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Fluid and Electrolytes in Adult Parenteral Nutrition

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Body fluid and serum electrolyte concentrations often become imbalanced in patients who require parenteral nutrition (PN) due to one or more factors, such as physiologic stress, wound drainage, blood loss, gastrointestinal fluid loss, organ malfunction, hormonal abnormalities, IV fluid use, various medications, and even unavoidable shortages of parenteral electrolyte products.

It's important to discuss each patient's clinical status with the physicians, pharmacists, and nurses involved in the patient's care to become fully informed about his or her clinical situation. With knowledge of fluid and electrolyte requirements, the conditions in which these needs are altered, and the physical signs of excesses or deficits, RDs can determine safe and reasonable adjustments to PN electrolyte content.

This continuing education course focuses on the role of fluid and electrolytes in PN and the clinical situations in which water and electrolytes may need to be adjusted in PN. It's intended for practitioners who have a good basic knowledge of and experience with PN.

Part 1: Requirements for Water and Electrolytes and Units of Measurement

The Institute of Medicine lists the Dietary Reference Intakes for oral nutrients in milligrams or grams.[1] Parenteral and oral requirements are, of course, different because intravenous administration bypasses normal digestion and absorption. For IV fluids and PN, the milliequivalent (mEq) is the unit of measurement used for sodium (Na), chloride (Cl), potassium (K), magnesium (Mg), calcium (Ca), and acetate, while the millimole (mM) or the milliequivalent can be used for phosphorus (P).[2,3]

Electrolytes are compounds or substances that dissociate in the solution to release positively and negatively charged ions that can carry electric current—thus, the term electrolyte. A milliequivalent measures chemical-combining capacity, or the number of electrolyte atoms that will combine with 1 mEq of another electrolyte. Milliequivalents can be calculated from milligrams using this formula:

mEq = (mg x valence) ÷ atomic weight

Example: How many milliequivalents in 1,500 mg Na?

mEq Na = (1500 x 1) ÷ 23

Answer: 65 mEq Na

A millimole is the amount of a substance equal to its atomic weight expressed in milligrams. Phosphorus is measured in millimoles because at a physiologic pH of 7.4, the phosphate ion exists partly in divalent and partly in monovalent forms. The ratio is 4 mM of hydrogen phosphate (divalent, HPO_4^{2-}) to 1 mM of dihydrogen phosphate (monovalent, $H_2PO_4^-$). Thus, for every 5 mM of phosphorus in the bloodstream, there are four divalent ions (4 x 2 = 8) and one monovalent ion, making nine total valence electrons, and since 9 ÷ 5 = 1.8, the valence is1.8.[3,4] Millimoles can be calculated using this formula:

mM = mg ÷ atomic weight

Example: How many millimoles in 700 mg P?

mM = 700 ÷ 31

Answer: 22.5 mM P

Table 1 shows adults' oral and parenteral water and electrolyte requirements, and Table 2 shows the relationship among milligrams, grams, milliequivalents, and millimoles. Refer to Table 2 to do calculations in the two previous equations.

Table 1: Oral and Parenteral Requirements for Water and Electrolytes

Component	**Daily Oral Requirements (Dietary Reference Intakes for adults[1*])**	**Daily Estimated Requirements for Parenteral Nutrition[2]**	**Salt Form Used in Parenteral Nutrition Solutions[5]**
Water	2.7 to 3.7 L	30 to 40 mL/kg	
Sodium	1.5 g (ages 19 to 50) [a] 1.3 g (ages 51 to 70) 1.2 g (ages 70 and older)	1 to 2 mEq/kg	Sodium chloride Sodium acetate Sodium phosphate
Chloride	2.3 g (ages 19 to 50)[a] 2 g (ages 51 to 70) 1.8 g (ages 70 and older)	As needed for acid-base balance	Sodium chloride Potassium chloride
Potassium	4.7 g [a] 5.1 g (lactation)	1 to 2 mEq/kg	Potassium chloride Potassium acetate Potassium phosphate
Magnesium	310 (females) and 400 (males) mg (ages 19 to 30)[b] 320 (females) and 420 (males) mg (ages 31 and older) 350 to 360 mg (pregnancy) 310 to 320 mg (lactation)	8 to 20 mEq	Magnesium sulfate
Calcium	1,000 mg (ages 19 to 50)[b] 1,200 mg (ages 51 and older)	10 to 15 mEq	Calcium gluconate

Phosphorus	700 mg[b]	20 to 40 mM	Sodium phosphate Potassium phosphate

[a] *AI = Adequate Intake;* [b] *RDA = Recommended Dietary Allowance*
Dietary Reference Intakes from: Dietary reference intakes and application. Institute of Medicine website. http://www.iom.edu/Activities/Nutrition/SummaryDRIs/DRI-Tables.aspx. Last updated September 12, 2011. Accessed July 8, 2012.

