion, such as dehydration and renal failure. Evaluation of the electrocardiogram (ECG) of patients at risk for this electrolyte disturbance is critical.

- Hyperkalemia causes cardiotoxicity by increasing the resting membrane potential of the cardiac myocyte, causing "membrane excitability" and hypo-depolarization as well as decreasing the duration of repolarization. At very high levels, potassium causes the depolarization threshold to rise, leading to overall depressed cardiac function.

- A serum potassium level of 10.0 mEq/L is usually fatal, but decompensation and death can occur at any level above 7 to 8 mEq/L.

CLINICAL FEATURES

- Hyperkalemia remains a difficult clinical diagnosis to make on clinical grounds alone.
- May have gastrointestinal effects such as nausea, vomiting, and diarrhea often in association with their underlying disease. Neuromuscular findings, including muscle cramps, generalized weakness, paresthesias, tetany, and focal or global paralysis seen in patients with severe hyperkalemia.
- Progressive muscle weakness, paresthesias, dyspnea, and depressed deep tendon reflexes are neither sensitive nor specific with a particular serum potassium level. Patients with severe hypokalemia may present with hemodynamic instability and cardiac arrhythmias.

ETIOLOGY OF HYPERKALEMIA

- Increased intake of potassium

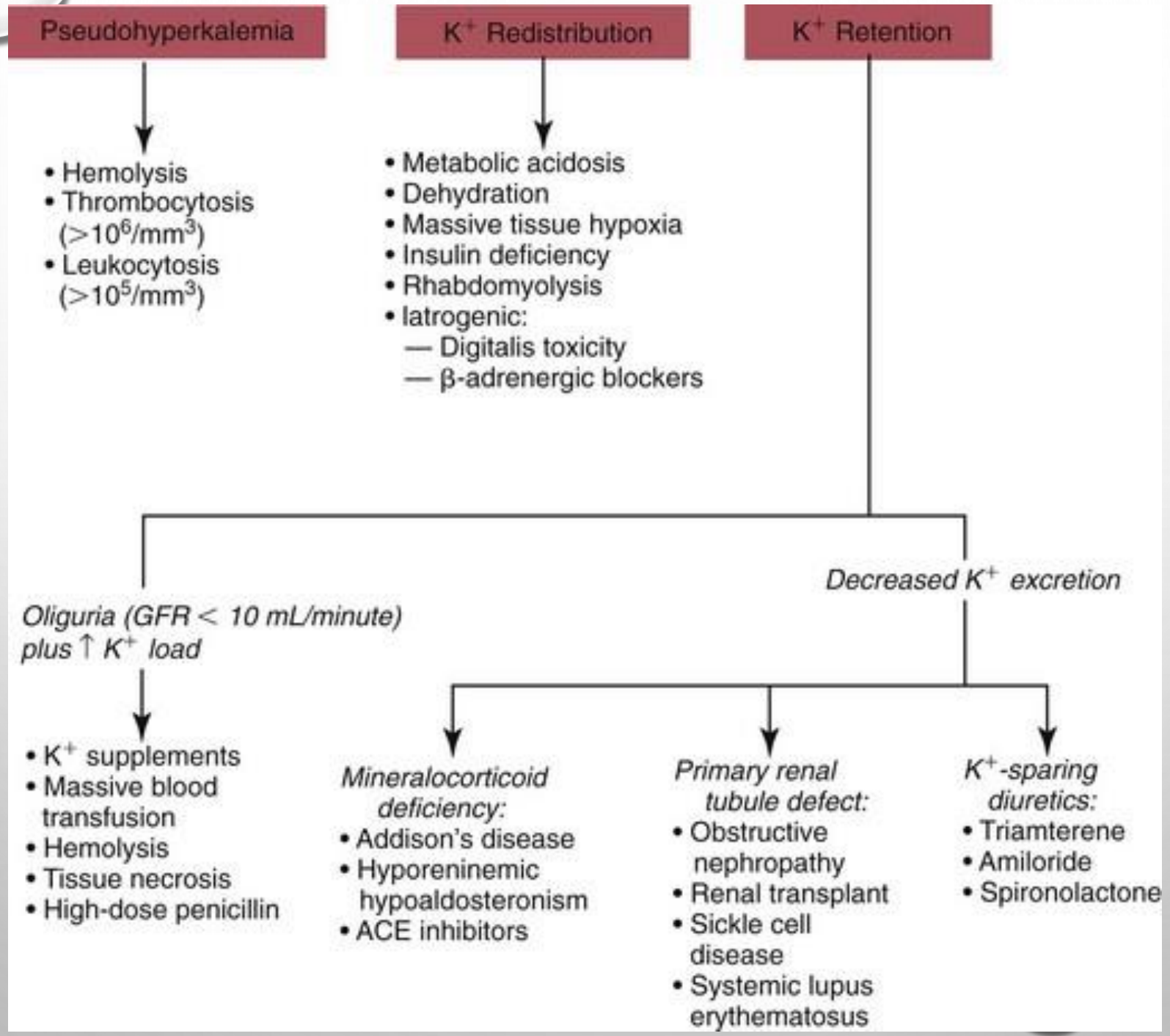
  Orally or intravenously

- Decreased renal excretion of potassium

- **Renal failure: acute or chronic**
- **hypoaldosteronism: addison's disease**
- **Potassium-sparing diuretics**

- Extracellular shift of potassium

- **Metabolic acidosis: Diabetic ketoacidosis, type 4 renal tubular acidosis**
- **Cell destruction: Rhabdomyolysis, tumor lysis syndrome, massive hemolysis**
- **Drugs: beta-blockers, acute digitalis overdose**

