Fluids, Electrolytes, and Nutrition

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Learning Objectives:

1. Calculate the osmolarity of intravenous fluids and compare with normal plasma osmolarity.
2. Recommend an appropriate intravenous fluid regimen and monitoring parameters based on a patient's clinical characteristics.
3. Discuss the appropriate use and risks of hypertonic and hypotonic saline, and recommend a treatment regimen and monitoring parameters to ensure safe and effective use of these intravenous fluids.
4. Assess electrolyte abnormalities and recommend an appropriate pharmacologic treatment plan based on individual patient signs and symptoms.
5. Discuss appropriate indications for the use of enteral and parenteral nutrition (EN and PN).
6. Recommend a patient-specific EN formula, infusion rate, and monitoring parameters.
7. Recommend a patient-specific PN formula and monitoring plan based on the type of intravenous access, nutritional needs, comorbidities, and clinical condition.
8. Discuss strategies for preventing complications associated with EN and PN.

Self-Assessment Questions:

Answers and explanations to these questions may be found at the end of this chapter.

1. A 74-year-old woman presents to the emergency department with a 3-day history of cough, temperature to 102°F, and lethargy. She has the following vital signs and laboratory values: blood pressure (BP) 72/40 mm Hg, heart rate (HR) 115 beats/minute, urine output 10 mL/hour, white blood cell count (WBC) 18,000, hemoglobin 12.5 g/dL, blood urea nitrogen/creatinine ratio (BUN/Cr) 28/1.7 mg/dL (baseline Cr 1.2 mg/dL), blood glucose 82 mg/dL, and weight 72 kg. After a 500-mL fluid bolus of 0.9% sodium chloride (NaCl), her BP and HR are 80/46 mm Hg and 113 beats/minute. Her chest radiograph is consistent with pneumonia. Her medical history includes coronary artery disease and arthritis. Which one of the following is the most appropriate treatment?

   A. Furosemide 40 mg intravenously.
   B. 5% albumin 500 mL infused over 4 hours plus norepinephrine titrated to maintain a systolic BP of 90 mm Hg or higher.
   C. 1000-mL fluid bolus with 5% dextrose (D5W) and 0.9% NaCl.
   D. 1000-mL fluid bolus with 0.9% NaCl.

2. An order has been received for 3% NaCl. Using 0.9% NaCl and 23.4% NaCl, first determine how much of each is necessary to prepare 1 L of 3% NaCl. Second, calculate the osmolarity of 3% NaCl. Finally, determine whether the resultant solution should be administered through a central or peripheral intravenous infusion (molecular weight [MW] of NaCl is 58.5, osmotic coefficient is 0.93).

   A. Mix 907 mL of 0.9% NaCl plus 93 mL of 23.4% NaCl; osmolarity = 954 mOsm/L; central intravenous infusion.
   B. Mix 907 mL of 0.9% NaCl plus 93 mL of 23.4% NaCl; osmolarity = 477 mOsm/L; peripheral intravenous infusion.
   C. Mix 850 mL of 0.9% NaCl plus 150 mL of 23.4% NaCl; osmolarity = 954 mOsm/L; central intravenous infusion.
   D. Mix 850 mL of 0.9% NaCl plus 150 mL of 23.4% NaCl; osmolarity = 513 mOsm/L; peripheral intravenous infusion.

3. A 68-year-old man is admitted to the hospital for worsening shortness of breath during the past 2 weeks attributable to heart failure. His serum Na concentration on admission was 123 mEq/L. Other abnormal laboratory values include brain natriuretic peptide 850 and Cr 1.7 mg/dL. Chest radiograph is consistent with pulmonary edema. The patient weighs 85 kg on admission, which is up 3 kg from his baseline weight. The patient is not experiencing nausea, headache, or mental status changes. The physician orders 3% NaCl to treat the hyponatremia. Which one of the following recommendations is best?

   A. 3% NaCl is an appropriate choice because the hyponatremia is likely acute.
   B. A 250-mL bolus of 3% NaCl is appropriate if used in combination with furosemide to prevent volume overload.
   C. 3% NaCl is appropriate as long as the serum Na does not increase more than 10 mEq/L in 24 hours.

D. The risks of 3% NaCl outweigh the potential benefit for this patient.

4. A 55-year-old man with diabetes and kidney disease presents with hyperkalemia. His laboratory values include potassium (K^+) 7.2 mEq/L, calcium (Ca) 9 mg/dL, albumin 3.5, and blood glucose 302 mg/dL. His electrocardiogram (ECG) is abnormal with peaked T waves. Which one of the following is the best recommendation for initial treatment of his hyperkalemia?

A. Regular insulin 10 units intravenously plus 50 g of glucose intravenously.
B. 10% Ca^{++} gluconate 10 mL intravenously over 5 minutes.
C. Kayexalate 15 g mixed with 100 mL of 20% sorbitol every 4 hours as needed.
D. Na^+ bicarbonate 50 mEq intravenously over 5 minutes.

5. A 68-year-old patient is admitted to the hospital after a cardioembolic stroke. Her medical history is significant for atrial fibrillation, acute myocardial infarction, and diabetes. She has been unconscious for 48 hours. The medical team decides to start feeding the patient. Her weight is 60 kg, and all laboratory values including glucose concentrations are normal. Although she currently has no enteral access, she does have a peripheral intravenous catheter. Which one of the following nutritional regimens is best for this patient?

A. Initiate parenteral nutrition (PN) containing 60 g of amino acids (AA), 500 mL of 10% lipid emulsion, 300 g of dextrose, standard electrolytes, multivitamins, and trace elements in a volume of 2000 mL administered over 24 hours.
B. Initiate PN containing 40 g of AA, 500 mL of 10% lipid emulsion, 200 g of dextrose, standard electrolytes, multivitamins, and trace elements in a total volume of 2000 mL administered over 24 hours.
C. Insert a nasogastric (NG) or nasoduodenal feeding tube and infuse Isocal (1 kcal/mL) starting at 25 mL/hour and advance to a goal rate of 65 mL/hour.
D. Insert a percutaneous endoscopic gastrostomy feeding tube and infuse Isocal (1 kcal/mL) starting at 25 mL/hour and advance to a goal rate of 100 mL/hour.

6. A 70-year-old man is admitted to the hospital with peritonitis caused by severe inflammatory bowel disease. The patient has received adequate fluid resuscitation, and he is prescribed appropriate antibiotics. The physician wants the patient to have several days of bowel rest, and he or she has consulted the pharmacist to recommend a PN formula to be administered through a central line. The patient is hemodynamically stable, with normal electrolyte concentrations. Weight is 55 kg, prealbumin 20 mg/dL, BUN/Cr 20/1.1 mg/dL, and WBC 17,000. Assuming that appropriate electrolytes, multivitamins, and trace elements are included, which one of the following PN formulas when administered over 24 hours will provide this patient adequate calories, AA, and lipid?

A. AA 10% 700 mL, dextrose 30% 325 mL, lipid 20% 500 mL.
B. AA 10% 450 mL, dextrose 70% 400 mL, lipid 10% 500 mL.
C. AA 10% 800 mL, dextrose 70% 350 mL, lipid 10% 500 mL.
D. AA 15% 900 mL, dextrose 50% 500 mL, lipid 10% 500 mL.

7. A 59-year-old man has been admitted to the hospital after several days of vomiting and diarrhea. In the emergency department, he had several runs of nonsustained ventricular tachycardia. His plasma K^+ on admission was 2.8 mEq/L. After receiving 200 mEq of potassium chloride (KCl) infused over 24 hours, his repeat K^+ is 3.2 mEq/L, and he continues to have runs of ventricular tachycardia. Other laboratory values include Na^+ 143 mEq/L, magnesium 1.4 mg/dL, phosphorus 3 mg/dL, Ca^{++} 9 mg/dL, and ionized Ca^{++} 1.1 mmol/L. Which one of the following suggestions is best to treat this patient's hypokalemia?

A. Administer KCl 20 mEq intravenously over 1 hour each × 4 doses and recheck K^+.
B. Administer magnesium sulfate as a 2-g slow intravenous infusion.

C. Administer K^+ phosphate 15 mmol intravenously over 4 hours.
D. Administer Ca^{++} gluconate 2 g intravenously over 5 minutes.

8. Which one of the following nutritional strategies can prevent gut mucosal atrophy and subsequent bacterial translocation?

A. PN enriched with glutamine.
B. PN enriched with branched-chain AA.
C. Enteral nutrition (EN).
D. Zinc supplementation.

I. FLUID MANAGEMENT

A. Distribution of total body fluid (TBF).

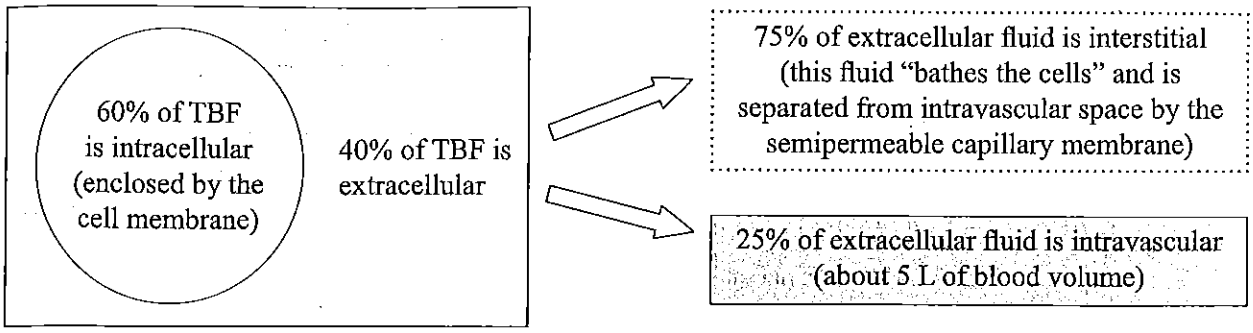

Figure 1. Distribution of total body fluid (TBF).

1. Estimated as 60% of lean body weight (LBW) in men and 50% in women; a normal adult has about 42 L of fluid
2. Total body water is further divided into intracellular (IC) space and extracellular (EC) space.
   a. Around 60% of TBF is IC, and 40% is EC; the IC and EC fluid compartments are separated by the cell membranes, which are highly permeable to water.
   b. The EC compartment is further divided into the interstitial (IS) space and the intravascular space; the IS and intravascular fluid compartments are separated by the capillary membrane, which is permeable to almost all solutes except proteins.
      i. Seventy-five percent of the EC fluid is in the IS space.
      ii. Twenty-five percent of the EC fluid is in the intravascular space; the EC fluid in the intravascular space is known as plasma and consists of about 3 L; if you also consider about 2 L of fluid found in red blood cells (thus, IC fluid), the total blood volume is around 5 L.
3. The approximate distribution of TBF into the IC and EC compartments with further distribution of the EC fluid into the IS and intravascular compartments is important to remember for determining the distribution of intravenous fluid.

