Fluids & Electrolytes

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Physiology - Background

• Man is 60% water.

• Fluid compartments
  – Total Body water 70kg man = 42 l water
  – Intracellular fluid/space = 55% = 23 l
  – Extracellular fluid/space = 27.5% = 12 l
    • Plasma space 7.5% = 3.5 l
    • Interstitial space 20% = 9 l
  – Others (Bone, Connective tissue & trans cellular & GI tract = 17.5%) “Third Space”
Fluid compartments

ICF
Fluid compartments

- Plasma
- Interstitial
- ICF
- ECF
Fluid compartments

- Plasma
- Interstitial
- ICF
- ECF
Fluid compartments

Capillary Membrane

Plasma

Interstitial

ICF

ECF
Fluid compartments

Capillary Membrane

Plasma

Interstitial

ICF

ECF
Fluid compartments

Capillary Membrane

Plasma

Interstitial

Cell Membrane

ICF

ECF
Colloid osmotic pressure

Capillary membrane freely permeable to water and electrolytes but not to large molecules such as proteins (albumin).
Capillary membrane is freely permeable to water and electrolytes but not to large molecules such as proteins (albumin).
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Colloid osmotic pressure

Capillary membrane freely permeable to water and electrolytes but not to large molecules such as proteins (albumin).

The albumin on the plasma side gives rise to a colloid osmotic pressure gradient favouring movement of water into the plasma.

This is balanced out by the hydrostatic pressure difference.
Cell membrane is freely permeable to H₂O but...
Cell membrane is freely permeable to H$_2$O but Na and K are pumped across this membrane to maintain a gradient!
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\[[K^+] = 4\]
Cell membrane is freely permeable to H2O but Na and K are pumped across this membrane to maintain a gradient!

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\[ Na^+ = 144 \]
Cell membrane is freely permeable to H₂O but Na and K are pumped across this membrane to maintain a gradient!

<table>
<thead>
<tr>
<th>Cell Membrane</th>
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<tbody>
<tr>
<td><strong>Interstitial</strong></td>
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<tr>
<td>Na⁺ = 144</td>
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<td><strong>ICF</strong></td>
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<td>Na⁺ = 10</td>
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## Normal Daily Inputs & Outputs - Water

<table>
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<th>Input</th>
<th>mL</th>
<th>Output</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Drink</td>
<td>1500</td>
<td>Urine</td>
<td>1500</td>
</tr>
<tr>
<td>Food</td>
<td>750</td>
<td>Faeces</td>
<td>100</td>
</tr>
<tr>
<td>Metabolic</td>
<td>350</td>
<td>Lungs</td>
<td>400</td>
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<tr>
<td></td>
<td></td>
<td>Skin</td>
<td>600</td>
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Normal daily water requirements by weight

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<tr>
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**but**

This represents basal requirements and not sick people
Normal Na and K requirements

- 1 mmol/kg/day of each!!
  - ie 70 mmol of Na and of K for the 70 kg man!
  
  - Remember we are much better at conserving Na and we can tolerate much lower levels of Na.
  - Also remember that we measure the K concentration in the extracellular fluid (ie from the blood sample) but K is mainly an Intracellular Ion. Only get low levels when whole body K is very depleted.
Cell membrane is freely permeable to H$_2$O but Na and K are pumped across this membrane to maintain a gradient!
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Prescribing fluid regimes for Patients

- Basal requirements
- Continuing abnormal losses over and above basal requirements
- Pre-existing dehydration and electrolyte loss
Assessment of Dehydration

- **Clinical**
  - Skin turgor, mucous membranes, thirst, urine output, urine concentration or specific gravity, tachycardia, and finally hypotension.