Table 2: Relationship of Milligrams, Grams, Milliequivalents, and Millimoles[3]

Electrolyte	Atomic Weight	Valence	Weight of 1 mEq	Weight of 1 mM	1 mM equals	mEq/g	mM/g
Sodium	23	1	23 mg	23 mg	1 mEq	43.5	43.5
Chloride	35.5	1	35.5 mg	35.5 mg	1 mEq	28.2	28.2
Potassium	39	1	39 mg	39 mg	1 mEq	25.6	25.6
Magnesium	24	2	12 mg	24 mg	2 mEq	83.3	41.6
Calcium	40	2	20 mg	40 mg	2 mEq	50	25
Phosphorus	31	1.8	17.2 mg	31 mg	1.8 mEq	58.1	32.2

The electrolyte content of PN is labeled and ordered on a per liter or per day basis, depending on a pharmacy or health care system's typical practices.The first bag of PN can contain standard parenteral electrolyte amounts as outlined in Table 1 or typically contains a hospital's established "standard" amount.

Dosing all PN components on a per day basis can help reduce errors, especially when the solution's total volume is changed. For example, the standard daily electrolyte content for PN at the University of Virginia Health System is 63 mEq Na, 72 mEq K, 18 mEq Mg, 18 mM P, 8.1 mEq Ca, and 53 mEq acetate.[6] These amounts change depending on a patient's needs and clinical conditions or sometimes because of nationwide shortages in various parenteral electrolyte products.[7]

Test Your Skills With These Practice Questions
Note: The graded examination follows the article.

1. How many milligrams are in 70 mEq K?
A. 2,730
B. 4,500
C. 5,000
ANSWER: A

2. A patient's PN contains 20 mM P. How is that expressed in milliequivalents and milligrams?
A. 11 mEq; 1,000 mg
B. 36 mEq; 620 mg
C. 42 mEq; 600 mg
D. 620 mEq; 20 mg
ANSWER: B

Explanation: Use the equations in the text (image 1 and image 2) and the information found in Table 2 to answer these questions.

Part 2: Water and Sodium

Water is necessary for biochemical reactions within cells, regulating body temperature, maintaining blood volume, transporting nutrients, and removing waste products.[1]
Total body water varies depending on a patient's percentage of fat and lean tissue, but in general it's estimated at an average of 60% of body weight for men and 50% of body weight for women.[8]

RDs need to adjust the water in PN depending on patients' intake and output. Water intake for the PN-dependent patient comes from IV fluids and, in some cases, oral intake. In addition, 300 mL water per day is generated from the oxidation of carbohydrate, protein, and fat . Water is lost from the body via urine, gastrointestinal fluids, wound drainage, chest tubes, and blood loss as well as insensible fluid loss from skin and lungs. Insensible losses for the average adult are approximately 800 to 1,100 mL/day and can be increased in certain conditions such as fever and burns.[3]

Two main compartments contain total body water: intracellular fluid (ICF), or water found inside cells, and extracellular fluid (ECF), or water found outside cells. Approximately two-thirds of total body water is ICF and one-third is ECF.

ECF is further categorized into three main types: interstitial, intravascular, and transcellular. Interstitial fluids are located between cells and in tissues, including lymph. Intravascular fluid is the plasma or noncellular portion of the blood and is approximately 25% of the ECF. Transcellular fluids are located in different parts of the body, such as the gastrointestinal tract and ocular, pleural, and cerebrospinal areas, and comprise a very small percentage of total body water. [3,8]

Table 3 shows the normal laboratory serum values for adults.

Table 3: Normal Laboratory Values for Adults

Electrolyte	**Serum Concentration**
Sodium (Na)	135 to 145 mEq/L
Chloride (Cl)	98 to 107 mEq/L
Potassium (K)	3.5 to 5 mEq/L
Carbon dioxide (CO_2) (total bicarbonate)	22 to 28 mEq/L
Calcium (Ca)	8.5 to 10.5 mg/dL
Magnesium (Mg)	1.8 to 3 mg/dL
Phosphorus (P)	2.5 to 4.5 mg/dL
Blood Urea Nitrogen (BUN)	8 to 20 mg/dL
Creatinine	0.6 to 1.2 mg/dL
Glucose	60 to 110 mg/dL (fasting)

Notes: Reference ranges may differ from one clinical laboratory to another. Normal serum concentrations differ for age, gender, and certain conditions, such as pregnancy.[3]
Bicarbonate is 90% to 95% of the serum carbon dioxide measurement.
Glucose levels differ for fasting and nonfasting states.