B. Distribution of Intravenous Fluid
   1. Crystalloids are intravenous fluids that can contain water, dextrose, Na^+, Cl^-, and other electrolytes. Lactated Ringer's (solution) (LR) is a crystalloid that contains mostly Na^+ and Cl^-, but also lactate, K^+, and Ca^{++}.
      a. Sodium and Cl^- do not freely cross into cells, but they will distribute evenly in the EC space.
      b. For 0.9% NaCl or LR, only 25% will remain in the intravascular space, and 75% will distribute in the IS space; therefore, when 1 L of 0.9% NaCl or LR is administered, about 250 mL of fluid will remain in the intravascular compartment.
   2. "Free" water is equivalent to D_5W.
      a. D_5W is metabolized to water and carbon dioxide.
      b. Water can cross any membrane in the body; therefore, it will be evenly distributed in TBF ("free" because it is free to cross any membrane).
         i. Many experts avoid the administration of D_5W whenever possible in patients with elevated intracranial pressure (ICP) because it can cross into cerebral cells, causing a further elevation in ICP.
         ii. Some practitioners avoid the use of D_5W because of the risk of hyperglycemia, even though D_5W contains only 5 g of dextrose/100 mL, which is equivalent to 17 kcal/100 mL.

c. For D_5W, 60% will distribute to the IC space and 40% to the EC space. Of the 40% distributed to the EC space, 25% will remain in the intravascular space, and 75% will distribute to the IS space. Therefore, when 1 L of D_5W is administered intravenously, about 100 mL of fluid will remain in the intravascular compartment.

3. Colloids include packed red blood cells, pooled human plasma (5% albumin, 25% albumin, 5% plasma protein fraction), semisynthetic glucose polymers (dextran), and semisynthetic hydroxyethyl starch (hetastarch),
   a. Colloids are too large to cross the capillary membrane; therefore, they will primarily remain in the intravascular space (although a small portion will "leak" into the IS space).
   b. Except for 25% albumin, administering 500 mL of colloid will result in a 500-mL intravascular volume expansion.
   c. Because 25% albumin has an oncotic pressure about 5 times that of normal plasma, it will cause a fluid shift from the IS space into the intravascular space. For this reason, 100 mL of 25% albumin will result in about 500 mL of intravascular volume expansion. This hyperoncotic solution should generally be avoided in patients requiring fluid resuscitation, because although the intravascular space will be expanded, fluid will shift out of the IS space, potentially causing dehydration. It may be useful in patients who do not require fluid resuscitation but who could benefit from a redistribution of fluid (e.g., ascites, pleural effusions).
   d. Hydroxyethyl starch and dextran products have been associated with a coagulopathy and therefore should be avoided in patients at increased risk of hemorrhage (e.g., active bleeding, increased risk of bleeding, kidney disease).

Table 1. Distribution of Intravenous Fluid

Intravenous Fluid	Infused Volume (mL)	Equivalent Intravascular Volume Expansion (mL)
NS	1000	250
LR	1000	250
D_5W	1000	100
Albumin 5%	500	500
Albumin 25% 12.5g/50mL	100	500
Hetastarch 6%	500	500

D_5W = 5% dextrose; LS = lactated Ringer's (solution); NS = normal saline.

C. Fluid Resuscitation
   1. Intravascular fluid depletion correlates with reduced cardiac function and organ hypoperfusion.
   2. Signs or symptoms usually occur when around 15% (750 mL) of blood volume is lost (e.g., hemorrhage) or shifts out of the intravascular space (e.g., severe sepsis).
   3. Fluid resuscitation is indicated for patients with signs or symptoms of intravascular volume depletion

Table 2. Signs/Symptoms of Intravascular Volume Depletion

Tachycardia (> 100 beats/minute)
Hypotension (SBP < 80 mm Hg)
Orthostatic changes in HR or BP
Increased BUN/Cr ratio > 10:1
Dry mucous membranes
Decreased skin turgor
Reduced urine output
Dizziness
Improvement in HR and BP after a 500- to 1000-mL fluid bolus

BP = blood pressure; BUN = blood urea nitrogen; Cr = creatinine; HR = heart rate; SBP = systolic blood pressure.

4. The goal of fluid resuscitation is to restore intravascular volume and prevent organ hypoperfusion.
5. Because intravascular volume depletion can cause organ dysfunction and death, prompt resuscitation is necessary.
   a. Intravenous fluids are infused rapidly, preferably through a central venous catheter.
   b. Intravenous fluids are administered as a 500- to 1000-mL bolus, after which the patient is reevaluated; this process is continued as long as signs and symptoms of intravascular volume depletion are improving.
6. Crystalloids (0.9% NaCl or LR) are recommended for fluid resuscitation.
   a. LR is historically preferred in surgery/trauma patients, but no evidence suggests superiority over NS for fluid resuscitation.
   b. The lactate in LR is metabolized to bicarbonate and can theoretically be useful for metabolic acidosis; however, lactate metabolism is impaired during shock. Thus, it may be an ineffective source of bicarbonate.
7. Colloids have not been shown to be superior to crystalloids and are associated with a considerably higher cost. The following are examples of more common, although controversial, uses of colloids:
   a. Semisynthetic colloids may be considered after fluid resuscitation with crystalloid (usually 4–6 L) has failed to achieve hemodynamic goals or when clinically significant edema limits the further administration of crystalloid.
   b. Albumin may be considered in patients with an albumin concentration less than 2.5 g/dL who have required a large volume of resuscitation fluids and have a relative contraindication (i.e., increased risk of hemorrhage) for a semisynthetic colloid.
   c. Albumin (theoretically, 25% is preferred) may be considered in conjunction with diuretics for patients with clinically significant edema (e.g., pulmonary edema causing respiratory failure) and an albumin concentration less than 2.5 g/dL, when appropriately dosed diuretics are ineffective.

D. Maintenance Intravenous Fluids
1. Maintenance intravenous fluids are indicated in patients who are unable to tolerate oral fluids.
2. The goal of maintenance intravenous fluids is to prevent dehydration and maintain a normal fluid and electrolyte balance.
3. Maintenance intravenous fluids are typically administered as a continuous infusion through a peripheral or central intravenous catheter.
4. Common methods of estimating the daily volume in children and adults
   a. Administer 100 mL/kg for first 10 kg; then 50 mL/kg for the next 10–20 kg (i.e., 1500 mL so far) plus 20 mL/kg for every kilogram greater than 20 kg or
   b. Administer 20–40 mL/kg/day (for adults only).
   c. Adjust fluids on the basis of the individual patient's input, output, and estimated insensible loss.

5. A typical maintenance intravenous fluid is D_5W with 0.45% NaCl plus 20–40 mEq of KCl per liter. The KCl content can be adjusted for the individual patient.

Patient Cases

1. A 65-year-old man with a 3-day history of temperature to 102°F, lethargy, and productive cough is hospitalized for CAP. His medical history includes hypertension and coronary artery disease. His vital signs include HR 104 beats/minute, BP 112/68 mm Hg, and temperature 101.4°F. His weight is 80 kg, urine output 10 mL/hour, BUN 16, Cr 1.7 mg/dL, and WBC 10.4. Other laboratory values are normal. Which one of the following is most appropriate at this time?

   A. Furosemide 40 mg intravenously.
   B. Albumin 25% intravenously over 60 minutes.
   C. Hetastarch 6% 500 mL intravenously over 60 minutes
   D. D_5W/0.45% NaCl plus KCl 20 mEq/L to infuse at 110 mL/hour.

2. After 2 days of appropriate antibiotic treatment, the patient in question 1 has WBC of 9, and he is afebrile. His BP is 135/85 mm Hg, and his urine output is now 45 mL/hour. His albumin is 3.2, BUN 14, and Cr 1.4 mg/dL. All other laboratory values are normal. His appetite is still poor, and he is not taking adequate fluids. He has peripheral intravenous access. Which one of the following is most appropriate to initiate?

   A. Peripheral PN to infuse at 110 mL/hour.
   B. Albumin 5% 500 mL intravenously over 60 minutes.
   C. D_5W/0.45% NaCl plus KCl 20 mEq/L to infuse at 110 mL/hour.
   D. LR solution to infuse at 110 mL/hour.

II. OSMOLALITY

A. Plasma Osmolality Is Normally Between 275 and 290 mOsm/kg.
   1. Terminology clarification
      a. Osmolality is a measure of the osmoles of solute per kilogram of solvent (Osm/kg), whereas osmolarity is a measure of osmoles of solute per liter of solution (Osm/L).
      b. Plasma osmolarity (mOsm/L) can be calculated by the following equation: osmolarity = osmolality × 0.995, illustrating that there is no clinically significant difference between them (i.e., plasma osmolarity is about 1% lower than plasma osmolality).
   2. Plasma osmolality is maintained within a normal range by thirst and secretion of arginine vasopressin (i.e., ADH) from the posterior pituitary.
   3. Sodium salts are the primary determinant of plasma osmolality and therefore regulate fluid shifts between the IC and EC fluid compartments.
   4. Plasma osmolality (in milliosmoles per kilogram) can be estimated: $(2 \times Na^+) + (\text{glucose}/18) + \text{BUN}/2.8$.
   5. Increases in plasma osmolality cause an osmotic shift of fluid into the plasma, resulting in cellular dehydration and shrinkage. (hypertonic solutions)
   6. Decreases in plasma osmolality cause an osmotic shift of fluid into cells, resulting in cellular overhydration and swelling. (hypotonic solutions)

B. Intravenous Fluids Can Be Classified by Their Osmolarity Relative to Plasma.
   1. Isotonic fluid will not result in a fluid shift between fluid compartments because the osmolarity is similar to plasma.
   2. Hypertonic fluid can cause fluid to shift from the IC to the EC compartment with subsequent cellular dehydration and shrinkage.
   3. Hypotonic fluid with an osmolarity less than 150 mOsm/L can cause fluid to shift from the EC to the IC compartment with subsequent cellular overhydration and swelling.
      a. Red blood cell swelling can cause cell rupture (i.e., hemolysis).
      b. Brain cells can swell, causing cerebral edema and herniation; this is most likely to occur with acute hyponatremia (occurring in less than 2 days).