- **Investigations**
  - Urea & Creatinine.
  - CVP
  - Weight
<table>
<thead>
<tr>
<th></th>
<th>Na</th>
<th>K</th>
<th>Cl (mmol)</th>
<th>Volume (L)</th>
</tr>
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<tbody>
<tr>
<td>Saliva</td>
<td>15</td>
<td>19</td>
<td>40</td>
<td>1.5</td>
</tr>
<tr>
<td>Stomach</td>
<td>50</td>
<td>15</td>
<td>140</td>
<td>2.5</td>
</tr>
<tr>
<td>Bile, Pancreas</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&amp; small bowel</td>
<td>130</td>
<td>7</td>
<td>80</td>
<td>4.2</td>
</tr>
<tr>
<td>Sweat</td>
<td>12-50</td>
<td>10</td>
<td>12-50</td>
<td>Variable</td>
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Message for Today

• Do not resuscitate sick patients with any Dextrose solution.
Hypernatremia

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Topics List

- Cases
- Background
- Pathophysiology
- Causes
- Clinical presentation
- Lab Studies
- Treatment
Background ; cont’

- It is not uncommon (0.12-3.5% in hospitalized patients), especially in elderly in-patients because:
  1. Poor water intake
  2. Inability to express thirst
  3. Insensible water loss
  4. Increased urinary water loss

- It is associated with a high mortality rate (>50% in most studies)

- Hypernatremia  \[ \text{Na} > 145 \text{ mEq/L} \]
Background

• Assessment and treatment focuses on 2 important questions:
  1. What is the patient's volume status?
  2. Is the problem acute or chronic?
Most common cause is relative *free water loss*, although it can be caused by salt loading.
Pathophysiology; cont’

Hypernatremia

Cell shrinkage

Electrolytes Trans-membrane Transport

Change in resting potential
Pathophysiology; cont’

• The effects of cellular dehydration are seen principally in the CNS
• Stretching of shrunken neurons and alteration of membrane potentials lead to ineffective functioning
• Severe shrinkage may cause intracranial hemorrhage
Risk Factors

- Age >65 years
- Mental or physical disability
- Hospitalization (intubation, impaired cognitive function)
- Residence in nursing home
- Inadequate nursing care
- Urine concentrating defect (diabetes insipidus)
- Solute diuresis (diabetes mellitus)
- Diuretic therapy
Causes; cont’

- Hypovolemic hypernatremia (i.e., water deficit >sodium deficit)
  - Extra-renal losses (Diarrhea, vomiting, fistulas, significant burns)
  - Renal losses (Osmotic diuretics, diuretics, post-obstructive diuresis, intrinsic renal disease)
  - Adipsic hypernatremia
Causes; cont'

- Hypervolemic hypernatremia (i.e., sodium gains > water gains)
  - Hypertonic saline
  - Sodium bicarbonate administration
  - Accidental salt ingestion (e.g., error in preparation of infant formula)
  - Mineralocorticoid excess (Cushing syndrome)
Causes; cont’

- Euvolemic hypernatremia
  - Extra-renal losses (Increased insensible loss e.g., hyperventilation)
  - Renal losses (Central DI, Nephrogenic DI)
- Most of the free water loss is from intracellular and interstitial spaces and <10% from the intravascular space
Clinical presentation

• Note signs of volume status (mucous membranes, skin turgor, orthostatic vital signs, and neck veins)

• Perform a thorough neurologic examination

• No specific symptoms
  – Anorexia, restlessness, nausea, and vomiting
  – Altered mental status, lethargy or irritability, seizure, ataxia, stupor or coma
  – Muscle twitching, hyperreflexia, ataxia, or tremor

• Focal deficits such as hemiparesis have been reported
Lab Studies; cont’

• Diagnosis is based on an elevated Na
• Urinary electrolytes and urine osmolality
  • Often Urine osmolality > 500 mOsm/kg/H2O
  • Urine osmolality < 100 mOsm/kg/H2O → Central DI
    400 mOsm/kg/H2O → Nephrogenic DI
• Hypokalemia and hypercalcemia may result in nephrogenic DI
• Check serum glucose level to ensure that osmotic diuresis has not occurred
• A CT scan of the brain may be helpful for central DI
Lab Studies

- Na > 190 mEq/L → usually long-term salt ingestion
- Na > 170 mEq/L → usually DI
- Na = 150-170 mEq/L → usually dehydration
Treatment cont’