Information on normal laboratory values adapted from: Common laboratory tests: selection and interpretation. In: Nicoll D, Lu CM, McPhee SJ ,Pignone M. ***Pocket Guide to Diagnostic Tests****. 6th ed. 2012*

The balance of water and solutes in a patient's bloodstream can be determined by reviewing laboratory values and estimating osmolarity and tonicity. Osmolarity is a measure of the number of particles per liter of solution (mOsm/L) while osmolality is a measure of the number of particles per kilogram of water (mOsm/Lg). These two terms are similar in meaning and are sometimes used interchangeably by different practitioners.

To equalize osmolarity, water moves between the ECF and the ICF, from areas of lower osmolarity to areas of higher osmolarity. Normal serum osmolarity is 280 to 300 mOsm/L. Sodium is the main determinant of serum osmolarity, and glucose and blood urea nitrogen (BUN) contribute to a smaller extent.[3,8] The following is a formula for estimating serum osmolarity:

$$mOsm/L = [2 \times Na\ (mEq/L)] + [glucose\ (mg/dL) \div 18] + [BUN\ (mg/dL) \div 2.8]$$

Tonicity is a measure of active osmolarity, which accounts for more than 90% of the ECF osmolarity and actual shifts of water across cell membranes. BUN is not included because urea moves easily across membranes and does not affect water flow.[3,8]

Normal tonicity is 275 to 290 mOsm/L. In clinical practice, the terms "hypertonic" and "hyperosmolar" refer to a serum osmolarity greater than 300 mOsm/L, while "hypotonic" or "hypo-osmolar" refer to a serum osmolarity below 280 mOsm/L.[3] The term "isotonic" refers to normal osmolarity. Tonicity can be estimated using the following equation:

$$mOsm/L = [2 \times Na\ (mEq/L)] + [glucose\ (mg/dL) \div 18]$$

In general, serum is hypertonic in situations of water deficit and hypotonic if there is excess water in the bloodstream.[3]

Sodium is the main extracellular electrolyte. Approximately 95% of total body sodium can be found in the ECF.[1] Sodium functions to regulate body fluid volume and electrical cell membrane potential, and is involved in the active transport of various substances in and out of cells.[1] Water and sodium are physiologically related so that an imbalance of one affects the other.

Effective circulating fluid volume (intravascular volume) is the portion of the ECF in the vascular system. Intravascular volume depletion (hypovolemia) occurs when ECF has decreased because of a decline or deficit in total body sodium content. Blood loss is the most obvious cause of intravascular volume depletion, but renal fluid losses, such as with the use of diuretics, and in hypoaldosteronism (adrenal insufficiency); osmotic diuresis in uncontrolled diabetes; and salt-wasting nephropathies are other possible causes.

Gastrointestinal fluid loss, such as through vomiting, gastric drainage, diarrhea, or excessive fistula or ostomy output, can also result in depletion of ECF volume. "Third-spaced fluid" is yet another cause of ECF volume depletion—the body has not lost this fluid but has redistributed it. Pleural effusions, ascites, or fluid accumulations caused by an intestinal obstruction or areas of inflammation are types of third-spaced fluids.[3,8] Weight loss usually occurs in ECF volume deficit except in the case of third-spaced fluids.

ECF volume depletion affects both the central nervous and cardiovascular systems. Effects on the central nervous system include apathy, fatigue, lethargy, and headaches, and severe depletion can cause stupor, delirium, and coma. Effects on the cardiovascular system include tachycardia, orthostatic hypotension, flat or collapsed veins, low pulse pressure, cool extremities, and decreased body temperature.

An ECF deficit causes BUN to rise. Since creatinine (Cr) does not rise in normal renal function, the BUN-to-creatinine ratio typically will increase to greater than 20:1.

ECF volume depletion typically is treated with IV isotonic saline (0.9% NaCl), lactated Ringers' solution, or blood products (in the case of severe blood loss).[3,8,9] For a patient on PN, more sodium and water can be added to the PN formula.

Hypervolemia, or an excess of ECF, occurs in renal failure (decreased excretion), nephrotic syndrome, cirrhosis, congestive heart failure, excessive IV fluid administration, or the retention of body fluid caused by the stress response. Sodium excess will cause water retention and ECF volume expansion. Signs of hypervolemia (and sodium excess) include edema and weight gain and, if the intravascular fluid is expanded, tachycardia, hypertension, and jugular venous distention.[3,8] In heart failure and cirrhosis, the effective circulating volume is reduced, and the kidneys continue to retain sodium and water, expanding the ECF. Other types of sodium excess in interstitial fluid occur with ascites, pulmonary edema, and other third-spaced fluids.