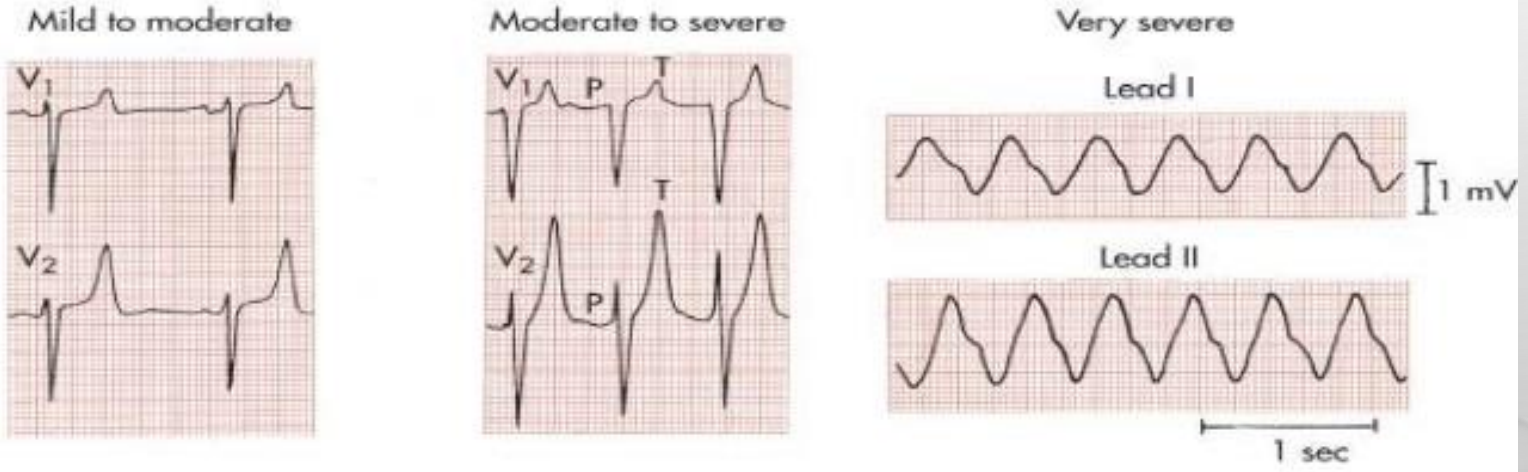

Pseudohyperkalemia
K+ Redistribution
K+ Retention
• Hemolysis
• Thrombocytosis (>10^6/mm^3)
• Leukocytosis (>10^5/mm^3)
• Metabolic acidosis
• Dehydration
• Massive tissue hypoxia
• Insulin deficiency
• Rhabdomyolysis
• Iatrogenic:
— Digitalis toxicity
— β-adrenergic blockers
Decreased K+ excretion
Oliguria (GFR < 10 mL/minute) plus ↑ K+ load
• K+ supplements
• Massive blood transfusion
• Hemolysis
• Tissue necrosis
• High-dose penicillin
Mineralocorticoid deficiency:
• Addison's disease
• Hyporeninemic hypoaldosteronism
• ACE inhibitors
Primary renal tubule defect:
• Obstructive nephropathy
• Renal transplant
• Sickle cell disease
• Systemic lupus erythematosus
K+-sparing diuretics:
• Triamterene
• Amiloride
• Spironolactone

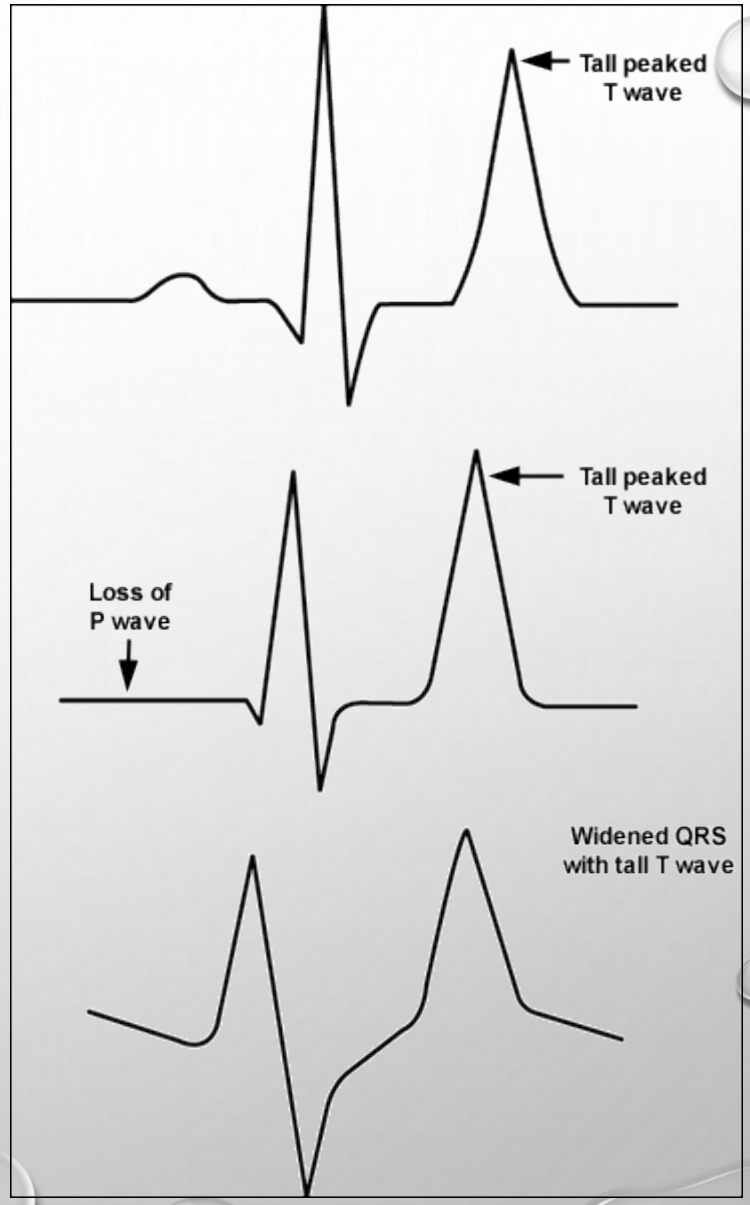
DIAGNOSTIC STRATEGIES

- **The ECG** is helpful in making the diagnosis of hyperkalemia and can be used in unstable patients to initiate treatment.
- **Classic electrocardiographic changes:** the peaked T wave, flattened p wave with prolonged PR interval or a totally absent P wave, wide QRS, and sine wave pattern.
- **Peaked T waves** usually appear as serum potassium levels exceed 5.5 to 6.5 meq/L; **P wave disappearance and PR prolongation** are common with levels above 6.5 to 7.5 meq/L; and **QRS prolongation** is seen with potassium levels above 7.0 to 8.0 meq/L.

ECG: Hyper K

Peaked T waves
Flattened P waves, increasing PR (suppression of atrial activity)
QRS widening and slurring. (conduction delay in ventricular tissue)





MANAGEMENT

- **The treatment strategy consists of three main steps**:

stabilization of the cardiac membrane, shifting of potassium into the cells, **and then** removal of potassium from the body.