Goals of management

1. Address the underlying cause
2. Correct hypertonicity

• Correcting the hypertonicity with free water, oral or parenteral
Treatment; cont’

1. Total body water (TBW) = weight (kg) x correction factor

   Estimation for 1 L of any infusate

• Correction factors
  1. Children = 0.6
  2. Non-elderly men = 0.6
  3. Non-elderly women = 0.5
  4. Elderly men = 0.5
  5. Elderly women = 0.45
Treatment; cont'

Free Water Deficit = (TBW) X [ (Serum Na) – 1 ]

140
Treatment; cont’

• Common infusates and their Na+ contents
  • 5% dextrose in water (D5W): 0 mmol/L
  • 0.2% sodium chloride in 5% dextrose in water (D52NS): 34 mmol/L
  • 0.45% sodium chloride in water (0.45NS): 77 mmol/L
  • Ringer's lactate solution: 130 mmol/L
  • 0.9% sodium chloride in water (0.9NS): 154 mmol/L
Hypernatremia

< 48 hrs (acute)
Correct 1-2 mmol/L/h

> 48 hrs (chronic)
Correct 0.5 mmol/L/h
Treatment

• Volume deficit + hypernatremia  →  First N/S prior to free water

• Volume overload + hypernatremia  →  Dialysis, maybe or diuresis and free water

• An obtunded patient requires parenteral treatment
Hyponatremia

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Topics

- Background
- Pathophysiology
- Causes
- Lab Studies
- Treatment
Background

- Sodium is the dominant extra cellular cation
- Normal serum level 135-145 Eq/L
- Hyponatremia is the most common electrolyte disorder
- Incidence of approximately 1% and a prevalence of approximately 2.5%
- Approximately 30% of ICU patients
Background

• Acute hyponatremia is less common than chronic hyponatremia
Pathophysiology

- Serum sodium concentration and serum osmolarity normally are controlled by:
  1. Stimulation of thirst
  2. Secretion of ADH
  3. Renal excretion
  4. Renin-angiotensin-aldosterone

“Correct chronic (> 48 hrs) hyponatremia, gradually”
Pathophysiology

Hypotonic Hyponatremia

- Hypervolemic hyponatremia
  - ECF ↑
    - cirrhosis, nephrotic syndrome, congestive heart failure

- Euvolemic hyponatremia
  - ECF
    - SIADH, exogenous free water load, hypothyroidism, cortisol deficiency

- Hypovolemic hyponatremia
  - ECF ↓
    - vomiting, diarrhea, excessive sweating, burns
      - GI fistulas or drainage tubes, Pancreatitis
Pathophysiology

- **Redistributive (hypertonic) hyponatremia**
  Intracellular water shift outside
  \[ \downarrow \]
  Dilution of Na
  \[ \text{Hyperglycemia} \]

- **Pseudohyponatremia (Isotonic)**
  Excessive proteins or lipids
  \[ \text{Hypertriglyceridemia} \]
Clinical Presentation

• Gradually decrease of Na induces minimally symptoms even as low as 110 mEq/L

• Symptoms

  Headache
  Anorexia
  Muscle cramp

  altered mental status

  Coma

  Obtundation
Clinical Presentation

- Hyponatremia is a presentation of:
  1. Pneumonia
  2. Active tuberculosis
  3. Pulmonary abscess, neoplasm, or asthma
  4. CNS infection, trauma, or neoplasm
  5. Consume large amounts of beer
  6. MDMA
  7. Hypothyroidism or adrenal insufficiency
Causes

• Hypovolemic hyponatremia
  1. Excess fluid losses
  2. Acute or chronic renal insufficiency
  3. Salt-wasting nephropathy
  4. Cerebral salt-wasting syndrome
  5. Prolonged exercise in a hot environment
Causes

- Euvolemic hyponatremia
  1. Psychogenic polydipsia
  2. Administration of hypotonic intravenous or irrigation fluids
  3. Infants have been fed with free water
  4. SIADH
Causes