C. Definitions
   1. Equivalent weight = MW divided by valence
      a. A milliequivalent (mEq) = 1/1000 of an equivalent.
      b. Examples of equivalent weight

Table 3. Electrolyte MW, Valence, and Equivalent Weight

Electrolyte	MW	Valence	Equivalent Weight (g)
Sodium	23	1	23
Potassium	39	1	39
Chloride	35.5	1	35.5
Magnesium	24	2	12

MW = molecular weight.

   2. Osmoles = number of particles in solution (assuming complete dissociation)
      a. A milliosmole = 1/1000 of an osmole.
      b. Examples of osmoles

Table 4. Osmoles

Salt	Osmoles
NaCl	2
KCl	2
$CaCl_2$	3

$CaCl_2$ = calcium chloride; KCl = potassium chloride; NaCl = sodium chloride.

   3. Converting MW to milliequivalents

Table 5. Converting Molecular Weight to Milliequivalents

Convert 23.4% NaCl (concentrated NaCl) to mEq/mL
Molecular weight (MW) of NaCl = 23 + 35.5 = 58.5 (add MW of Na + Cl)
$\frac{23.4\text{ g}}{100\text{ mL}} \times \frac{1\text{ equiv}}{58.5\text{ g}} \times \frac{1000\text{ mEq}}{1\text{ equiv}} = 4\text{ mEq/mL}$

D. Calculate the Osmolarity of Intravenous Fluids in Milliosmoles per Liter.

1. The osmotic coefficient can be used to calculate the osmolarity of intravenous fluids because salt forms do not completely dissociate in solution.
   a. With NaCl, for example, there is some ionic attraction between Na^+ and Cl, so they do not completely dissociate, but rather, they are about 93% dissociated in solution (thus, the osmotic coefficient is 0.93).
   b. In clinical practice, most do not consider the osmotic coefficient when calculating the osmolarity of NaCl or other electrolytes, and in reality, the osmotic coefficient is probably not clinically relevant (but is used in the following examples for completeness).
2. Normal saline (NS; 0.9% NaCl)

Table 6. Calculation for NS

MW	Osmoles	Osmotic Coefficient
58.5 g/mol	2	0.93

$$\frac{0.9\text{ g}}{100\text{ mL}} \times \frac{1\text{ mol}}{58.5\text{ g}} \times \frac{2\text{ Osm}}{1\text{ mol}} \times \frac{1000\text{ mOsm}}{1\text{ Osm}} \times \frac{1000\text{ mL}}{1\text{ L}} \times 0.93 = 287\text{ mOsm/L}$$

3. D_5W (MW 180 g/mol)

Table 7. Calculation for D_5W

$$\frac{5\text{ g}}{100\text{ mL}} \times \frac{1\text{ mol}}{180\text{ g}} \times \frac{1000\text{ mOsm}}{1\text{ mol}} \times \frac{1000\text{ mL}}{1\text{ L}} = 278\text{ mOsm/L}$$

4. Osmolarity of D_5W/NS = 287 mOsm/L + 278 mOsm/L = 565 mOsm/L
5. Osmolarity of NS + KCl 20 mEq/L

Table 8. Calculation for NS Plus KCl

Step 1: Convert mEq to weight (g)

$$20\text{ mEq} \times \frac{1\text{ equiv}}{1000\text{ mEq}} \times \frac{74.5\text{ g}}{1\text{ equiv}} = 1.49\text{ g of KCl}$$

Step 2: Calculate mOsm/L

$$\frac{1.49\text{ g}}{\text{L}} \times \frac{1\text{ mol}}{74.5\text{ g}} \times \frac{2\text{ Osm}}{1\text{ mol}} \times \frac{1000\text{ mOsm}}{1\text{ Osm}} = 40\text{ mOsm/L}$$

Step 3: Add osmolarity of NS + KCl = 287 mOsm/L + 40 mOsm/L = 327 mOsm/L

III. HYPERTONIC SALINE

A. Concentration: Typically prepared as 3% (954 mOsm/L), 7.5% (2393 mOsm/L), or 23.4% (7462 mOsm/L)

B. Common Uses of Hypertonic Saline (HS)
   1. HS is used in traumatic brain injury to reduce an elevated ICP and to increase BP.
      a. Typically used if ICP is greater than 15 mm Hg using an ICP monitor
      b. If the serum Na concentration is close to the upper limit of normal (i.e., 145 mEq/L), then it is preferable to use a lower concentration of HS (i.e., 3%).
   2. HS is used for symptomatic hyponatremia (symptoms described in hyponatremia section below).
      a. Symptoms generally do not occur unless serum Na^+ is 120 mEq/L or less, and they increase in severity as Na^+ decreases.
      b. In an effort to prevent severe symptoms from occurring, some practitioners treat asymptomatic hyponatremia if the serum Na^+ is 120 mEq/L or less because of the increased risk of symptoms below this level.

C. Inappropriate Use of HS
   1. Chronic asymptomatic hyponatremia
      a. Asymptomatic syndrome of inappropriate secretion of antidiuretic hormone (ADH) is usually treated with fluid restriction of 1000–1200 mL of fluid per day.
      b. Hyponatremia is generally a water problem (i.e., an excess of free water) rather than a deficiency of Na; thus, HS makes little sense in the absence of symptoms (see Hyponatremia section below).
   2. Hyponatremia associated with severe hyperglycemia (i.e., diabetic ketoacidosis)
      a. Typically, serum Na will decrease in a non-linear fashion in response to hyperglycemia (i.e., Na^+ decreases by about 1.6 mEq/L for every 100-mg/dL elevation in glucose between 100 and 400 mg/dL, but Na^+ will decrease by about 2.4 mEq/L for every 100-mg/dL elevation in glucose above 400 mg/dL).
      b. As hyperglycemia is corrected with insulin, the serum Na^+ will normalize.
   3. Hyponatremia associated with hypervolemia (i.e., heart failure leads to tissue hypoperfusion, which triggers the secretion of ADH, causing reabsorption of water from the kidneys and leading to hyponatremia)
      a. In general, this situation is treated with fluid restriction.
      b. Symptomatic hyponatremia is uncommon in patients with heart failure.
      c. HS could be considered in symptomatic patients; however, they may need diuresis to prevent worsening volume overload.

D. Preparation of HS

Table 9. Calculations to Prepare HS

Steps	Example
Choose base solutions	For this example, use concentrated NaCl available as 23.4% vials and sterile water to make 1000 mL of 7.5% HS
Set up alligation	23.4% → 7.5% → ; 0 → 7.5% →
Add and subtract	23.4% → 7.5% → 7.5 parts (from 23.4% NaCl); 0 → 7.5% → 15.9 parts (from sterile water); 23.4 parts total
Divide	7.5 parts/23.4 parts = x/1000 mL; x = 320.5 mL of 23.4% NaCl 15.9 parts/23.4 parts = x/1000 mL; x = 679.5 mL of sterile water

HS = hypertonic saline; NaCl = sodium chloride.

E. HS Dose

1. Dose options for traumatic brain injury
   a. 3% HS 250 mL or 2–4 mL/kg intravenously over 1–15 minutes administered for elevated ICP
   b. 23.4% HS 30 mL over 20–30 minutes administered for elevated ICP
      i. Standing orders such as 30 mL every 4 or 6 hours are NOT recommended.
      ii. If HS is needed for prolonged reduction in ICP, a 3% HS concentration is generally recommended.
2. Dose options for patients with symptomatic hyponatremia
   a. Treatment of patients with symptomatic hyponatremia involves a small but quick increase in serum Na^+ by 0.75–1 mEq/L/hour to a concentration of 120 mEq/L. Then, infusion can be reduced so that Na^+ increases by 0.5 mEq/L/hour.
   b. Estimate an infusion rate of 3% HS by multiplying ideal body weight (IBW) by desired rate of serum Na increase per hour. (Note: IBW is used to avoid overdosing obese patients.)
      i. For example, 70 kg × 1 mEq/L/hour = 70 mL/hour to increase serum Na^+ by 1 mEq/L in 1 hour. The infusion can be adjusted to achieve goal changes in serum Na^+.
      ii. Infusion rate of 3% HS is generally 1–2 mL/kg/hour.
      iii. In general, 3% HS is not recommended in asymptomatic patients; if used in an asymptomatic patient, the administration rate should generally not exceed 0.5–1 mL/kg/hour.
   c. Alternatively, some practitioners recommend a 250-mL bolus of 2%–3% HS over 30 minutes or 50 mL of 3% HS administered as a bolus every 30 minutes for two doses.

F. Administration of HS

1. Use central intravenous access because the osmolarity is greater than 900 mOsm/L.
2. If no central line is available, can use 2% HS
3. Some practitioners use 3% HS through a peripheral intravenous access site in an emergency because the osmolarity is close to the cutoff range for peripheral administration. If a peripheral site is used, monitor for phlebitis and obtain central access as soon as possible.

G. Clinical Goals and Monitoring for Administering HS in Patients with Symptomatic Hyponatremia

1. Goals
   a. Stop symptoms (described below).
   b. Safe serum Na^+ achieved usually in the range of 120–125 mmol/L to avoid adverse neurologic outcomes. Note that the immediate goal for patients with symptomatic hyponatremia is not a normal serum Na^+.
   c. Reached maximal safe amount of change in serum Na^+
      i. Maximal safe amount of change is generally regarded as 10–12 mmol/L (or 10–12 mEq/L) in 24 hours.
      ii. Some practitioners suggest a maximal change of 8 mmol/L in 24 hours.
2. Monitor serum Na^+ every 1–4 hours depending on severity of symptoms.