- **Intravenous calcium is used to stabilize the cardiac membrane by restoring the electrical gradient. Calcium increases the depolarization threshold and the calcium gradient across the cardiac membrane, quieting myocyte excitability , thus narrowing the QRS. Calcium does not decrease serum potassium levels, and its effect is rapid but transient. Authors prefer calcium gluconate rather than calcium chloride on the basis of the reduced risk of tissue necrosis should it extravasate at the injection site.**

- **Insulin** is the most reliable agent to move potassium into cells, but beta2- adrenergic receptor agonists also provide benefit in some patients. Insulin, given intravenously in combination with glucose to prevent hypoglycemia.

- **Sodium bicarbonate** is effective only in hyperkalemic patients who are acidotic .

- **Hemodialysis** should be instituted early in the treatment of life-threatening hyperkalemia in patients with renal failure.

HYPOKALEMIA

- Is the most common electrolyte abnormality encountered in clinical practice. When it is defined as a value of less than 3.5 meq/L.
- Hypokalemia is often seen in association with hypomagnesemia, and patients with low serum potassium levels should be assumed to be hypomagnesemic also.
- Is found in more than 20% of hospitalized patients and in 10 to 40% of patients treated with thiazide diuretics in the outpatient setting.

ETIOLOGY OF HYPOKALEMIA

- Decreased intake of potassium

  Orally or intravenously

- Increased excretion of potassium

- Gastrointestinal: vomiting, gastric
- Suction, diarrhea, laxatives
- Renal: diuretics, hyperaldosteronism,
- Diabetic ketoacidosis

- Intracellular shift of potassium

- Metabolic alkalosis
- Excess of aldosterone, catecholamines,
- Insulin

- Dietary potassium deficiency should be considered in the severely malnourished patient
- Hypokalemia from thiazide diuretics occurs through increases in distal sodium delivery in the nephron and by activation of the renin-angiotensin-aldosterone system.
- Hypokalemia can also occur from gastrointestinal and dermal losses, is often seen after protracted vomiting or nasogastric suctions, only 5 to 10 mEq/L of potassium is lost in gastric fluid.
- Large doses of laxatives and repeated enemas cause excessive potassium loss in the stool and can cause hypokalemia.
- An acute shift of potassium from the extracellular compartment into cells. This is most commonly seen in patients with metabolic alkalosis, in patients with hyperventilation, and in those patients taking medications such as beta-agonists

CLINICAL FEATURES

- Usually asymptomatic but can be manifested with nonspecific complaints, including palpitations, skeletal muscle weakness, easy fatigability, depression, and muscle pain, occur when the serum potassium level is less than 2.5 meq/L.

- However, in patients with cardiac ischemia or heart failure increases the likelihood of cardiac arrhythmias secondary to potassium's effect on the action potential.

- Hypokalemic patients can demonstrate first- and second-degree heart block, atrial fibrillation, ventricular fibrillation, and asystole. Hypokalemia can also promote metabolic acidosis.

DIAGNOSTIC STRATEGIES

- Is rarely suspected on the basis of clinical presentation, and the diagnosis is typically made by measurement of the serum potassium, If there is any suspicion for hypokalemia or a patient presents with generalized weakness, palpitations, or arrhythmias.

- An ECG should be obtained, a flattened T wave can be seen in hypokalemia. U waves, which are small deflections after the T wave, may also be seen.

- It may also cause a prolonged QT interval. Once the QT interval becomes longer than 500 milliseconds, the risk of malignant ventricular arrhythmias.

- A severe degree of hypokalemia with paralysis is a potentially life-threatening medical emergency.

HYPOKALEMIA

"POTASSIUM IS DANGEROUS WHEN IT IS TOO LOW!" (SERUM K^+ < 3.5 mEq/L)

Respiratory alkalosis via hyperventilation

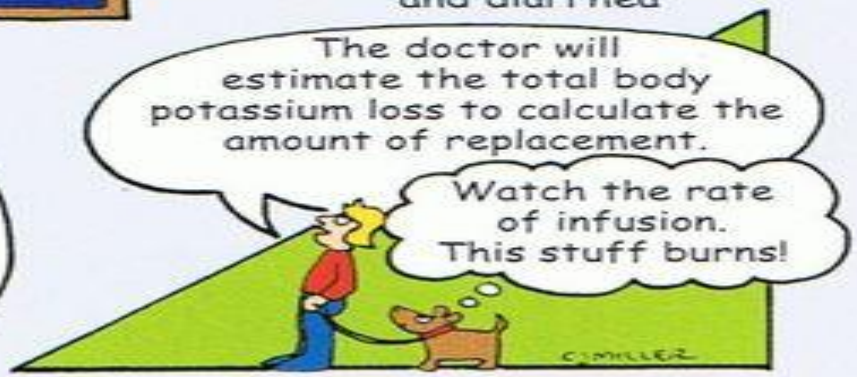
Treatment of pernicious anemia

Metabolic alkalosis via diuretic use and increased urine output

Nasogastric suctioning

Severe vomiting and diarrhea

Watch for skeletal muscle weakness, starting in the arms and legs, progressing to the diaphragm to potentially cause paralysis and respiratory arrest. Look for smooth muscle atony, causing constipation and paralytic ileus, as well as flattened T waves, ST segment elevation, PVCs.



MANAGEMENT

- Potassium is an intracellular cation, so a low serum potassium level almost always reflects a significant total potassium deficit.
- Obtain a spot urine potassium level before starting therapy to assess whether the patient's kidneys are inappropriately wasting potassium from a renal or endocrine cause.
- Potassium chloride is the most commonly used supplementation, and 40 to 60 meq orally every 2 to 4 hours is typically well tolerated.
- Hypokalemia is associated with hypomagnesemia. Magnesium replacement should usually accompany potassium repletion. Unless the patient receives at least 0.5 g/hr of magnesium sulfate along with potassium replacement, potassium will not move intracellularly and the patient will lose potassium through excretion.