• Hypervolemic hyponatremia
  1. Acute or chronic renal failure
  2. Hepatic cirrhosis, congestive heart failure, or nephrotic syndrome
  3. Uncorrected hypothyroidism or cortisol deficiency
  4. Consumption of large quantities of beer or use of the recreational drug MDMA
Lab Studies

- Na concentration is diluted by a factor of 1.6 mEq/L for each 100 mg/dL increase above normal serum glucose concentration
- Above 400 mg/dL, 2.4 mEq/L for each 100 mg/dL increase in glucose
Treatment

• Acute hyponatremia
  – The goal is to
    • increase the serum Na level rapidly by 4-6 mEq/L over the first 1-2 hours (1-2 mEq/L/h) or until plasma Na is over 120 mEq/L
    • Arrest seizures, severe confusion, coma, or signs of brainstem herniation
Treatment

• Chronic hyponatremia
  • Correction associates with the development of central pontine myelinolysis
  • Symptoms of CPM (eg, dysarthria, dysphagia, seizures, altered mental status, quadriplegic, hypotension) typically begin 1-3 later
  • Correction rate should be less than 0.5 mEq/L/h or 12 mEq/L/d
  • correct the hyponatremia to a safe range     (nearly 120 mEq/L)
  • Patients with CNS sign correct like acute one
Treatment

• Chronic hyponatremia
  • Hypovolemic hyponatremia
    – Mild to moderately severe → N/S
  • Hypervolemic hyponatremia
    – sodium and water restriction
  • Euvolemic hyponatremia
    – free water restriction and correction of the underlying condition
Treatment

• Hypertonic (3%) saline
  • Contains 513 mEq/L of NaCl
  • Measured [Na] * (0.6 * W) = mEq [Na] administered
Treatment

• Other modalities
  – Furosemide
  – Demeclocycline
  – Conivaptan (Vaprisol)
hyperkalemia
• The treatment of hyperkalemia is dependent upon the cause and severity of the rise in the plasma potassium concentration.

• Hyperkalemia is most often due to potassium retention in the setting of renal insufficiency and therapy is ultimately aimed at inducing potassium loss.

• In some cases, however, movement of potassium out of the cells is the primary problem, even though the patient's total body potassium may be reduced.
• Symptoms generally do not become manifest until the plasma potassium concentration exceeds 7.0 meq/L.
• Muscle weakness usually begins with the lower extremities and progresses to the trunk and upper extremities.

• If severe, it can progress to flaccid paralysis. It is rare to have respiratory muscle weakness, and

• patients usually have intact sphincter tone and normal cranial nerve exam.
A tall peaked T wave with shortened QT interval is the first change seen on the ECG in a patient with hyperkalemia.

This is followed by progressive lengthening of the PR interval and QRS duration.

The P wave may disappear, and ultimately the QRS widens further to a "sine wave."

Ventricular fibrillation or standstill are the most severe consequences.
• A variety of other conduction disturbances, including right bundle branch block, left bundle branch block, bifascicular block, and advanced atrioventricular block may also be seen

• At plasma potassium concentrations above 7.0 or greater, severe muscle weakness, or marked electrocardiographic changes are potentially life-threatening and require immediate treatment. Immediate therapy is warranted if electrocardiographic changes or peripheral neuromuscular abnormalities are present, regardless of the degree of hyperkalemia
In comparison, an asymptomatic patient with a plasma potassium concentration of 6.5 meq/L whose electrocardiogram does not manifest signs of hyperkalemia can be treated only with a cation exchange resin (Kayexalate®), while patients with a value below 6 meq/L can often be treated with a low potassium diet and diuretics.
Specific treatment of hyperkalemia is directed at antagonizing the membrane effects of potassium, driving extracellular potassium into the cells, or removing excess potassium from the body.
• **Calcium** directly antagonizes the membrane actions of hyperkalemia.