Treatment for hypervolemia usually involves restriction of both sodium and water, and often the use of diuretic medications. Dialysis or hemofiltration may be necessary in situations of renal failure.[3,8] Both volume and sodium content of PN should be minimized when a PN patient is hypervolemic and/or when the physician wants the patient's fluid intake to be restricted.

Abnormal Serum Sodium Concentrations

Low serum sodium (hyponatremia) and high serum sodium concentrations (hypernatremia) can occur in a wide variety of conditions. Accurately diagnosing and treating a patient with

abnormal serum sodium requires physical and other clinical information as well as data on fluid intake and output. It's important to discuss fluid and sodium issues with the patient's physicians, especially if the patient is in the ICU.

Hyponatremia (serum sodium concentration less than 135 mEq/L): Hyponatremia usually indicates body water excess, sodium deficit, or both of these conditions. Symptoms are caused by cerebral edema and include nausea and weakness in mild cases and headaches, altered mental status, seizures, coma, and respiratory arrest in severe cases.

There are three main types of hyponatremia: hypotonic, isotonic, and hypertonic. Hypotonic hyponatremia can occur in hypervolemic, hypovolemic, and euvolemic conditions, and will be discussed in more detail later in this section.[3,8,10]

Isotonic hyponatremia is not common but can occur in situations of hyperlipidemia or hyperproteinemia. It has been termed "pseudohyponatremia," which means that measured serum sodium is low because of displacement of serum water by lipids or protein; serum osmolarity is normal and no water or sodium changes are necessary.[3]

Hypertonic hyponatremia can occur in situations of hyperglycemia or after IV mannitol administration. In hyperglycemia, an excess of glucose causes serum osmolarity to increase, and this causes an osmotic fluid shift from ICF to ECF. For every 100 mg/dL increase in serum glucose above normal, serum sodium concentration decreases by approximately 1.6 to 2.4 mEq/L.[3,11]

Use the following formula to estimate a corrected serum sodium level for patients with hyperglycemia:

1.6 x [(glucose-100) ÷ 100] = n
Add "n" to the measured Na level

Example: glucose = 300; Na = 130

1.6 x [(300-100) ÷ 100] = 3.2
"Corrected" Na = 133

Hypotonic hyponatremia can occur in hypervolemic, hypovolemic, and euvolemic (isovolemic) patients. In hypervolemic hypotonic hyponatremia, the serum sodium concentration is low, with an excess of both total body sodium and water. This occurs, for example, in situations of congestive heart failure and edema, cirrhosis with ascites, and renal failure.

Conversely, hypotonic hyponatremia also can be present in the hypovolemic patient because of both water and sodium loss. Some causes of renal sodium and water loss are nephropathy, diuretic use, adrenal insufficiency, and cerebral salt wasting. Also, gastrointestinal loss of sodium and water occurs from vomiting, diarrhea, and ostomy or fistula drainage. Other

causes of sodium and water deficit include blood loss, wound drainage, and third-spaced fluids.[3,4,8]

Euvolemic hypotonic hyponatremia occurs when total body sodium is normal, but there is excess body water and the sodium concentration of the ECF is decreased. Causes of euvolemic hypotonic hyponatremia include water intoxication (very rare), adrenal insufficiency, hypothyroidism, hypokalemia, various drugs, and the syndrome of inappropriate antidiuretic hormone (SIADH). In SIADH, ADH (also called vasopressin) is secreted inappropriately, causing the body to retain water and plasma osmolarity is decreased because of dilution. Malignant tumors, central nervous system disorders, pulmonary diseases, or excessive ADH secretion during the postoperative period can cause SIADH. SIADH is identified by hyponatremia, urine osmolarity greater than serum osmolarity, and urine sodium concentration greater than 20 mEq/L with normal renal, thyroid, cardiac, and adrenal function.[3,8,9]

Treating hyponatremia: First determine the cause of hyponatremia to ensure the correct treatment. If there is a deficit of both water and sodium (hypovolemic hypotonic hyponatremia), PN water and electrolyte content can be increased, and additional IV fluids can be used if the patient requires replacement of larger amounts of lost body fluid. Monitoring all sources of intake and output is important. Table 4 shows estimated electrolyte content of some gastrointestinal fluids.

Table 4: Approximate Amount of Electrolytes in Some Gastrointestinal Fluids [3,12,13]

Electrolyte Content (mEq/L)	Gastric Fluid	Pancreatic Fluids	Ileostomy Output	Bile	Colon (diarrhea, colostomy output)
Sodium	60 to 100	140	130 to 140	140 to 145	50 to 120
Chloride	100 to 130	55 to 75	100 to 105	100 to 110	40 to 90
Bicarbonate	-	90 to 115	30	15 to 35	30 to 45
Potassium	10	5	5	5	20 to 30
Hydrogen	60	-	-	-	-

Use Table 4 as a guide for how much electrolytes to add to PN formula (and IV fluid, if used).