HYPERCALCEMIA

- Normal ca+ levels are usually between 9 and 10.5 mg/dl, Mostly in ECF.
- Hypercalcemia is usually defined as a serum calcium level above 10.5 mg/dl; levels higher than 14 mg/dl can be life-threatening.
- There are five major causes of hypercalcemia:

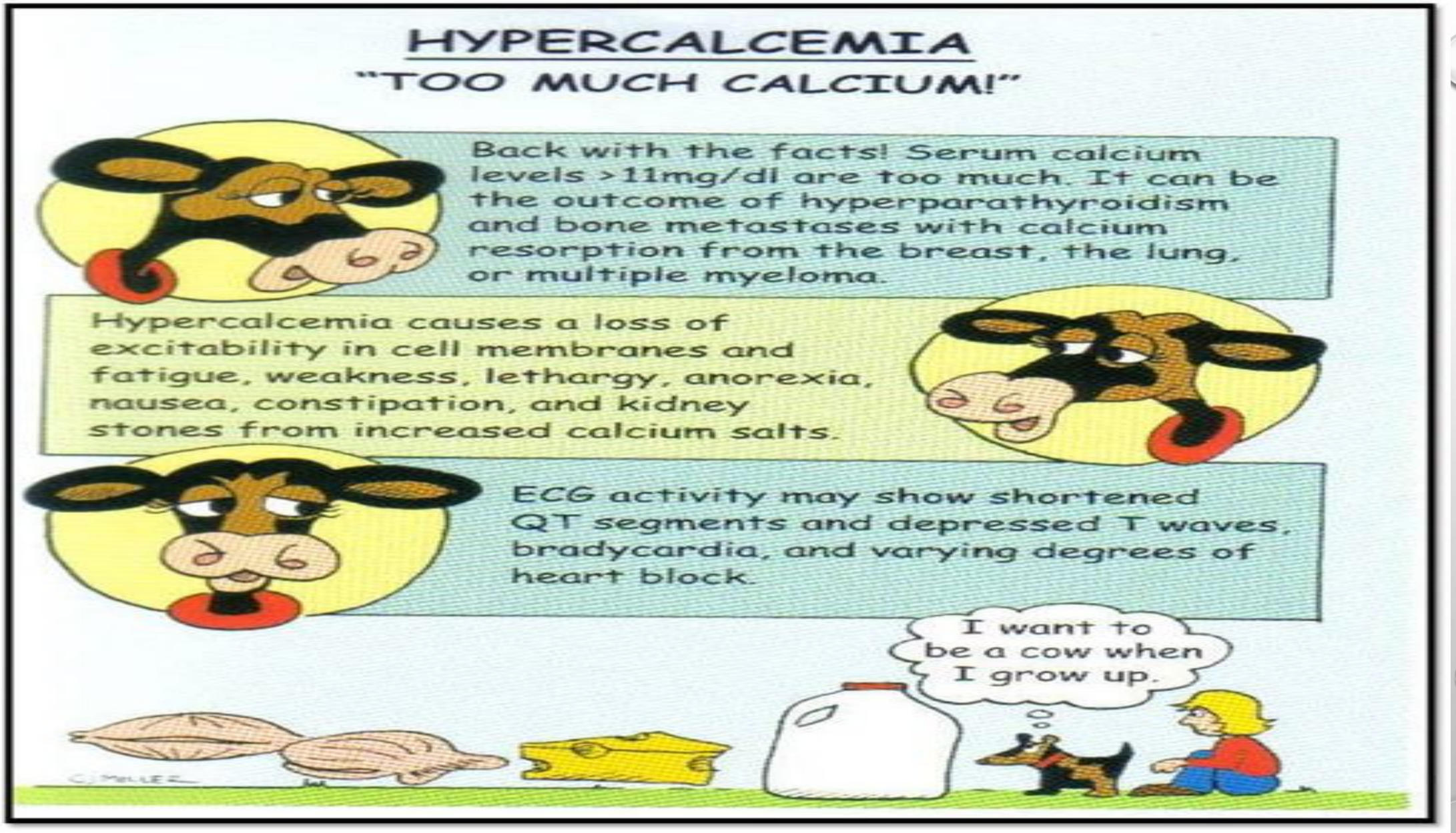
- Malignant disease: multiple myeloma, cancer metastatic to bone
- Endocrine : hyperparathyroidism, hyperthyroidism, pheochromocytoma, adrenal insufficiency
- Granulomatous disease: sarcoidosis, tuberculosis, histoplasmosis
- Pharmacologic agents: vitamins A and D, thiazide diuretics
- Miscellaneous: dehydration, prolonged immobilization, iatrogenic, rhabdomyolysis, familial, laboratory error

CLINICAL FEATURES

- The clinical presentation of hypercalcemia is often vague and nonspecific.
- Symptoms include nonfocal abdominal pain, constipation, fatigue, diffuse body aches, anorexia, nausea, and vomiting
- Some patients complain of polyuria or polydipsia. Neuropsychiatric disturbances include anxiety, depression, confusion, and hallucinations. The CNS manifestations that often predominate in more severe cases.
- Death due to hypercalcemia is usually related to complications caused by coma, dehydration, or electrolyte disturbances.
- Cardiac conduction abnormalities may occur; bradydysrhythmias are the most common. Also has been associated with sinus arrest, atrioventricular block, atrial fibrillation, and ventricular tachycardia.

DIAGNOSTIC STRATEGIES

- Calcium is measured by determination of either a total serum calcium level or an ionized calcium level. Ionized calcium is more accurate, but it does need to be routinely evaluated in hypercalcemia.
- The serum total calcium level should be corrected on the basis of the albumin concentration. The adjustment to serum albumin is accomplished by adding or subtracting 0.08 mg/dl to the measured total serum calcium for every 1.0 g/L of albumin below or above 4 g/L albumin, respectively.
- A short QT interval can be seen in hypercalcemia and is considered a classic finding. QT shortening appear to be correlated with the degree of hypercalcemia.
- ST segment elevation may be the least documented but most consistent ECG finding, and hypercalcemia should be considered in the differential diagnosis of ST segment elevation.
- In severe cases of hypercalcemia, sinus bradycardia, bundle branch block, and high-degree atrioventricular block may also been seen.


HYPERCALCEMIA
"TOO MUCH CALCIUM!"
Back with the facts! Serum calcium levels >11mg/dl are too much. It can be the outcome of hyperparathyroidism and bone metastases with calcium resorption from the breast, the lung, or multiple myeloma.
Hypercalcemia causes a loss of excitability in cell membranes and fatigue, weakness, lethargy, anorexia, nausea, constipation, and kidney stones from increased calcium salts.
ECG activity may show shortened QT segments and depressed T waves, bradycardia, and varying degrees of heart block.
I want to be a cow when I grow up.