• The protective effect of calcium begins within minutes, but is relatively short-lived. As a result, calcium infusions are indicated only for severe hyperkalemia (as with widening of the QRS complex or loss of P waves, but not peaked T waves alone), when it is potentially dangerous to wait the 30 to 60 minutes for insulin and glucose or sodium bicarbonate to act.
• Calcium should also be administered only when absolutely necessary (as with widening of the QRS complex or loss of P waves).

• Calcium is irritating to veins and extravasation can cause tissue necrosis. As a result, a central or deep vein is preferred for administration of calcium chloride. Calcium gluconate can be given peripherally, ideally through a small needle or catheter in a large vein.
• The usual dose of calcium gluconate is 1000 mg (10 mL of a 10 percent solution) infused slowly over two to three minutes, with constant cardiac monitoring. The usual dose of calcium chloride is 500 to 1000 mg (5 to 10 mL of a 10 percent solution) infused slowly over two to three minutes, with constant cardiac monitoring. The dose of either formulation can be repeated after five minutes if the ECG changes persist.
• **Insulin and glucose:** Hyperinsulinemia can be induced in either by giving insulin (10 units plus 50 mL of a 50 percent glucose solution as a intravenous bolus followed by a glucose infusion to prevent hypoglycemia) or by the intravenous administration of glucose alone (50 mL of a 50 percent glucose solution), which will rapidly enhance endogenous insulin secretion.
• Effective therapy usually leads to a 0.5 to 1.5 meq/L fall in the plasma potassium concentration, an effect that begins in 15 minutes, peaks at 60 minutes, and lasts for several hours.

• insulin alone is sufficient if the patient is already hyperglycemic.
• Sodium bicarbonate: The potassium-lowering action of sodium bicarbonate is most prominent in patients with metabolic acidosis; it begins within 30 to 60 minutes and persists for several hours.

• The usual dose of bicarbonate is 45 meq (50 mL of a 7.5 percent sodium bicarbonate solution) infused slowly over 5 minutes; this dose can be repeated in 30 minutes if necessary. Alternatively, the sodium bicarbonate can be added to a glucose and saline solution.
Beta-2-adrenergic agonists: drive potassium into the cells by increasing Na-K-ATPase activity. As a result, these drugs can be effectively used in the acute treatment of hyperkalemia, lowering the plasma potassium concentration by 0.5 to 1.5 meq/L. The following doses have been used: albuterol (10 to 20 mg in 4 mL of saline by nebulization over 10 minutes or 0.5 mg by intravenous infusion), or epinephrine (0.05 µg/kg per minute by intravenous infusion). The peak effect is seen within 30 minutes with intravenous infusion, but occurs at 90 minutes with nebulization.
As with sodium bicarbonate, there is a limitation to the use of epinephrine in advanced renal failure, since there is a blunted hypokalemic response in this setting.

Albuterol can be added to insulin plus glucose to maximize the reduction in the plasma potassium concentration in patients with severe hyperkalemia and end-stage renal disease.

One problem in patients on maintenance hemodialysis is that lowering the plasma potassium concentration with albuterol can diminish subsequent potassium removal during the dialysis session (from 50 to 29 meq in one report), possibly leading to rebound hyperkalemia after dialysis.
• Loop or thiazide diuretics: patients with persistent hyperkalemia typically have an abnormality in renal potassium secretion and are unlikely to have a good response to diuretic therapy.

• Cation exchange resin: sodium polystyrene sulfonate (SPS, Kayexalate®); In the gut, this resin takes up potassium (and calcium and magnesium to lesser degrees) and releases sodium. Each gram of resin may bind as much as 1 meq of potassium and release 1 to 2 meq of sodium. Thus, a potential side effect is exacerbation of edema due to sodium retention. The oral dose is usually 15 to 30 grams, and is typically provided in 60 to 120 mL of a 20 percent sorbitol solution (to prevent constipation)
• The dose can be repeated every four to six hours as necessary. Lower doses (5 or 10 grams) are generally well tolerated (no nausea or constipation) and can be given one to three times per day to control chronic mild hyperkalemia in patients with renal insufficiency, often without the necessity of concurrent laxative therapy.