When working with patients preparing for long-term home PN, it's important to wean the extra IV fluids while they're still in the hospital to ensure they can maintain fluid and electrolyte status with the PN alone.

Conversely, if there is an excess of body water, water restriction is necessary. Excess body water is present in both hypervolemic and euvolemic hyponatremia. Restriction of water is accomplished by decreasing or discontinuing maintenance IV fluids and decreasing or minimizing PN fluid content, In cases of SIADH, physicians also may use medications such as diuretics and demeclocycline.[9] If a patient is hypervolemic (total body sodium excess), restrict both sodium and water.

To minimize fluid volume in PN, calculate the minimum volume of stock solutions needed to provide PN nutrients. Stock solutions typically are 70% dextrose, 10% amino acids, and 20% lipid. Solutions of 20% amino acids and 30% lipid also may be available.

If using a 10% amino acid solution, a minimum of 100 mL of fluid is needed for every 10 g of amino acid (10% amino acids = 10 g amino acids/100 mL). For example, if a patient's protein need is 95 g/day, the minimum amount of solution can be calculated this way:

95 g x [100 mL ÷ 10 g] = 950 mL amino acid solution

or

[10 g ÷ 100 mL] = [95 g ÷ n mL]
Solving for n = 950 mL

If using a 70% dextrose solution, a minimum of 100 mL of fluid is needed for every 70 g of dextrose (70% dextrose = 70 g dextrose/100 mL). For example, if a patient's dextrose need is 1,400 kcal/day and 1 g of dextrose contains 3.4 kcal, the PN will need to contain 412 g of dextrose (1,400 kcal dextrose ÷ 3.4 kcal/g), or 589 mL of dextrose solution.

412 g dextrose x [100 mL ÷ 70 g] = 589 mL dextrose solution

or

[70 g ÷ 100 mL] = [412 g ÷ n mL]
Solving for n = 589 mL

If using a 20% lipid solution, a minimum of 100 mL of fluid is needed for every 200 kcal of lipids (2 kcal/mL). For example, if the PN should contain 500 lipid calories, this can be provided by 250 mL of 20% lipids (500 ÷ 2).

The inclusion of electrolytes, vitamins, and trace elements in the PN mixture requires an additional amount of approximately 120 mL or more, of fluid.[5]

Hypernatremia (serum sodium greater than 145 mEq/L): Water deficit generally results in hypertonic serum and hypernatremia. Symptoms include altered mental status, thirst, convulsions, and coma. Tachycardia and fever also can be present if there is a fluid volume deficit.

Hypovolemic hypernatremia can occur because of hypotonic fluid loss from the gastrointestinal tract, and from excessive renal fluid loss (diuretics, glycosuria) and increased insensible water loss from the skin and lungs. Hypernatremia due to water loss can occur in diabetes insipidus (DI), in which the kidneys fail to concentrate urine (nephrogenic DI), or from suppression of ADH (central DI).

Hypervolemic hypernatremia can occur from excess sodium and water that can result from excessive IV sodium administration and in primary hyperaldosteronism.[3,8,14]

Treating hypernatremia: First determine the cause of hypernatremia to ensure prescribing the correct treatment. If a water deficit exists, water should be repleted cautiously. Water deficit can be calculated in this manner:[3]

Water deficit = [(serum Na ÷ 140) - 1] x weight (kg) x 0.5 (female) or 0.6 (male)
Serum Na = 154; weight = 60 Kg; sex = male
Water deficit = (154 ÷ 140) - 1 x 60 x 0.6
Water deficit = 3.6 L

One-half of the calculated deficit should be given on the first day. Reduction in serum sodium concentration should be no greater than 10 mEq/L/day (0.5 mEq/L/hour). If water is repleted too quickly, cerebral edema, permanent neurological damage, or death can occur.[3,8] For patients in the ICU, it's important to discuss fluid requirements with the patient's physicians to determine whether water repletion is appropriate.

For a patient on PN, water can be given by increasing the PN water content, adding IV fluids and, in some cases, orally—if the patient can swallow and has sufficient bowel absorptive capacity. IV fluid choices that can provide free water include 5% dextrose and water (D5W), half normal saline (0.45% NaCl), or quarter normal saline (0.225% NaCl).[3,8,9] Table 5 shows the content of some commonly used IV fluids.