MANAGEMENT

- Patients in hypercalcemic crisis are usually dehydrated, they require intravenous access with a normal saline infusion and close monitoring. Normal saline will inhibit proximal tubule reabsorption of calcium and also correct the patient's volume depletion.
- Administration of higher volumes of saline may is much more likely to result in increased morbidity and mortality from volume overload, pulmonary edema, and myocardial ischemia .
- Other electrolyte values should be carefully monitored with attention to serum potassium levels.
- Drugs that inhibit osteoclast-mediated bone resorption include the bisphosphonates.
- In cases of hypercalcemic crisis resulting from primary hyperparathyroidism, urgent parathyroidectomy is potentially curative.

HYPOCALCEMIA

- Hypocalcemia is usually defined as a serum calcium level below 8.5 mg/dl.
- **THERE ARE MULTIPLE CAUSES OF HYPOCALCEMIA:**
  - HYPOALBUMINEMIA
  - HYPOPARATHYROIDISM: INHERITED, POSTSURGICAL, AUTOIMMUNE.
  - VITAMIN D DEFICIENCY AND VITAMIN D RESISTANCE: MALABSORPTION SYNDROME, LIVER DISEASE, MALNUTRITION.
  - CHRONIC RENAL FAILURE
  - HYPOMAGNESEMIA
  - RESPIRATORY ALKALOSIS
  - SEVERE PANCREATITIS
  - DRUGS: BISPHOSPHONATES, CALCITONIN

- **Hypoalbuminemia** is the most common, hypoalbuminemia will cause a fall in the measured serum calcium by about 0.8 mg/ dl for every 1 g/dl reduction in serum albumin.

- **Hypoparathyroidism** is a common cause of hypocalcemia, develops because of surgery for head and neck cancers. It develops in 1 to 2% of patients after total thyroidectomy.

- **Hyperphosphatemic patients** often have hypocalcemia because of phosphate's affinity to bind calcium, whereas hypomagnesemia causes end-organ resistance to parathyroid hormone and inhibits the hypocalcemic feedback loop.

- Hypocalcemia in acute pancreatitis is caused primarily by precipitation of calcium soaps in the abdominal cavity, but glucagon-stimulated calcitonin release and decreased parathyroid hormone secretion may play a role.

CLINICAL FEATURES

- There are many neuromuscular and cardiovascular findings predominate. Severe, symptomatic hypocalcemia may result in cardiovascular collapse, hypotension, and dysrhythmias.

- The patient may complain of muscle cramping, perioral or finger paresthesias, shortness of breath secondary to bronchospasm, and tetanic contractions.

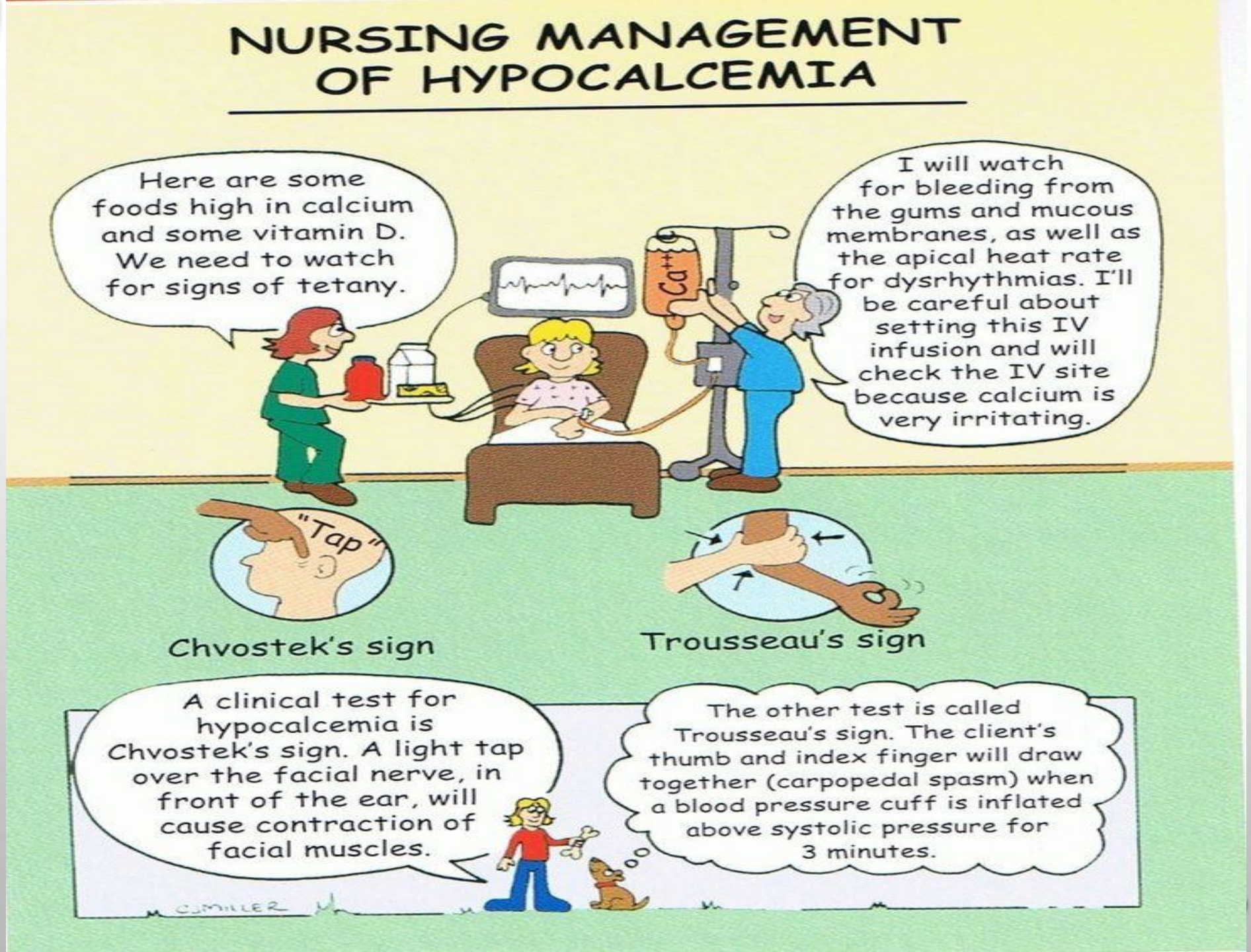
- Chvostek's sign may be present, trousseau's sign may also be present. Trousseau's sign is relatively specific for hypocalcemia

DIAGNOSTIC STRATEGIES

- Hypocalcemia are discovered by clinical suspicion AND BY laboratory testing, A serum calcium level less than 8.5 mg/dl or an ionized calcium level less than 2.0 meq/L .