H. Complications of HS
1. Central pontine myelinolysis (also referred to as osmotic demyelination syndrome) can occur with rapid correction of hyponatremia.
   a. Characterized by permanent neurologic damage such as paraparesis, quadriparesis, dysarthria, dysphagia, and coma
   b. More likely to occur with rapid correction of chronic hyponatremia compared with acute hyponatremia. This partly explains why it is advisable not to administer HS in patients with chronic asymptomatic hyponatremia.
   c. Prevent by avoiding changes in serum Na^+ of more than 10–12 mmol/L in 24 hours or more than 18 mmol/L in 48 hours.
2. Hypokalemia can occur with large volumes of HS.
3. Hyperchloremic acidosis can occur because of the administration of Cl^- salts (i.e., NaCl). Can prevent by administering HS in a 1:1 or 2:1 ratio of NaCl and Na^+ acetate
4. Hypernatremia
5. Phlebitis if administered in a peripheral vein
6. Heart failure
   a. Fluid overload can occur because of initial volume expansion.
   b. Over time, HS can have a diuretic effect, leading to intravascular volume depletion.
7. Coagulopathy caused by platelet dysfunction
8. Hypotension if HS is administered rapidly

I. Other Considerations When Using HS
1. Because hypokalemia can cause hyponatremia, remember to correct K^+ depletion if present. As K^+ is replaced, serum Na^+ will increase.
2. If 150 mEq of Na^+ bicarbonate is added to 850 mL of 0.9% NaCl, the resultant solution is equivalent to about 3% HS. When an infusion of 150 mEq of Na^+ bicarbonate per liter is indicated, it is recommended to add Na^+ bicarbonate to D_5W instead of 0.9% NaCl.

IV. HYPOTONIC INTRAVENOUS FLUIDS

A. Hypotonic Fluids Administered Intravenously Can Cause Cell Hemolysis and Patient Death.
1. Albumin 25% diluted with sterile water to make albumin 5% has an osmolarity of about 60 mOsm/L and can cause hemolysis.
2. "Quarter NS" or 0.225% NaCl has an osmolarity of 68 mOsm/L and can cause hemolysis.

B. Avoid Using Intravenous Fluid with an Osmolarity Less than 150 mOsm/L.

1. Sterile water should NEVER be administered intravenously.
2. Some prescribers use hypotonic saline for a patient with hypernatremia.
   a. In reality, a patient with hypernatremia generally needs water, not Na^+.
   b. Therefore, for patients with hypernatremia, enteral administration of water is preferable.
   c. If the enteral route is unavailable, recommend D_5W administered intravenously.

C. Prevent a Potentially Fatal Error by Recommending One of the Following Alternatives in Place of 0.225% NaCl:
1. Recommend changing 0.225% NaCl to D_5W alone or a combination of D_5W and 0.225% NaCl.
2. Alternatively, if there are concerns related to hyperglycemia with using D_5W (50 g of dextrose or 170 kcal/L), recommend using 2.5% dextrose and 0.225% NaCl.
3. Alternatively, could recommend adding KCl to increase osmolarity
4. Recommend administering water enterally (by mouth or feeding tube)

V. HYPONATREMIA AND HYPO-OSMOLAL STATES

A. Sodium Salts Are the Primary Determinants of Plasma Osmolality (and subsequent fluid shifts between the IC and EC compartments).
1. A reduction in serum Na^+ less than 136 mEq/L usually correlates with a reduction in plasma osmolality.
2. Hyponatremia with subsequent hypo-osmolality causes fluid to shift into cells (cellular overhydration). Hypotonic hyponatremia can be divided into three types on the basis of volume status:

Table 10. Classification of Hyponatremia

	Hypervolemic Hyponatremia	Normovolemic Hyponatremia	Hypovolemic Hyponatremia
Description	Caused by excess Na^+ and fluid but fluid excess predominates	Normal total body Na^+ with excess fluid volume (i.e., dilutional)	Deficit of both Na^+ and fluid, but total Na^+ is decreased more than total body water
Example	Heart failure, cirrhosis, nephrotic syndrome	SIADH	Fluid loss (e.g., emesis, diarrhea, fever), third-spacing, renal loss (diuretics)
Diagnosis	Urine $Na^+ < 25$ mEq/L indicates edematous disorders (i.e., heart failure, cirrhosis, nephritic syndrome); Urine $Na^+ > 25$ mEq/L indicates acute or chronic renal failure	Urine osmolality > 100 mOsm/Kg (indicates impaired water excretion); urine Na > 40 mEq/L	Urine $Na^+ < 25$ mEq/L indicates a nonrenal loss of Na^+ (e.g., emesis, diarrhea); urine $Na^+ >$ 40 mEq/L indicates a renal loss of Na^+
Treatment	Sodium and water restriction; treat underlying cause; vasopressin receptor antagonists (e.g., conivaptan, tolvaptan)	If drug-induced SIADH, remove offending agent; fluid restriction; demeclocycline; vasopressin receptor antagonists (e.g., conivaptan, tolvaptan)	Fluid resuscitation (see above)

SIADH = syndrome of inappropriate secretion of antidiuretic hormone.

3. In select cases, hyponatremia will be associated with either a normal or elevated plasma osmolality.
   a. This is known as pseudohyponatremia because Na^+ content in the body is not actually reduced, but rather, shifts from the EC compartment into the cells to maintain plasma osmolality in a normal range (thus, serum Na^+ will be low).
      i. Severe hyperlipidemia can be associated with a normal or elevated plasma osmolality.
      ii. Severe hyperglycemia (i.e., during diabetic ketoacidosis) is associated with an elevated plasma osmolality. + hypokalemia too
   b. Once the underlying condition is corrected, Na^+ will shift out of the cells, and hyponatremia will resolve.

B. Causes of Hyponatremia
   1. Replacement of lost solute with water
      a. Loss of solute (e.g., vomiting, diarrhea) usually involves the loss of isotonic fluid; therefore, alone, it will not cause hyponatremia.
      b. After the loss of isotonic fluid, hyponatremia can develop when the lost fluid is replaced with water.
      c. One of the most common causes of hyponatremia in hospitals is the postoperative administration of hypotonic fluid.
   2. Volume depletion and organ hypoperfusion stimulate ADH secretion to increase water reabsorption in the collecting tubules, potentially causing hyponatremia.
   3. Syndrome of inappropriate ADH secretion and cortisol deficiency are both related to excessive release of ADH.
   4. Medications, including thiazide diuretics and antidepressants (especially selective serotonin reuptake inhibitors, but also tricyclic antidepressants), can cause hyponatremia. Drug-induced hyponatremia is more likely in elderly patients and in those who drink large volumes of water.
   5. Renal failure impairs the ability to excrete dilute urine, predisposing to hyponatremia.

C. Symptoms of Hyponatremia

Table 11. Symptoms of Hyponatremia

Serum Sodium (mEq/L)	Clinical Manifestations
120–125	Nausea, malaise
115–120	Headache, lethargy, obtundation, unsteadiness, confusion
110–115	Delirium, seizure, coma, respiratory arrest, death

   1. Symptoms are generally attributable to hypo-osmolality, with subsequent water movement into brain cells causing cerebral overhydration.
   2. If hyponatremia occurs chronically, cerebral cell swelling is prevented by osmotic adaptation.
      a. Solutes move out of brain cells to prevent the osmotic shift of water into brain cells.
      b. For this reason, patients with chronic hyponatremia may show less severe or no symptoms.
   3. Neurologic symptoms are related to the rate of change in the serum Na^+ and to the degree of change in serum Na^+.
   4. Acute hyponatremia occurs over 1–3 days.

D. Treatment of Hyponatremia
   1. Treat underlying cause.
   2. Raise serum Na^+ at a safe rate, defined as a change no greater than 10–12 mEq/L in 24 hours.

3. Treatment depends on volume status, the presence and severity of symptoms, and the onset of hyponatremia (the latter two have been discussed previously).
   a. If the patient is normovolemic or edematous, there are two treatment options:
      i. Fluid restriction is the typical first-line recommendation. Note that Na administration is not recommended and that it can worsen edema.
      ii. Vasopressin antagonists (e.g., intravenous conivaptan, oral tolvaptan) can be used in normovolemic (i.e., SIADH [syndrome of inappropriate secretion of antidiuretic hormone]) or hypervolemic (i.e., heart failure) patients to promote aquaresis, increase serum Na^+, alleviate symptoms, and reduce weight; however, this approach is costly and has not been shown to improve clinical outcomes (i.e., fall prevention, hospitalization, hospital length of stay, mortality) in prospective randomized controlled trials. Vasopressin antagonists are substrates and inhibitors of cytochrome P450 3A4 isoenzymes; monitor for drug interactions with other 3A4 inhibitors that could increase effect and lead to a rapid increase in serum Na^+. Fluid restriction in combination with a vasopressin antagonist during the first 24 hours can also increase the risk of overly rapid correction of serum Na^+. If needed, fluid restriction can be used after 24 hours.
   b. If patient has intravascular volume depletion, need to replace volume first with intravenous crystalloids (e.g., 0.9% NaCl)
      i. Until intravascular volume is restored, patient will continue to secrete ADH, causing water reabsorption and subsequent hyponatremia.
      ii. Once intravascular volume is restored, ADH secretion will decrease, causing water to be excreted. This can lead to a rapid correction of serum Na^+; careful monitoring is required to prevent overly rapid correction.
      iii. Volume status can be assessed by skin turgor, jugular venous pressure, and urine Na^+.
   c. Once intravascular volume is restored, may still need to administer Na^+ to patients who experienced volume depletion, diuretic-induced hyponatremia, or adrenal insufficiency
      i. The amount of Na^+ (in milliequivalents) needed to raise the serum Na^+ to a safe concentration of about 120 mEq/L is estimated using LBW as follows: $0.5(LBW) \times (120 - Na^+)$ for women (multiply LBW by 0.6 for men). LBW has been estimated using weight in kilograms and height in centimeters for males as LBW = [(0.3)(kg) + (0.3)(cm) − 29] or for females as LWB = [(0.3)(kg) + (0.4)(cm) −43]; formula published in 1966 (J Clin Pathol 1966;19:389)
      ii. Alternatively, the equation above can be modified to estimate the Na^+ deficit in the following manner: $0.5(LBW) \times (140 - Na^+)$ for women (multiply LBW by 0.6 for men). If calculating the Na^+ deficit, it is recommended to administer 25%–50% of the deficit during the first 24 hours to prevent the overly rapid correction of serum Na^+.
      iii. Regardless of the method used to estimate Na^+ replacement, the amount of Na^+ administered should be guided by serial serum Na^+ concentrations.
   d. Patients with symptomatic hyponatremia should be treated with HS (see section on HS).
4. Correct hypokalemia if present with hyponatremia.
   a. Hypokalemia will cause a reduction in serum Na^+ because Na^+ enters cells to account for the reduction in IC K^+ to maintain cellular electroneutrality.
   b. Administration of K^+ will correct hyponatremia.
   c. Use caution when giving K^+ to prevent overly rapid correction of serum Na^+.