• As a side effect: Colonic Necrosis must be considered.

• When given as an enema, 50 g of resin is mixed with 150 mL of tap water (NOT sorbitol); this solution should be kept in the colon for at least 30 to 60 minutes and preferably two to four hours. The enema can be repeated every two to four hours.
• Dialysis: Dialysis can be used if the conservative measures listed above are ineffective, if the hyperkalemia is severe, or if the patient has marked tissue breakdown and is releasing large amounts of potassium from the injured cells.
Hypokalemia:
• The severity of the manifestations tends to be proportionate to the degree and duration of hypokalemia.

• Symptoms generally do not become manifest until the serum potassium is below 3.0 meq/L, unless the serum potassium falls rapidly or The patient has a potentiating factor such as a predisposition to arrhythmia due to the use of digitalis.
• Muscle weakness usually does not occur at potassium concentrations above 2.5 meq/L if the hypokalemia develops slowly. However, significant weakness may occur with sudden decreases, as occurs in hypokalemic periodic paralysis.

• It usually begins with the lower extremities, progresses to the trunk and upper extremities, and can worsen to the point of paralysis.
Hypokalemia can also result in the following:

- **Respiratory muscle weakness**, which can be severe enough to result in respiratory failure and death.
- **Involvement of gastrointestinal muscles**, resulting in ileus and its associated symptoms of distension, anorexia, nausea and vomiting.
- **Cramps, paresthesias, tetany, muscle tenderness and atrophy.**
• Cardiac arrhythmias and ECG abnormalities: premature atrial and ventricular beats, sinus bradycardia, paroxysmal atrial or junctional tachycardia, atrioventricular block, and ventricular tachycardia or fibrillation.

Hypokalemia produces characteristic changes on the ECG. There is depression of the ST segment, decrease in the amplitude of the T wave, and an increase in the amplitude of U waves which occur at the end of the T wave. U waves are often seen in the lateral precordial leads V4 to V6.
Rhabdomyolysis: Severe potassium depletion (serum potassium less than 2.5 meq/L) can lead to muscle cramps, rhabdomyolysis, and myoglobinuria.

Monitoring of the ECG and muscle strength are indicated to assess the functional consequences of the hypokalemia. At serum potassium concentrations lower than 2.5 meq/L, severe muscle weakness, or marked electrocardiographic changes are potentially life-threatening and require immediate treatment.

Immediate therapy is warranted if electrocardiographic changes or peripheral neuromuscular abnormalities are present.
• In chronic hypokalemia, a potassium deficit of 200 to 400 meq is required to lower the serum potassium concentration by 1 meq/L. Once the serum potassium level falls to approximately 2 meq/L, continued potassium losses will not produce much more hypokalemia due to release of potassium from the cell stores.
• **TREATMENT:**

An intravenous or oral potassium chloride preparation is generally preferred over potassium citrate or potassium bicarbonate, in particular among patients with metabolic alkalosis due to diuretic therapy, vomiting, and hyperaldosteronism.

On the other hand, potassium citrate or potassium bicarbonate is often preferred in patients with hypokalemia and metabolic acidosis. This most often occurs in renal tubular acidosis and chronic diarrheal states.
• Oral potassium chloride can be given in crystalline form (salt substitutes), as a liquid, or in a slow-release tablet or capsule.
• Increasing the intake of potassium-rich foods (such as oranges and bananas) is generally less effective.
• Potassium chloride can be given intravenously to patients who are unable to eat or as an adjunct to oral replacement in patients who have severe symptomatic hypokalemia.
• In most patients, intravenous potassium is administered as an additive in intravenous fluids at concentrations of 20 to 40 meq per liter of fluid through a peripheral vein. A concentration up to 60 meq/L can also be used, but such higher concentrations are often painful.
• A saline rather than a dextrose solution is recommended for initial therapy, since the administration of dextrose can lead to a transient 0.2 to 1.4 meq/L reduction in the serum potassium concentration, particularly if only 20 meq/L of potassium chloride is provided.