- ECG and cardiac monitoring are recommended in suspected hypocalcemia patients to evaluate the QT interval and to provide continuous monitoring for potential dysrhythmias.

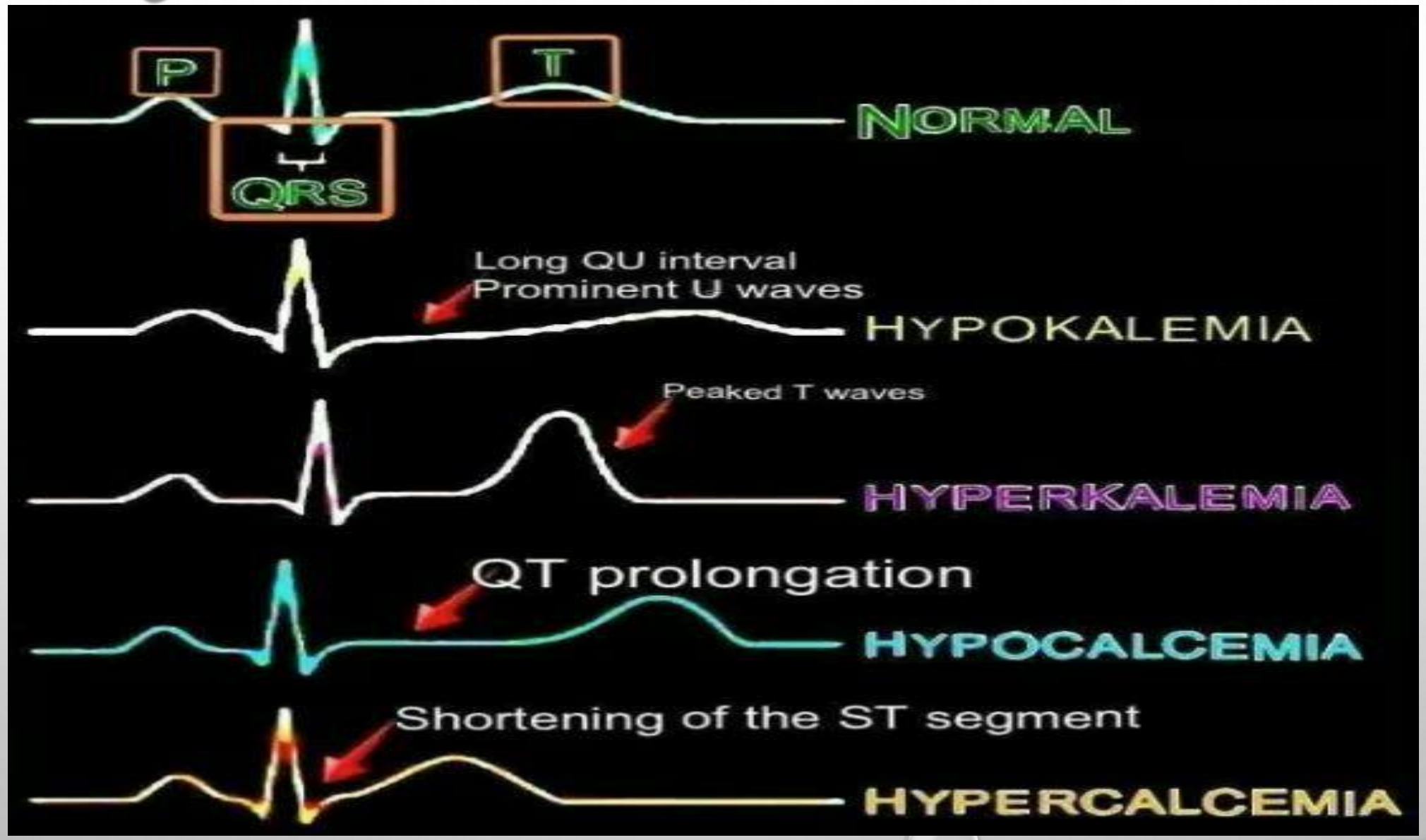
NURSING MANAGEMENT OF HYPOCALCEMIA



MANAGEMENT

- Those with mild symptoms can be treated with oral calcium supplementation, such as calcium carbonate.

- Intravenous calcium gluconate, to patients with moderate to severe symptoms; patients should be admitted to the hospital for monitoring and treatment of nausea, vomiting, hypertension, and bradycardia


P
T
QRS
NORMAL
Long QU interval
Prominent U waves
HYPOKALEMIA
Peaked T waves
HYPERKALEMIA
QT prolongation
HYPOCALCEMIA
Shortening of the ST segment
HYPERCALCEMIA

HYPERMAGNESEMIA

- Is a relatively rare electrolyte abnormality defined as a serum magnesium concentration above 2.2 mg/dl.
- **Most common causes of hypermagnesemia**

- Oral administration laxatives, antacids
- Impaired elimination (hypomotility): bowel obstruction, chronic constipation
- Impaired elimination (medications): anticholinergics, narcotics
- Miscellaneous hypothyroidism, tumor lysis syndrome, adrenal insufficiency, milk-alkali syndrome, near-drowning in dead sea

CLINICAL FEATURES

- Magnesium is a CNS and neuromuscular depressant and can cause cardiac instability, also acts as a calcium channel blocker and also blocks potassium channels needed for repolarization .
- Hypermagnesemia may present with flushing, nausea, vomiting, headache, and diminished deep tendon reflexes .

EFFECT	LEVEL (mg/dL)
Decreased deep tendon reflexes	4-5
Hypotension	5-7
Respiratory insufficiency	10
Heart block	10-15
Cardiac arrest	10-24

DIAGNOSTIC STRATEGIES

- **Measured plasma magnesium levels** often **do not reflect** total magnesium content, making it difficult to consistently correlate symptoms to specific magnesium levels. Only total body magnesium needs to be followed in hypermagnesemic patients.