Patient Cases

3. A 72-year-old woman with a history of hypertension has developed hyponatremia after starting hydrochlorothiazide 3 weeks earlier. She experiences dizziness, fatigue, and nausea. Her serum Na^+ is 116 mEq/L. Her weight is 60 kg, BP is 86/50 mm Hg, and HR is 122 beats/minute. Which one of the following initial treatment regimens is most recommended?

   A. 0.9% NaCl infused at 100 mL/hour.
   B. 0.9% NaCl 500-mL bolus.
   C. 3% NaCl infused at 60 mL/hour.
   D. 23.4% NaCl 30-mL bolus as needed.

4. For the patient described above, which one of the following is the best treatment goal for the first 24 hours in correcting her serum Na^+ from her initial value of 116 mEq/L?

   A. Increase Na^+ concentration to 140 mEq/L.
   B. Increase Na^+ concentration to 132 mEq/L.
   C. Increase Na^+ concentration to 126 mEq/L.
   D. Maintain serum Na^+ between 116 mEq/L and 120 mEq/L.

5. One day later, the patient from the question above has improved somewhat. Her BP is now 122/80 mm Hg, and her HR is 80 beats/minute. Her serum Na^+ is 120 mEq/L, and K^+ is 3.2 mEq/L; she still feels tired. She is eating a regular diet. Her ECG is normal. Which one of the following is the best recommendation?

   A. D_5W/0.9% NaCl plus KCl 40 mEq/L to infuse at 100 mL/hour.
   B. 0.9% NaCl infused at 100 mL/hour.
   C. 3% NaCl infused at 60 mL/hour.
   D. Micro-K 20 mEq by mouth every 6 hours × 4 doses.

VI. HYPERNATREMIA AND HYPEROSMOLAL STATES

A. Serum Na^+ Greater than 145 mEq/L Generally Causes Hyperosmolality.
   1. The osmotic gradient associated with hypernatremia causes water movement out of cells and into the EC space.
   2. Symptoms are primarily related to the dehydration of brain cells.

B. Causes of Hypernatremia
   1. Loss of water because of fever, burns, infection, renal loss, gastrointestinal (GI) loss
   2. Retention of Na^+ because of the administration of HS or any form of Na^+

C. Prevention of Hypernatremia Through Osmoregulation
   1. Plasma osmolality is maintained between 275 and 290 mOsm/kg despite changes in water and Na^+ intake.
   2. Hypernatremia is prevented first by the release of ADH, causing water reabsorption.
   3. Hypernatremia is also prevented by thirst.
      a. Hypernatremia primarily occurs in adults with altered mental status who have an impaired thirst response or do not have access to or the ability to ask for water.
      b. Hypernatremia can also occur in infants.

D. Cerebral Osmotic Adaptation
   1. Similar to patients with hyponatremia, patients with chronic hypernatremia can have cerebral osmotic adaptation.
      a. Brain cells take up solutes, Na^+, and K^+, thus limiting the osmotic gradient between the IC and EC fluid compartments.
      b. This prevents cellular dehydration, and it will increase the brain volume toward a normal value despite hypernatremia.
   2. Because of osmotic adaptation, patients with chronic hypernatremia may be relatively asymptomatic.

E. Symptoms of Hypernatremia Are Primarily Neurologic.
   1. Similar to hyponatremia, the symptoms of hypernatremia are related to the rate of increase in plasma osmolality and the degree of increase in plasma osmolality.
   2. Earlier symptoms include lethargy, weakness, and irritability.
   3. Symptoms can progress to twitching, seizures, coma, and death if the serum Na^+ is greater than 158 mEq/L.
   4. Cerebral dehydration can cause cerebral vein rupture with subsequent intracerebral or subarachnoid hemorrhage.

F. Treatment of Hypernatremia
   1. Rapid correction of chronic hypernatremia can result in cerebral edema, seizure, permanent neurologic damage, and death.
      a. With osmotic adaptation, the brain volume is raised toward normal despite an elevated serum osmolarity.
      b. Osmotic adaptation combined with a rapid reduction in plasma osmolality can cause an osmotic gradient, causing water to move into brain cells with subsequent cerebral edema.
   2. In patients with symptomatic hypernatremia, serum Na^+ should be reduced slowly by no more than 0.5 mEq/L/hour or 12 mEq/L/day.
   3. Treat hypernatremia by replacing water deficit slowly over several days to prevent overly rapid correction of serum Na^+.
      a. Using LBW, the estimated water deficit (in liters) = (0.4 × LBW) × [(serum Na^+/140) − 1] in women (multiply LBW by 0.5 in men).
      b. Note that in women and men, the total body water is typically about 50% and 60% of LBW. Thus, some sources recommend a variation on the above equation as follows: water deficit = (0.5 × LBW) × [(serum Na^+/140) − 1] in women (multiply LBW by 0.6 in men). However, hypernatremic patients are generally water depleted; thus, the equation using the lower values above (i.e., 40% or 0.4 and 50% or 0.5) is reasonable.
   4. Administer free water orally or intravenously as D_5W.
   5. If concurrent Na^+ and water depletion (e.g., vomiting, diarrhea, diuretic-induced depletion), can use a combination of D_5W and 0.25% NaCl
   6. If patient is hypotensive because of volume depletion, first restore intravascular volume with 0.9% NaCl to restore tissue perfusion. NS is the preferred crystalloid for fluid resuscitation, and in the hypernatremic patient, it is still relatively hypotonic.

Patient Case

6. A 74-year-old woman has been receiving Jevity tube feedings at 60 mL/hour for the past 8 days through her gastrostomy feeding tube. She recently suffered an ischemic stroke; she is responsive, but she does not communicate. Her serum Na^+ was 142 mg/dL on the day the Jevity was started, and it has risen steadily to 149, 156, and 163 mg/dL on days 3, 4, and 8, respectively, after the start of the tube feedings. Her weight is 50 kg. Which one of the following is the best treatment for her hypernatremia?

   A. Administer sterile water intravenously at 80 mL/hour.
   B. Administer D_5W intravenously at 80 mL/hour.
   C. Administer D_5W/0.2% NaCl intravenously at 80 mL/hour.
   D. Administer water by enteral feeding tube 200 mL every 6 hours.

VII. DISORDERS OF K^+

A. Normal Plasma K^+ Concentrations Are 3.5–5 mEq/L.

B. Potassium Is the Primary IC Cation (maintains electroneutrality with Na, the primary EC cation).

C. Potassium Balance Is Maintained Between the IC and EC Compartments by Several Factors, Including:
   1. β_2-adrenergic stimulation (caused by epinephrine) promotes cellular uptake of K^+.
   2. Insulin promotes cellular uptake of K^+.
   3. Plasma K^+ concentration directly correlates with movement of K^+ in and out of cells owing to passive shifts based on the concentration gradient across the cell membrane. (A normal response to diarrhea-induced hypokalemia is for K^+ to shift out of the cells passively, minimizing the reduction in plasma K^+ concentration.)

D. Normal Plasma Concentrations of K^+ Are Maintained by Renal Excretion.

E. Hypokalemia (K^+ concentration less than 3.5 mEq/L)
   1. Causes of hypokalemia
      a. Reduced intake seldom causes hypokalemia because renal excretion is minimized because of increased renal tubular absorption.
      b. Increased shift of K^+ into cells can occur with the following:
         i. Increased pH
         ii. Insulin or a carbohydrate load
         iii. β_2-receptor stimulation caused by stress-induced epinephrine release or administration of a β-agonist (e.g., albuterol, dobutamine)
         iv. Hypothermia
      c. Increased GI losses of K^+ can occur with vomiting, diarrhea, intestinal fistula or enteral tube drainage, and chronic laxative abuse.
      d. Increased urinary losses can occur with mineralocorticoid excess (e.g., aldosterone) and diuretic use (e.g., loop and thiazide type).
      e. Hypomagnesemia is commonly associated with hypokalemia caused by increased renal loss of K^+; correction of plasma K^+ requires simultaneous correction of serum magnesium.

2. Symptoms of hypokalemia generally occur when plasma K^+ is below 3 mEq/L and can include the following.
   a. Muscle weakness occurs most commonly in the lower extremities but can progress to the trunk, upper extremities, and respiratory muscles. Muscle weakness in the GI tract can manifest as paralytic ileus, abdominal distention, nausea, vomiting, and constipation.
   b. ECG changes (flattened T waves or elevated U wave)
   c. Cardiac arrhythmias (bradycardia, heart block, ventricular tachycardia, ventricular fibrillation)
   d. Digoxin toxicity can occur despite normal serum digoxin concentrations in the presence of hypokalemia.
   e. Rhabdomyolysis can occur because hypokalemia can cause reduced bloodflow to skeletal muscle.
3. Treatment of hypokalemia
   a. Potassium deficit can be estimated as 200–400 mEq of K^+ for every 1-mEq/L reduction in plasma K^+ (assuming normal distribution of K^+ between EC and IC compartments).
   b. Although the K^+ deficit can be estimated, K^+ replacement is guided by K^+ concentrations; recheck every 2–4 hours if K^+ is less than 3 mEq/L.
   c. Potassium Cl^- is the preferred salt in patients with concurrent metabolic alkalosis because these patients typically lose Cl^- through diuretics or GI loss. This is the most common presentation of hypokalemia.
   d. Potassium acetate can be administered intravenously, or K^+ bicarbonate can be administered orally for patients with a metabolic acidosis that requires frequent K^+ supplementation.
   e. Guidelines for administering K^+
      i. Patients without ECG changes or symptoms of hypokalemia can be treated with oral supplementation.
      ii. Avoid mixing K^+ in dextrose, which can cause insulin release with a subsequent IC shift of K^+.
      iii. To avoid irritation, no more than about 60 mEq/L should be administered through a peripheral vein.
      iv. Recommended infusion rate is 10–20 mEq/hour to a maximum of 40 mEq/hour (the faster rate should be infused through a central venous catheter).
      v. Patients who receive K^+ at rates faster than 10–20 mEq/hour should be monitored using a continuous ECG.

Table 12. K^+ Replacement

Plasma K^+ (mEq/L)	Treatment[a]	Comments
3–3.5	Oral KCl 60–80 mEq/day if no signs or symptoms (doses greater than 60 mEq should be divided to avoid GI adverse effects)	Recheck K^+ daily
2.5–3	Oral KCl 120 mEq/day or IV 60–80 mEq administered at 10–20 mEq/hour if signs or symptoms	Monitor K^+ closely (i.e., 2 hours after infusion)
2–2.5	IV KCl 10–20 mEq/hour	Consider continuous ECG monitoring
Less than 2	IV KCl 20–40 mEq/hour	Requires continuous ECG monitoring

[a]Treatment doses are for patients with normal kidney function and should be reduced for patients with kidney dysfunction or elderly patients.
ECG = electrocardiogram; GI = gastrointestinal; IV = intravenous; KCl = potassium chloride; K^+ = potassium.