Table 5: Electrolyte Content in 1 L of Some Common IV Fluids[3]

Normal saline (0.9% NaCl): 154 mEq NaCl
Half-normal saline (0.45% NaCl): 77 mEq NaCl
Quarter-normal saline (0.225% NaCl): 38.5 mEq NaCl
Lactated Ringers' solution: 130 mEq Na, 4 mEq K, 3 mEq Ca, 109 mEq Cl, and 28 mEq lactate

In hypovolemic hypernatremia, volume expansion with isotonic or hypotonic saline is needed.[8,9] The volume and sodium in the PN can be increased as well, if necessary.

In hypervolemic hypernatremia, it's usually necessary to decrease a patient's fluid and sodium intake. To restrict sodium, avoid using sodium-containing IV fluids and decrease the amount of sodium in the PN. Since the standard amount of sodium in PN, such as 65 mEq/day, is not high, decreasing the amount in PN likely will not be beneficial. To determine whether PN is a

significant source of sodium, compare its sodium content with typical IV fluids and the body's sodium requirements.

Be sure to discuss a patient's fluid and sodium status with the patient's physician before planning PN fluid and electrolyte changes, especially for patients in the ICU. Patients may need to be maintained in a fluid-deficient state to help wean them from ventilator support or to decrease the demand on the cardiopulmonary system. Sometimes hypernatremia is maintained to prevent cerebral edema,[15] and sometimes IV normal saline is necessary, even in hypernatremic patients, to maintain blood pressure.[9]

Test Your Skills With These Practice Questions
Note: The graded examination follows the article.

Your patient requires 1,600 kcal and 95 g of protein per day. The patient has gained 7 kg since being admitted to the hospital, with 3+ edema in the lower extremities and 2+ edema in the hands. You have calculated the need for 720 dextrose calories (212 g of dextrose), 95 g of amino acids (380 kcal), and 500 kcal from lipids. The hospital pharmacy carries 70% dextrose, 10% amino acids, and 20% lipid stock solutions.

Serum lab values: Na: 128, Cl: 99, K: 3.9, Mg: 1.7, P: 3, BUN: 12, Cr: 1.4, glucose: 125

1. Which type of water and sodium problem does this patient have?
A. Hypertonic hypervolemic hyponatremia
B. Hypotonic hypervolemic hyponatremia
C. Isotonic hyponatremia
D. Hypotonic hypovolemic hyponatremia
ANSWER: B

2. This patient needs which of the following?
A. Sodium and fluid restriction
B. Fluid and sodium supplementation
C. Sodium supplementation and fluid restriction
D. Water supplementation and sodium restriction
ANSWER: A

Explanation: Hypotonic hyponatremia: sodium less than 135; hypervolemia with sodium and water excess as evidenced by edema and weight gain[3,8,10]

3. What is the minimum amount of dextrose solution you will need to treat this patient?
A. 302 mL
B. 500 mL
C. 750 mL
D. 1 L
ANSWER: A

4. What is the minimum amount of amino acid solution you will need?
A. 217 mL
B. 250 mL
C. 310 mL
D. 950 mL
ANSWER: D

5. What is the approximate total fluid volume of PN?
A. 1,200 mL
B. 1,650 mL
C. 2,000 mL
D. 2,200 mL
ANSWER: B

Explanation: See examples in the text. 302 mL dextrose + 950 mL amino acid + 250 mL 20% lipid + 120 mL (approximate) electrolytes, vitamins, and trace element solutions = 1,622 mL[5]

Part 3: Potassium, Magnesium, Calcium, and Phosphorus

Potassium
Potassium is the main intracellular cation and is needed for neurologic function, vascular tone, muscle contraction,[1] maintenance of cellular volume, pH regulation, glycogen and protein synthesis, enzyme activity, and resting cell membrane potential.[3]

The bloodstream contains only 2% of total body potassium, with the remaining 98% contained inside cells. Normal serum potassium is maintained by the kidneys and also by the process of transfer into and out of cells. Serum potassium regulation is critical, as a transfer of only 2% of intracellular potassium to the ECF increases serum levels high enough to cause death.[3] Abnormal serum potassium concentrations (hypokalemia or hyperkalemia) usually are an indication that the amount of potassium in the PN needs to be changed.