- **ECG changes**, including **QRS widening** and QT and PR prolongation, begin to occur when serum magnesium levels rise **above 7 mg/dl**.

MANAGEMENT

- Most stable or asymptomatic hypermagnesemic patients can be treated with cessation of their magnesium therapy. As symptoms become more pronounced, intravenous isotonic fluids are administered to dilute the extracellular magnesium.

- In patients with severe symptoms, renal consultation should be initiated immediately to arrange for dialysis.

- Calcium directly antagonizes the neuromuscular and cardiovascular effects of magnesium and is recommended in hypotensive patients.

HYPOMAGNESEMIA

- Is a common electrolyte abnormality that often goes undetected. Normal serum magnesium levels range from 1.5 to 3.0 meq/L. Symptoms of hypomagnesemia typically begin to be manifested at serum levels below 1.2 meq/L.

- Because most of the body's magnesium is intracellular, and thus a single blood sample with a low serum magnesium level may not accurately reflect total body magnesium or the extent of true hypomagnesemia. Magnesium exists in three states: ionized magnesium, protein bound, and complexed to serum anions.

➢ There are many causes of hypomagnesemia:

- **Patients Maintained with Diuretics:** loop or thiazide diuretics, they can inhibit magnesium reabsorption.
- **Malnourished Patients:** hypomagnesemia is common in patients with chronic protein-calorie malnutrition.
- **Patients with Hypokalemia:** Both potassium and magnesium are critical to help stabilize the membrane potential, to decrease cell excitability, and for function of the Na+,K+-ATPase pump.
- **Patients with Acute Coronary Artery Disease and Ventricular Arrhythmias:** Patients who have a myocardial infarction are more likely to be hypomagnesemic.
- **Patients Receiving Specific Medications:** nephrotoxic drugs, including aminoglycosides, and cisplatin can produce magnesium wasting. long-term use of proton pump inhibitors may be associated with changes in intestinal absorption of magnesium.

CLINICAL FEATURES

- Patients with hypomagnesemia typically have hypokalemia, hypocalcemia, and hyponatremia. Symptoms are reported to correlate with hypomagnesemia, including muscle cramping, diffuse weakness, palpitations, vertigo, ataxia, depression, and seizures.
- Patients may present with hyperactive deep tendon reflexes, trousseau's and chvostek's signs, and dysphagia from esophageal dysmotility.
- Dysrhythmias including atrial fibrillation, multifocal atrial tachycardia,, ventricular tachycardia, and ventricular fibrillation are the most common cardiovascular manifestations of hypomagnesemia. Arrhythmias are likely to be due to concurrent hypokalemia, hypomagnesemia, or both, resulting in a prolonged QT interval and increases in spontaneous depolarization

DIAGNOSTIC STRATEGIES

- Symptoms of hypomagnesemia begin to be manifested at serum levels below 1.2 meq/L, but symptoms do not always correlate with the total serum magnesium level.

- ECG findings in hypomagnesemia are nonspecific. Hypomagnesemia should be suspected whenever ECG findings of hypokalemia are noted, including PR and QT interval prolongation, ST segment depression, flattening and widening of the T waves, and U waves.

MANAGEMENT

- **Parenteral magnesium** is recommended for life-threatening conditions. 1 to 2 g of magnesium sulfate is an appropriate loading dose, followed by a standard maintenance dose of 0.5 to 1 g/ hr until symptoms have resolved.

- **Magnesium gluconate oral supplementation** can be given if the patient is only mildly hypomagnesemic and asymptomatic.

Normal range	Causes of elevation	Causes of decline
Sodium (Na): 135 – 145 mEq/L	**Hypernatremia:** Excessive loss of water through GI system, lungs, or skin; fluid restriction, certain diuretics, hypertonic IV solutions, tube feeding; hypothalamic lesions, hyperaldosteronism, corticosteroid use, Cushing's syndrome, diabetes insipidus	**Hyponatremia:** Congestive heart failure, cirrhosis, nephrosis, excess fluid intake, syndrome of inappropriate antidiuretic hormone secretion (dilutional hyponatremia); sodium depletion, loss of body fluids without replacement, diuretic therapy, laxatives, nasogastric suctioning, hypoaldosteronism, cerebral salt-wasting disease
Potassium (K): 3.5 – 5.0 mEq/L	**Hyperkalemia:** Aldosterone deficiency, sodium depletion, acidosis, trauma, hemolysis of red blood cells, potassium-sparing diuretics	**Hypokalemia:** Lack of dietary intake of potassium, vomiting, nasogastric suctioning, potassium-depleting diuretics, aldosteronism, salt-wasting kidney disease, major GI surgery, diuretic therapy with inadequate potassium replacement
Calcium (Ca): 8.5 – 10.5 mg/dL	**Hypercalcemia:** Excessive vitamin D, immobility, hyperparathyroidism, potassium-sparing diuretics, ACE inhibitors, malignancy of bone or blood	**Hypocalcemia:** Hypoparathyroidism, malabsorption, insufficient or inactivated vitamin D or inadequate intake of calcium, hypoalbuminemia, diuretic therapy, diarrhea, acute pancreatitis, bone cancer, gastric surgery
Magnesium (Mg): 1.5 – 2.5 mg/dL	**Hypermagnesemia:** Excessive use of magnesium-containing antacids and laxatives, untreated diabetic ketoacidosis, excessive magnesium infusions	**Hypomagnesemia:** Malabsorption related to GI disease, excessive loss of GI fluids, acute alcoholism/cirrhosis, diuretic therapy, hyper- or hypothyroidism, pancreatitis, preeclampsia, nasogastric suctioning, fistula drainage

HYPERPHOSPHATEMIA

- Is defined as a serum level above 2.5 mg/dl, but it is usually clinically significant only when levels are greater than 5 mg/dl. It is extremely common in patients with renal insufficiency or renal failure.
- **Hyperphosphatemia can occur because of four major pathways**:

1. Decreased phosphate excretion.
2. Excessive phosphate intake.
3. Increased renal tubular reabsorption.
4. Shift of phosphate from intracellular to extracellular space.

MOST COMMON CAUSES OF HYPERPHOSPHATEMIA

- **Decreased phosphate excretion**

  Acute and chronic renal failure.

- **Increased renal tubular reabsorption**

  Hypoparathyroidism.

- **Excessive phosphate intake**

  excess vitamin d administration, oral phosphate administration, phosphate enemas or laxatives .