F. Hyperkalemia
1. Causes of hyperkalemia
   a. Increased intake
   b. Shift of K^+ from the IC to the EC compartment causes hyperkalemia and can occur with the following:
      i. Acidosis
      ii. Insulin deficiency
      iii. β-Adrenergic blockade
      iv. Digoxin overdose
      v. Rewarming after hypothermia (e.g., after cardiac surgery)
      vi. Succinylcholine
   c. Reduced urinary excretion can occur with
      i. Kidney dysfunction
      ii. Intravascular volume depletion
      iii. Hypoaldosteronism
      iv. Potassium-sparing diuretics
      v. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers
2. Symptoms of hyperkalemia
   a. Muscle weakness or paralysis is caused by changes in neuromuscular conduction and typically occurs when plasma K^+ exceeds 8 mEq/L.
   b. Abnormal cardiac conduction can first manifest as peaked, narrowed T waves (typically, when plasma K^+ exceeds 6 mEq/L), widening of the QRS, and can progress to ventricular fibrillation and asystole.
   c. Not all patients will experience ECG changes, and the initial manifestation of hyperkalemia can be ventricular fibrillation; thus, consider emergency treatment even in patients with no ECG changes if plasma K^+ exceeds 6.5 mEq/L.
   d. Conduction disturbances are enhanced by hypocalcemia, hyponatremia, acidosis, and rapid elevation in the plasma K^+ concentration.
3. Pseudohyperkalemia should be suspected if there is no apparent cause or symptoms of hyperkalemia.
   a. Can occur if K^+ is released from cells while – or after – obtaining the blood specimen; usually because of trauma during venipuncture
   b. Can occur from measurement of the serum rather than the plasma K^+ concentration; caused by K^+ release during coagulation
4. Treatment of hyperkalemia
   a. Patients with an asymptomatic elevation in the plasma K^+ who do not have signs or symptoms can be treated with a cation exchange resin (e.g., Na polystyrene sulfonate) alone.
   b. Urgent and immediate treatment is required for patients with the following signs or symptoms:
      i. Plasma K^+ above 6.5 mEq/L
      ii. Severe muscle weakness
      iii. ECG changes
   c. Calcium should be administered intravenously to patients with symptomatic hyperkalemia to prevent hyperkalemia-induced arrhythmias even if patients are normocalcemic.
      i. Calcium gluconate can be administered peripherally and is preferred over calcium chloride (CaCl) because of a reduced risk of tissue necrosis; dose is 10 mL (equivalent to 1 g, 90 mg elemental, or 4.65 mEq) of 10% Ca^{++} gluconate administered over 2–10 minutes; may repeat in 5 minutes if no improvement in ECG. Calcium chloride can be used if central intravenous access is available; however, the dose should be adjusted because 10 mL (1 g, 270 mg elemental, or 13.6 mEq) provides 3 times the amount of elemental Ca as calcium gluconate.

ii. Onset is within minutes, but duration is short (30–60 minutes).
iii. Will not reduce plasma K^+, but will antagonize the effect of K^+ in cardiac conduction cells
iv. Use in urgent circumstances while waiting for other measures (e.g., insulin and glucose) to lower plasma K^+.
v. Avoid use in patients receiving digoxin because hypercalcemia can precipitate digoxin toxicity, and there are reports of sudden death.

d. The following treatment options are transient, causing a temporary shift of K^+ from the EC fluid into the cells, and should be used for symptomatic hyperkalemia.
   i. Insulin and glucose
      (a) Dose is regular insulin 10 units intravenously plus 25–50 g of glucose administered as a 50% dextrose intravenous push to prevent hypoglycemia.
      (b) Typically lowers plasma K^+ by 0.5–1.5 mEq/L within 1 hour and may last for several hours
      (c) If patients are hyperglycemic, insulin alone can be administered.
      (d) More predictable in patients with kidney failure than sodium bicarbonate or β_2-adrenergic agonists
      (e) Caution with increased risk of insulin errors when used in emergency situations (e.g., nurses preparing insulin infusions). Errors involving calculations (100 units/mL) and use of 4- or 10-mL syringes instead of an insulin syringe are common.
   ii. Sodium bicarbonate
      (a) Dose is 50 mEq of sodium bicarbonate infused slowly over 5 minutes; may repeat in 30 minutes if needed
      (b) May lower plasma K^+ within 30–60 minutes and persist for several hours
      (c) The efficacy of bicarbonate is disputed, and it seems least effective in patients with advanced kidney disease; may be effective in patients with underlying metabolic acidosis
   iii. β_2-adrenergic agonists (e.g., albuterol)
      (a) Dose is albuterol 10–20 mg nebulized over 10 minutes or 0.5 mg intravenously (not available in the United States).
      (b) Will lower plasma K^+ by 0.5–1.5 mEq/L
      (c) Onset is within 90 minutes with inhalation.
      (d) Avoid use in patients with coronary ischemia because of risk of tachycardia.
      (e) Up to 40% of patients will not respond to inhaled albuterol; therefore, it is not recommended as a single agent for urgent treatment of hyperkalemia; consider use in combination with insulin.

e. The treatment options above should be followed by one of the following agents to remove excess K^+ from the body.
   i. Diuretics
      (a) Loop or thiazide-type diuretics increase K^+ renal excretion.
      (b) Ineffective in patients with advanced kidney disease
   ii. Cation-exchange resin
      (a) Exchanges Na^{++} for K^+, resulting in GI excretion of K^+
      (b) Because of a slow onset (2 hours) and unpredictable efficacy, Na^{++} polystyrene sulfonate (Kayexalate) is not indicated for emergency treatment of hyperkalemia.
      (c) Oral dose of sodium polystyrene sulfonate is 15 g repeated every 6 hours as needed. This can be mixed in 20–100 mL of water or syrup, but it is no longer recommended to mix in 70% sorbitol because of the risk of intestinal necrosis (there are reports as well with the premixed 33% sorbitol suspension, but 70% sorbitol appears to have a stronger correlation with intestinal necrosis). Oral sorbitol can prevent constipation associated with the resin; however, the highest risk of intestinal necrosis occurs when administered to patients within 1 week of surgery (occurs in about 1.8% of patients).

(d) Although the oral route is more effective, can also give 30–50 g as a retention enema mixed in 100–200 mL of an aqueous vehicle (e.g., water, 10% dextrose) that has been warmed to body temperature and kept in colon for 30–60 minutes or up to 3 hours. Irrigate colon after enema.
   (1) Sorbitol is not recommended as a vehicle for rectal use because of the risk of intestinal necrosis and other serious GI adverse events. Patients with kidney disease are at increased risk of these GI adverse effects.
   (2) Caution in patients with kidney failure or heart failure caused by Na retention

iii. Dialysis
   (a) Used when other measures are ineffective or when severe hyperkalemia is present
   (b) Plasma K^+ falls by more than 1 mmol/L in the first hour of dialysis and by about 2 mmol/L after 3 hours of dialysis.
   (c) Hemodialysis removes K^+ faster than peritoneal dialysis.
   (d) Monitor for rebound increase in K^+ after dialysis.
   (e) Used in patients with advanced kidney disease

Patient Case

7. A 61-year-old man is brought to the emergency department with shortness of breath and bilateral lower leg edema. Pertinent vital signs and laboratory values include HR 30 beats/minute, BP 102/57 mm Hg, K^+ 7.9 mEq/L, Na^+ 139 mEq/L, glucose 228 mg/dL, Ca^{++} 8.8 mg/dL, digoxin 2.0 ng/mL, BUN 49, and Cr 2.4 mg/dL. His ECG shows wide QRS and peaked T waves. His medical history includes heart failure, atrial fibrillation, coronary artery disease, peripheral arterial disease, and diabetes. The patient has peripheral intravenous access and an external pacemaker. Which one of the following is most appropriate?

A. Calcium gluconate 10 mL intravenously over 2 minutes. - No dig level ted
B. Insulin 10 units intravenously.
C. Sodium bicarbonate 50 mEq intravenously over 10 minutes.
D. Albuterol 10 mg nebulized over 10 minutes. - CAD

VIII. DISORDERS OF MAGNESIUM HOMEOSTASIS

A. Normal Serum Magnesium Concentration Is 1.7–2.3 mg/dL (1.4–1.8 mEq/L or 0.85–1.15 mmol/L).

B. Hypomagnesemia (serum magnesium concentration less than 1.7 mg/dL)
   1. Usually associated with impaired intestinal absorption (e.g., ulcerative colitis, diarrhea, pancreatitis, chronic laxative abuse), inadequate intake, hypokalemia, or increased renal excretion (e.g., diuretic use)
      a. Common in hospitalized patients
      b. Usually associated with alcoholism and delirium tremens
   2. Often occurs concurrently with hypokalemia and hypocalcemia
   3. Signs and symptoms
      a. Neuromuscular symptoms include tetany, twitching, and seizures.
      b. Cardiovascular symptoms include arrhythmias, sudden cardiac death, and hypertension.
   4. Treatment
      a. Oral supplements (e.g., magnesium oxide, magnesium-containing antacids or laxatives) can be used for asymptomatic patients; however, treatment is limited by the high frequency of diarrhea.

b. Symptomatic patients should initially be treated with 1–4 g (8–32 mEq) of magnesium sulfate by slow intravenous infusion (about 1 g/hour to avoid hypotension and/or increased renal excretion because of rapid administration), followed by about 0.5 mEq/kg/day added to intravenous fluid and administered as a continuous infusion. For emergency treatment (e.g., torsades), magnesium can be administered by intravenous push.
c. Reduce dose by half in patients with kidney insufficiency.
d. Around half of administered magnesium is excreted in the urine; therefore, magnesium replacement should occur over 3–5 days.

C. Hypermagnesemia (serum magnesium greater than 2.3 mg/dL)
1. Rarely occurs and is generally associated with chronic kidney disease
2. Signs and symptoms include nausea, vomiting, bradycardia, hypotension, heart block, asystole, respiratory failure, and death; signs and symptoms rarely occur unless magnesium concentration is greater than 4–5 mg/dL.
3. Treatment
a. Discontinue all magnesium-containing medications.
b. Asymptomatic patients with normal kidney function can be treated with 0.9% NaCl and loop diuretics.
c. Symptomatic patients should be treated with 100–200 mg of elemental Ca administered intravenously over 5–10 minutes for cardiac stability.
d. Hemodialysis may be needed for patients with kidney disease.