Hypokalemia (serum potassium less than 3.5 mEq/L): There are three main causes of hypokalemia: insufficient intake, increased excretion, and ECF to ICF transfer. Insufficient potassium intake refers to either an inadequate amount of potassium in the PN formula or poor dietary potassium intake just prior to starting PN therapy. Increased potassium excretion can occur from urinary losses due to diuretics, renal tubular acidosis, or an excess of mineralocorticoid, such as aldosterone. ECF to ICF transfer occurs in metabolic alkalosis, anabolism, refeeding syndrome, and hypothermia, and with the use of catecholamine drugs and insulin. Magnesium deficiency also can cause hypokalemia.[3,12]

Signs and symptoms of hypokalemia include cardiac arrhythmias, smooth muscle weakness or paralysis, and polyuria with loss of renal ability to concentrate urine.[3,9]

For PN-dependent patients, physicians usually treat hypokalemia with IV potassium chloride or potassium phosphate. Initial doses of 20 to 40 mEq of potassium chloride are used for mild to moderate hypokalemia, with the safest rate being 10 mEq of potassium per hour. Higher

and/or more frequent doses are necessary for severe and symptomatic hypokalemia, along with cardiac monitoring and rechecking serum potassium concentrations.[3,9] For patients at risk of refeeding syndrome, check serum potassium and supplement as needed, then initial underfeeding of calories and cautious advancement of calories for the first three to five days helps to prevent hypokalemia.[16]

Treating hypokalemia: Potassium can be increased in the PN. Adjust PN potassium content by using the daily requirements and the amounts of IV potassium chloride supplementation the patient has been receiving as a guide. For example, if a patient is receiving 20 to 40 mEq of IV potassium chloride per day in addition to the amount in the PN, the amount of potassium in the PN can be increased by 20 to 30 mEq/day. Since too much potassium in PN can lead to hyperkalemia, it's usually better to add a smaller amount of potassium than what has been used for supplementation. If the patient develops hyperkalemia, it may be necessary to stop PN and discard the whole bag. It's also possible to supplement potassium orally or enterally for patients with adequate bowel absorptive capacity.

It's also important to make sure a patient receives adequate magnesium to help maintain normal serum potassium concentration.[3]

Hyperkalemia (serum potassium greater than 5 mEq/L): There are three main causes of hyperkalemia: excessive intake, insufficient excretion, and ICF to ECF transfer.

For PN-dependent patients, excessive intake may occur if the PN contains too much potassium, potassium has been administered via IV bolus or other IV fluids, or a patient has received oral or enteral potassium supplementation.

The most common reason for hyperkalemia is insufficient excretion caused by renal failure. Certain medications can decrease potassium excretion, such as potassium-sparing diuretics (eg, spironolactone), non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors, and heparin. Insufficient potassium excretion also can occur in hypoaldosteronism and severe hypovolemia.[3,9]

ICF to ECF transfer of potassium can occur in metabolic acidosis. Hydrogen ions move intracellularly and potassium ions move out of cells. ICF to ECF transfer also can occur in hyperglycemia with an insulin deficit and in situations of cell breakdown that occur in severe catabolic states, trauma, and rhabdomyolysis. Drugs that can cause ICF to ECF transfer of potassium include succinylcholine and beta-andrenergic blockers.[3,9]

Signs and symptoms of hyperkalemia include cardiac arrhythmias and weakness or paralysis of cardiac and skeletal muscle.

Treating hyperkalemia: Physicians treat hyperkalemia with IV dextrose and insulin, sodium bicarbonate, or albuterol to promote potassium transfer into cells. Therapies that cause potassium removal from the body include potassium-wasting diuretics, sodium polystyrene sulfonate to bind potassium in the gastrointestinal tract, and hemodialysis for patients with renal failure.[9]

Before omitting or decreasing potassium in PN, keep in mind that potassium levels could decline because of ECF to ICF transfer—for example, if a patient is hyperglycemic and will receive insulin or if a malnourished patient has refeeding syndrome.[16]

Magnesium

Magnesium is a cofactor for enzymes involved in more than 300 biochemical processes in the body, including glycolysis and energy production. It's also involved in bone health and maintaining intracellular potassium and calcium levels. ECF contains only 1% of total body magnesium, with most of it contained in the bones, muscles, and soft tissues.[3,17] Abnormal serum magnesium concentrations usually indicate a need to adjust the amount of magnesium in PN.

Hypomagnesemia (serum magnesium less than 1.5 mg/dL): Causes of hypomagnesemia include inadequate intake, increased excretion, and intracellular transfer. Magnesium loss occurs through gastrointestinal fluid loss, increased renal excretion caused by the use of some medications (eg, amphotericin B, cyclosporine, diuretics), alcohol use, ECF volume expansion, and metabolic acidosis. [3]

Endocrine disorders that can cause hypomagnesemia include hyperthyroidism, hyperaldosteronism, and hyperparathyroidism. ECF to ICF transfer of magnesium occurs during refeeding syndrome and in anabolism as magnesium enters cells.[3,16] Signs of hypomagnesemia include cardiac arrhythmias, delirium, psychosis, seizures, and coma. Refractory hypokalemia and hypocalcemia also can occur.[9]