- **Shift of phosphate from intracellular to extracellular space**

  diabetic ketoacidosis

- **spurious hyperphosphatemia**

  - Tumor lysis
  - Rhabdomyolysis
  - Hyperbilirubinemia
  - Hemolysis
  - Hyperlipidemia

CLINICAL FEATURES & MANAGEMENT

- Hyperphosphatemia causes hypocalcemia by precipitating calcium out of the blood and decreasing vitamin D production. It is this secondary hypocalcemia that can cause muscle cramping, tetany, and seizures. Chronic hyperphosphatemia can also lead to metastatic calcifications in joints, tissues, and arteries.

- Treatment focuses on the correction of both electrolytes.

- Patients with normal renal function, phosphate excretion can be increased by saline infusion coupled with loop diuretics. Patients with hyperphosphatemia with renal failure, hemodialysis or peritoneal dialysis should be considered early in the management

HYPOPHOSPHATEMIA

- Is defined as mild (2-2.5 mg/dl), moderate (1-2 mg/dl), or severe (<1 mg/dl).
- Often goes unrecognized, although most patients remain asymptomatic. Severe hypophosphatemia may result in potentially life-threatening complications.
- Hyperventilation, glucose, insulin, and resolving acidosis lead to hypophosphatemia by rapid intracellular shift.
- The ED patients most likely to have hypophosphatemia are those who are malnourished with alcohol withdrawal, acute hyperventilation, or sepsis and patients with DKA.

CLINICAL FEATURES

- Mild to moderate hypophosphatemia is usually asymptomatic, but major clinical manifestations can occur with severe hypophosphatemia. Hypophosphatemia can cause a wide variety of symptoms because phosphate is an essential component to adenosine triphosphate.

- Nonspecific complaints including joint pain, myalgias, irritability, and depression. Severe hypophosphatemia manifested as seizures, arrhythmias, cardiomyopathy, insulin resistance, acute tubular necrosis, rhabdomyolysis, and acute respiratory failure.

MANAGEMENT

- Therapy is essential as levels fall below 1.0 mg/dl. Because hypophosphatemia often is manifested with hypokalemia, phosphate repletion is considered in conjunction with potassium administration.

- Oral phosphorous, can be given to stable or asymptomatic patients. Intravenous preparations are available as sodium phosphate or potassium phosphate, should be given based on severity of hypophosphatemia and presence of symptoms.

- Monitoring of patients for the development of hypocalcemia, hyperkalemia, and hyperphosphatemia is required while intravenous phosphate is administered, especially in patients with renal insufficiency.

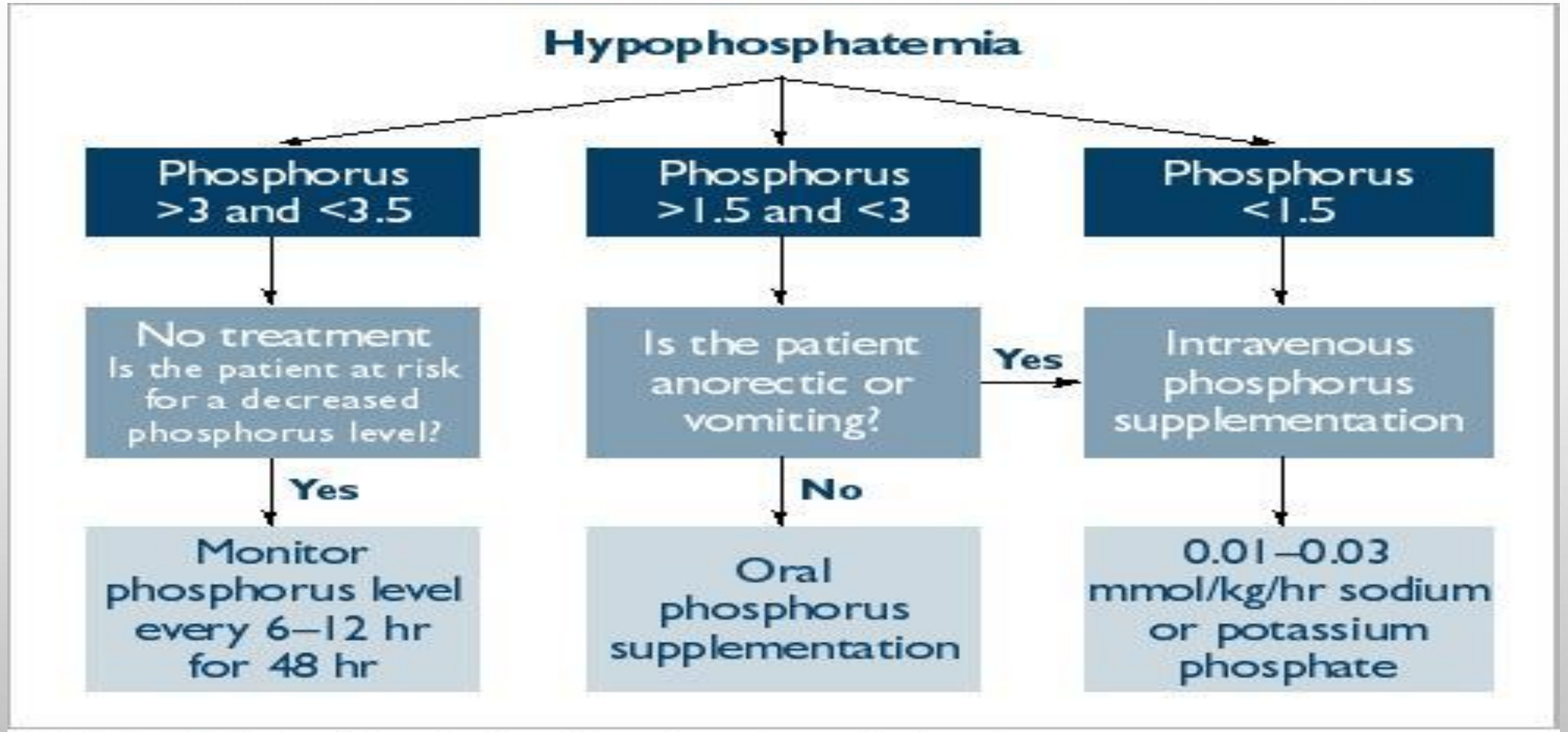


Figure 2. Algorithm for treating hypophosphatemia.

THANK YOU