IX. DISORDERS OF PHOSPHORUS HOMEOSTASIS

A. Normal Serum Phosphorus Concentration Is 2.5–4.5 mg/dL.

B. Hypophosphatemia (serum phosphorus concentration less than 2.5 mg/dL)
1. Causes of hypophosphatemia
a. Increased renal elimination (e.g., diuretics, glucocorticoids, sodium bicarbonate)
b. Rapidly refeeding patients with chronic malnutrition (see refeeding syndrome in Nutrition section)
c. Respiratory alkalosis
d. Treatment of diabetic ketoacidosis; phosphorus shifts into the IC compartment as diabetic ketoacidosis is corrected
2. Signs and symptoms
a. Tissue hypoxia can occur because of a decrease in oxygen release to peripheral tissues.
b. Neurologic manifestations include confusion, delirium, seizures, and coma.
c. Pulmonary and cardiac symptoms can include respiratory failure, difficulty weaning from mechanical ventilation, heart failure, and arrhythmias.
d. Other organ systems affected include muscle, hematologic, bone, and kidney.
3. Prevention and treatment
a. Prevent hypophosphatemia by supplementing intravenous fluid with 10–30 mmol/L intravenous phosphorus in patients at risk of hypophosphatemia (e.g., malnourished, alcoholism, diabetic ketoacidosis).
b. Oral phosphorus products (e.g., K-Phos Neutral; also contain K^+ and Na) can be used for asymptomatic patients, but they are poorly absorbed.
c. Symptomatic patients should receive 15–30 mmol (or 0.5–0.75 mmol/kg of IBW) of phosphorus (Na^+ phosphate or K^+ phosphate) administered intravenously over 3–6 hours (maximal rate is 7.5 mmol/hour). Note: Na^+ content (4 mEq per 3 mmol phosphate) and K^+ content (4.4 mEq per 3 mmol phosphate)

C. Hyperphosphatemia
   1. Typically occurs in patients with chronic kidney disease or hypoparathyroidism
   2. In general, patients are asymptomatic, but they can have signs and symptoms including hypocalcemia, ECG changes, paresthesias, and vascular calcifications.
   3. Treatment (see treatment of hyperphosphatemia in the Nephrology chapter)

X. DISORDERS OF CALCIUM HOMEOSTASIS

A. Normal Serum Ca^{++} Concentration Is 8.5–10.5 mg/dL (total Ca^{++} includes bound and unbound Ca^{++}), and Normal Ionized Ca^{++} Is 1.1–1.3 mmol/L.

B. Distribution of Ca
   1. EC fluid contains less than 1% of the total body stores of Ca^{++}; 99% of total body stores of Ca^{++} is in skeletal bone.
      a. About half of Ca^{++} in the EC compartment is bound to plasma proteins (primarily albumin).
      b. The active form of Ca^{++} is the unbound or ionized Ca^{++}.
   2. Ionized Ca^{++} is regulated by parathyroid hormone, phosphorus, vitamin D, and calcitonin.

C. Hypocalcemia
   1. Occurs in patients with chronic kidney disease, hypoparathyroidism, vitamin D deficiency, alcoholism, and hyperphosphatemia, as well as in patients receiving large amounts of blood products or patients undergoing continuous renal replacement therapy (CRRT [i.e., Ca^{++} chelates with citrate in blood products or CRRT])
   2. Factors that cause an increase in EC Ca^{++} binding to albumin (e.g., metabolic alkalosis) can cause a reduction in plasma-ionized Ca^{++} concentration, leading to symptomatic hypocalcemia.
   3. A low serum albumin will cause a falsely low total serum Ca, so an adjustment is necessary. For every 1-g/dL decrease in serum albumin less than 4 g/dL, add 0.8 mg/dL to total serum Ca concentration to correct the value.
   4. Signs and symptoms include tetany, muscle spasms, hypoactive reflexes, anxiety, hallucinations, lethargy, hypotension, and seizures.
   5. Treatment
      a. Asymptomatic hypocalcemia associated with hypoalbuminemia is typically associated with normal ionized Ca^{++} concentrations and therefore does not require treatment.
      b. Asymptomatic hypocalcemia can be treated with oral Ca^{++} supplements at a dose of 2–4 g/day of elemental Ca^{++}; patients may also require vitamin D supplementation.
      c. Symptomatic hypocalcemia is treated with 200–300 mg of elemental Ca^{++} administered intravenously over 5–10 minutes, followed by a continuous infusion.
         i. Equivalent to 1 g of $CaCl_2$ (273 mg of elemental Ca^{++}) administered through a central intravenous catheter; peripheral administration of $CaCl_2$ can result in severe limb ischemia
         ii. Equivalent to 2–3 g of Ca^{++} gluconate (180–270 mg of elemental Ca^{++}); preferred for peripheral intravenous administration
         iii. Do not infuse Ca^{++} at a rate faster than 60 mg of elemental Ca^{++} per minute; rapid administration, which is not recommended, is associated with hypotension, bradycardia, or asystole.
         iv. The duration of a bolus dose of Ca^{++} is ideally 1–2 hours, followed by a continuous infusion rate of 0.5–2 mg/kg/hour of elemental Ca^{++}.

D. Hypercalcemia (serum Ca^{++} concentration greater than 10.5 mg/dL) is usually related to malignancy or hyperparathyroidism; see Oncology Supportive Care chapter.

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ANSWERS AND EXPLANATIONS TO PATIENT CASES

1. Answer C

Although this patient's BP is not necessarily low, it is likely low compared with his baseline, considering his history of hypertension. In addition to his relatively low BP, his other signs and symptoms of intravascular volume depletion include an increased BUN/Cr ratio and a reduced urine output. Crystalloids or colloids are appropriate fluids for resuscitation, making hetastarch (Answer C) the best option. Furosemide (Answer A) may increase his urine output, but at the cost of further depleting the intravascular volume. Albumin 25% (Answer B) should be avoided for fluid resuscitation because it causes a shift of fluid from the IS space into the intravascular space, which can potentiate his dehydration. Answer D would be appropriate for a maintenance infusion; however, D_5W/0.45% NaCl plus KCl 20 mEq/L would not provide optimal replacement of the intravascular space given the distribution in TBF.

2. Answer C

This patient has no current signs or symptoms of intravascular volume depletion, so he does not require fluid resuscitation. Because he is not taking adequate fluids by mouth, he should be administered maintenance intravenous fluid to prevent dehydration and electrolyte imbalances. This is typically accomplished by a combination of free water (as D_5W) and 0.45% NaCl with K^+. The infusion rate is calculated as 1500 mL + (60 kg × 20 mL/kg) = 2700 mL/24 hours or about 110 mL/hour. Parenteral nutrition (Answer A) is inappropriate because there is no evidence that the patient's GI tract is nonfunctional. Albumin 5% (Answer B) or LR solution (Answer D) should be reserved for fluid resuscitation in patients with signs or symptoms of intravascular volume depletion.

3. Answer B

Although this patient is experiencing symptomatic hyponatremia, she is also showing signs of intravascular volume depletion. This intravascular volume depletion is a potent stimulus for ADH secretion, which will potentiate hyponatremia. In patients with hyponatremia and intravascular volume depletion, it is important to restore intravascular volume first to prevent organ hypoperfusion as well as to inhibit the secretion of ADH. Fluid resuscitation should be accomplished with 0.9% NaCl as a fluid bolus, followed by a reevaluation of fluid status. A slower infusion of 0.9% NaCl (Answer A) will not quickly restore intravascular volume. Once the intravascular volume is restored, secretion of ADH will cease. This can be followed by a water diuresis with a subsequent rise in the serum Na^+ concentration. Of importance, the patient should be monitored closely to prevent a rise in serum Na^+ greater than 10–12 mEq/L/day. If serum Na^+ rises too fast, 0.45% NaCl can be infused to slow the rate of rise of serum Na^+ concentration. Hypertonic saline (Answer C and Answer D) would not be advisable unless the patient continued to have symptoms of hyponatremia after appropriate fluid resuscitation.

4. Answer C

To prevent central pontine myelinolysis in patients with hyponatremia, it is recommended that the serum Na^+ concentration be raised by no more than 10–12 mEq/L in 24 hours. Of emphasis, the goal is not to achieve a normal serum Na^+ concentration in 24 hours. Rapid correction of chronic hyponatremia can cause permanent neurologic damage.

5. Answer D

This patient has hyponatremia and hypokalemia. In patients with hypokalemia, there is a reduction in IC K^+. To maintain cellular electroneutrality, Na^+ will shift into cells. As K^+ is replaced, Na^+ will shift out of cells, and the serum Na^+ concentration will rise. Therefore, in this case, the hypokalemia should be corrected first, which will cause a subsequent improvement in the hyponatremia. Because this patient has no ECG changes related to the hypokalemia, oral supplementation with K^+ is recommended over intravenous replacement (Answer A). A dose of 60–80 mEq/day should cause an increase in the K^+ concentration of about 1.5 mEq/L. Because the patient is eating a regular diet, she should no longer require intravenous fluids (Answer B). Hypertonic saline (Answer C) is incorrect because this patient is not experiencing serious symptoms of hyponatremia.

6. Answer D

This patient has not been given enough water, and she is unable to communicate (or feel) thirst. This medical error can be prevented by administering about 1 mL of water for every calorie administered. It should

also be prevented by monitoring serum Na concentrations and adjusting water intake as needed. To correct the hypernatremia, water should be administered preferably through the enteral feeding tube. If this is not possible, then it can be readministered intravenously as D_5W (Answer B or C) but never as sterile water (Answer A). Sterile water administered intravenously can cause hemolysis and death. The patient's water deficit (in liters) can be estimated by the equation 0.4 × LBW × [(Na^+/140) − 1]. Water should be replaced over several days, taking care to avoid changes in serum Na^+ greater than 10–12 mEq/L in 24 hours.

7. Answer B

This patient has ECG changes consistent with hyperkalemia. Insulin (Answer B) will have the fastest onset and most predictable action of lowering serum potassium. Calcium gluconate (Answer A) should be avoided in this patient because it can potentiate digoxin toxicity and bradycardia. The efficacy of sodium bicarbonate (Answer C) is not well established. Albuterol (Answer D) can be efficacious when added to insulin, but it may not be effective in about 40% of patients and is therefore not recommended as initial therapy or as monotherapy for hyperkalemia.