• In patients who cannot tolerate large volumes of fluid, more concentrated solutions (200 to 400 meq/L) can be infused into large veins in patients with severe symptomatic hypokalemia.
• Most hypokalemic patients have a serum potassium concentration between 3.0 and 3.5 meq/L;
• this degree of potassium depletion usually produces no symptoms, except for patients with heart disease (particularly if they are taking digitalis or undergoing cardiac surgery or patients with advanced cirrhosis.
• Treatment in this setting is directed toward replacing the lost potassium and toward treating the underlying disorder (such as vomiting or diarrhea). Treatment is usually started with 10 to 20 meq of potassium chloride given two to four times per day (20 to 80 meq per day), depending on the severity of hypokalemia and on whether hypokalemia developed acutely or is chronic.
• Potassium must be given more rapidly to patients with severe (serum potassium <2.5 to 3.0 meq/L) or symptomatic (arrhythmias, marked muscle weakness) hypokalemia.

• Potassium repletion is most easily done orally. The serum potassium concentration can transiently rise acutely by as much as 1 to 1.5 meq/L after an oral dose of 40 to 60 meq, and by 2.5 to 3.5 meq/L after 135 to 160 meq.
• Thus, potassium chloride can be given orally in doses of 40 to 60 meq, three to four times/day. If tolerated, this should be continued until the serum potassium concentration is persistently above 3.0 to 3.5 meq/L, and/or symptoms resolve; thereafter, the dose and frequency of administration can be reduced to avoid gastric irritation.

• During chronic replacement, serum potassium concentration should be monitored approximately every three to four months, or if clinically indicated.
• Potassium chloride can be given intravenously as an adjunct to oral replacement in patients who have severe symptomatic hypokalemia. Potential constraints to intravenous therapy for severe hypokalemia include a potential risk of volume overload in susceptible subjects and hyperkalemia due to excessive repletion.

• in patients with diabetic ketoacidosis or nonketotic hyperglycemia who present with hypokalemia: 40 to 60 meq/L of potassium chloride in half-isotonic saline can usually be given to supply potassium and volume repletion, with a low risk of pulmonary congestion in this setting.
• (the combination of 40 to 60 meq of potassium in half-isotonic saline is almost the osmotic equivalent of isotonic saline).

• The maximum recommended rate of intravenous potassium administration is 10 to 20 meq/h; higher rates of administration carry the risk of hyperkalemia.

• Potassium solutions with concentrations over 60 meq/L are often painful, and should be infused into a large vein (central vein).
• Asymptomatic, potassium >3 meq/L — In the absence of ongoing losses, we suggest administration of 10 to 20 meq of potassium chloride given two to four times per day (20 to 80 meq per day), depending upon the severity of hypokalemia (Grade 2B).

• Mild to moderate symptoms, or potassium <3.0 meq/L — In the absence of ongoing losses, we suggest oral administration of potassium chloride (40 to 60 meq three to four times/day, total 120 to 240 meq), until the plasma serum potassium concentration remains above 3.0 to 3.5 meq/L, and symptoms resolve (Grade 2B).
• Severe symptoms or unable to take oral medication — We recommend intravenous potassium chloride in saline solution for patients with severe symptoms of hypokalemia and in patients who are unable to take oral medications, especially if they are also receiving insulin therapy for diabetic ketoacidosis or nonketotic hyperglycemia, or bicarbonate therapy for metabolic acidosis (Grade 1B).
• When using intravenous potassium, we recommend the following:
• Do not use a dextrose-containing solution, since the associated stimulation of insulin secretion will drive potassium into the cells and limit acute correction of the hypokalemia.
• A maximal intravenous repletion rate in most cases of 10 to 20 meq/h, and maximal concentration of 100 to 200 meq/L (prepared in 100 mL). Follow the serum potassium concentration closely to avoid overcorrection.
• Continuous ECG monitoring in patients receiving potassium at 10 to 20 meq/h or faster.
Thank you