8. Answer C

This patient is receiving a calorie-dense EN formula that typically has less water than other enteral products. Therefore, although she is not currently hypernatremic, the patient is at risk of developing hypernatremia because of insufficient water intake. This can be prevented by administering additional water. The preferred route is enteral if possible. The additional water needed on a daily basis can be estimated as 1 mL/kcal. Therefore, if this patient is receiving the enteral feeding at 35 mL/hour × 2 kcal/mL, she is receiving 1680 kcal/day. She is only receiving 710 mL/L of water of enteral formula, which amounts to 596 mL/day for the 840 mL of enteral feeding daily (35 mL/hour × 24 hours = 840 mL/day). Because she is receiving 1680 kcal, she needs about 1680 mL/day of water. Subtracting the water from the feedings from the total needed, 1680 − 596 = 1084 mL is needed. This can be divided and administered through the gastric feeding tube as around 180 mL/dose every 4 hours. Of note, the patient should be monitored for fluid overload, especially given her chronic kidney disease. Given this patient's stable kidney disease and her adequate urine output, she should be able to tolerate this amount of free water. The amount of free water needed on a daily basis is an estimation and should be adjusted on the basis of specific patient parameters (e.g., serum Na^+, input, output, daily weight, edema). Free water should not be administered as intravenous dextrose (Answer B) unless enteral administration is not feasible. Answer A is incorrect because reducing Na will not prevent hypernatremia; the problem is related to too little water rather than too much Na^+. Answer D is incorrect because the caloric goals should not be sacrificed, and they would not eliminate the problem of insufficient water administered.

9. Answer D

This patient is developing a respiratory acidosis possibly because of overfeeding. Although dextrose is metabolized to water and CO_2, it generally will not cause a respiratory acidosis unless the patient is being overfed. Therefore, by reducing the total calories to 25 kcal/kg, the risk of overfeeding is diminished, and reintubation can be avoided. Answer A is incorrect because patients can be overfed with either PN or EN. Answer B is incorrect because even with reducing the dextrose in the PN, the patient can still develop symptoms of overfeeding. Answer C is incorrect because the underlying (overfeeding) cause should be corrected, rather than adjusting the acetate to treat a respiratory acidosis.

10. Answer D

To determine the amount of calories provided by propofol, it must first be determined how many milliliters a day are infused. For this patient receiving 45 mcg/kg/minute of propofol and weighing 70 kg, 454 mL is infused daily (assuming a constant infusion rate). Next, assuming that a 10% lipid emulsion provides about 1.1 kcal/mL, it can be calculated that 454 mL/day of propofol provides around 500 kcal/day.

11. Answer C

This patient has developed a metabolic alkalosis likely secondary to the loss of gastric fluid through NG suctioning. The low serum Cl^- and elevated serum bicarbonate concentrations support this theory. In addition, the acid base is consistent with a metabolic alkalosis with a compensatory respiratory acidosis. The treatment in this circumstance is to replace the lost fluid with 0.9% NaCl, which is being done. In addition, Na

and K^+ can be administered as the Cl^- salt, rather than the acetate salt. For this case, only Na^+ is converted to Cl^- salt, and K^+ is left as the acetate salt initially. With daily monitoring, the ratio of Cl^- to acetate can be adjusted further if needed. Answer B is incorrect because it would likely worsen the metabolic alkalosis as Na^+ acetate is converted to bicarbonate. Answer A is incorrect because hypercapnia is a compensatory response, not the primary acid-base disturbance. Answer D is incorrect for several reasons. First, it is never advisable to add Na^+ bicarbonate to PN because of incompatibility and increasing the risk of Ca^{++}-phosphate precipitation. Second, Na^+ bicarbonate is the incorrect treatment for a metabolic alkalosis because it can worsen alkalosis.

12. Answer B

The total calories are calculated by adding the calories provided by dextrose, lipid, and AA. Dextrose provides 714 kcal (210 g × 3.4 kcal/g); lipid provides about 300 kcal (30 g × 10 kcal/g); and AA provides 300 kcal (75 g × 4 kcal/g). Adding these together provides calories of 1314 kcal/50 kg = 26.3 kcal/kg.

13. Answer D

This patient's hyperglycemia could be attributable to either stress or corticosteroids. Because the corticosteroid dose is being tapered downward, the blood glucose concentrations will also likely decrease with time. For patients with a fluctuating blood glucose concentration, it can be difficult to add insulin to PN because it cannot be adjusted in a timely manner. Regardless, Answer A is incorrect because long-acting insulin should not be added to PN. If insulin is added to PN, it should be regular insulin. Although some experts promote "permissive underfeeding," Answer B is incorrect because it would provide insufficient calories for this patient. Sliding scales of insulin can be useful when used in conjunction with a baseline of insulin in patients with a fluctuating blood glucose concentration. However, a sliding scale (Answer C) should not be used as the primary intervention for blood glucose control because it is reactive and fails to prevent hyperglycemia. In addition, the sliding scale described recommends insulin only when the blood glucose reaches 200, which is too high. Answer D is correct because it provides a baseline of insulin that can be adjusted in a timely manner as the blood glucose concentrations fluctuate on the basis of the patient's status.

ANSWERS AND EXPLANATIONS TO SELF-ASSESSMENT QUESTIONS

1. Answer: D

This patient continues to have hypotension and tachycardia, both signs of intravascular volume depletion. The improvement in BP and tachycardia after a fluid bolus also indicates intravascular volume depletion. Fluid administration should continue until there is no further improvement in vital signs. Patients with intravascular volume depletion require a rapid bolus of crystalloid (either 0.9% NaCl or LR) in the amount of 500–1000 mL (or about 20 mL/kg), followed by a reassessment. A rapid bolus is essential to prevent organ dysfunction caused by hypoperfusion. Although the patient has poor urine output, administration of furosemide (Answer A) will worsen volume depletion. As volume is replaced, urine output will likely increase. Administration of 5% albumin in combination with a vasopressor (Answer B) should not be the initial treatment as long as vital signs are improving with the administration of fluid boluses with 0.9% NaCl. In addition, colloids are more expensive, and there is no evidence showing improved outcomes for fluid resuscitation compared with crystalloids. Furthermore, infusion of albumin over 4 hours is incorrect because it would not restore intravascular volume rapidly enough to prevent organ dysfunction. Intravenous fluid containing D_5W (Answer C) is not appropriate for fluid resuscitation regardless of the blood glucose concentration.

2. Answer A

To answer this question, an alligation must first be set up using 0.9% and 23.4% NaCl. If 0.9% NaCl contains 154 mEq/L, then 3% should contain around 513 mEq/L. After completing the alligation, the correct amounts can be double-checked by verifying the amount of NaCl in the prepared product: 907 mL of 0.9% NaCl contains 140 mEq of NaCl; 93 mL of 23.4% NaCl contains 372 mEq of NaCl; therefore, 140 mEq + 372 mEq = 512 mEq/L of NaCl in the final product. The osmolarity is calculated as (3 g/100 mL) × (1 mol/58.5 g) × (2 Osm/mol) × (1000 mOsm/Osm) × (1000 mL/L) × 0.93 = 954 mOsm/L. Because of the osmotic coefficient (0.93), the NaCl does not completely dissociate in solution. Although use of the osmotic coefficient provides a more accurate osmolarity, it is likely not clinically relevant in calculating the osmolarity of intravenous NaCl. Therefore, it is safe to estimate the osmolarity of NaCl as either 954 or 1026 mOsm/L because there is no apparent clinical difference between these osmolarities. Because the osmolarity is greater than 900 mOsm/L, the infusion should be administered through a central line, if possible, to prevent pain and irritation.

3. Answer D

In this case, hyponatremia is likely because of congestive heart failure and has likely developed over a prolonged period (not acute onset). Patients with chronic hyponatremia because of heart failure are typically asymptomatic. Rapid correction of chronic hyponatremia is associated with permanent neurologic damage caused by central pontine myelinolysis. Furthermore, HS can worsen volume overload in patients with heart failure. Although hyponatremia is a sign of worsening heart failure, correction of hyponatremia in patients with heart failure does not improve outcomes. For these reasons, the risks of correcting the serum sodium with HS (Answer A, Answer B, and Answer C) outweigh the potential benefits.

4. Answer B

Patients with hyperkalemia and ECG changes should be treated first with Ca^{+} for cardiac stability. After Ca^{++} administration, other measures can be taken to shift K^{+} from the EC compartment to the IC compartment. Insulin (Answer A) can accomplish this; however, in this hyperglycemic patient, insulin should be administered without glucose. Sodium polystyrene sulfonate (Kayexalate; Answer C) can be administered, but it is not effective immediately and is therefore not appropriate for first-line treatment of symptomatic hyperkalemia. Sodium bicarbonate (Answer D) is incorrect because it does not treat cardiac instability.

5. Answer C

This patient is not taking adequate nutritional intake because of her mental status. Because her GI tract is functional, it should be used for feeding to prevent gut mucosal atrophy. An NG or nasoduodenal feeding tube is appropriate for enteral access for short-term nutritional support. A percutaneous gastrostomy tube (Answer D) requires a surgical procedure and is used for long-term nutritional support. The patient should receive between 25 and 35 kcal/kg/day. The PN formulas (Answer A and

Answer B) should not be used in a patient with a functional GI tract. Although Answer B would be an appropriate PN formula for peripheral administration, PN is associated with more complications than EN.

6. Answer C

This correct formula provides about 30 kcal/kg of calories, 1.5 g of protein per kilogram (AA), and 30% of total calories as lipid. Answer A is incorrect because it provides 1000 calories as lipid, which is about 62% of the total calories provided. Answer B is incorrect because it contains only 0.8 g/kg of AA, an insufficient amount considering the patient's stress and the apparent absence of kidney injury. Answer D is incorrect because it contains too much AA.

7. Answer B

This patient has hypomagnesemia and hypokalemia. Correction of hypokalemia requires correction of hypomagnesemia to prevent renal loss of K^+. Magnesium should be administered slowly to avoid hypotension and increased renal excretion caused by rapid administration.

8. Answer C

Enteral nutrition prevents gut mucosal atrophy and subsequent bacterial translocation. Bacterial translocation is the crossing of bacteria from the GI tract into the systemic circulation. Enteral nutrition is associated with fewer infectious complications than is PN, which may be partly because of a reduction in bacterial translocation.