Treating hypomagnesemia: For critically ill patients with symptomatic hypomagnesemia, physicians replace magnesium via IV bolus in varying amounts—for example, 1 to 1.5 mEq/kg for patients with normal renal function and one-half or less of those amounts for patients with renal insufficiency.[9]

A commonly prescribed dose of magnesium sulfate for asymptomatic patients is 2 g (16 mEq of elemental magnesium) at a maximum rate of 1 g/hour. A normal-functioning kidney excretes approximately 50% of an IV dose of magnesium, which is distributed slowly in the body; thus magnesium is more likely to be retained by the body when given over a longer time period.[3,9] Therefore, increasing the amount of magnesium in PN is helpful for maintaining normal serum levels. For example, if a patient receives 18 mEq of magnesium per day in PN but requires IV magnesium replacement for persistently low levels, recommend increasing the amount of magnesium in PN to approximately 24 to 30 mEq/day with continued monitoring. Underfeeding of calories and cautiously advancing feeding during the first three to five days helps to prevent hypomagnesemia in patients at risk of refeeding syndrome.[16]

Hypermagnesemia (serum magnesium greater than 2.4 mg/dL): Causes of hypermagnesemia include renal failure or drugs that interfere with magnesium excretion, such as lithium and spironolactone. Magnesium-containing antacids or laxatives also can contribute to hypermagnesemia. Somnolence, sweating, nausea, vomiting, loss of reflexes, hypotension,

and bradycardia can occur in mild or moderate hypermagnesemia. In severe cases, respiratory depression, cardiac arrest, and death can occur.

Treating hypermagnesemia: In patients with severe symptomatic hypermagnesemia, physicians use IV calcium to alleviate the neuromuscular and cardiovascular effects. The primary treatment is to eliminate magnesium from IV fluids and PN; however, consider the potential for ECF to ICF shifts when decreasing magnesium in PN. Other treatments for hypermagnesemia include diuretics to increase magnesium excretion and, in renal failure, hemodialysis.[3,9]

Calcium

The skeleton contains more than 99% of the body's calcium, with the remaining 1% found in blood, ECF, muscle, and other tissues. Calcium has many functions, including providing structure for bones and teeth, muscle contraction, vascular contraction and dilation, transmission of nerve impulses,[1] endocrine and exocrine secretions, and blood coagulation.[9]

Approximately 50% of serum calcium is ionized and the rest is protein bound, mostly to serum albumin.[3] If a patient's serum albumin level is low, the serum calcium (as listed on the laboratory report) can be "corrected" by using the following equation: ([4 - serum albumin (g/dL)] x 0.8) + measured calcium (mg/dL). Or check the patient's serum ionized calcium level to get a more accurate picture of his or her serum calcium status.[3,9] Keep in mind that serum calcium levels do not indicate total body calcium status, as serum calcium is maintained using calcium from the bone.

Hypocalcemia (serum calcium less than 8.5 mg/dL): Hypocalcemia can occur in patients with hypoparathyroidism, hypomagnesemia, hyperphosphatemia, and acute pancreatitis as well as in those who are vitamin D deficient. The main symptoms of hypocalcemia are tetany and other nerve and muscle problems, including paresthesias, muscle aches and weakness, facial muscle spasms, lightheadedness, irritability, and seizures.[3] Long-term calcium deficiency results in osteoporosis.

Parathyroid hormone is secreted in response to hypocalcemia and causes mobilization of calcium from the bone as well as renal phosphate excretion and increased intestinal calcium absorption.

Because most PN-dependent patients can't enterally absorb adequate nutrients, dietary intake usually is not possible to improve serum calcium status; thus, bone stores are compromised. PN is not a medium to replace low serum calcium because of the danger of calcium-phosphate precipitation in the solution.[2] However, based on this author's experience, PN can contain about 10 mEq of calcium gluconate per liter, depending on the phosphorus content,to provide estimated needs of 10-20 mEq calcium per day. In times of parenteral calcium gluconate shortage or for patients who need more calcium than is contained in PN, it's reasonable to use oral supplementation if the patient has some functional small bowel, with the hope that some of the calcium will be absorbed.

Treating hypocalcemia: Physicians treat mild to moderate hypocalcemia with 1 to 2 g of calcium gluconate (4.65 to 9.3 mEq of calcium) over a period of 30 to 60 minutes. They treat severe symptomatic hypocalcemia with higher doses given more rapidly—for example, 3 g of IV calcium gluconate or 1 g of calcium chloride (13.6 mEq of calcium) over 10 minutes.[9] IV calcium should not be infused in the same catheter as PN or other phosphate-containing solutions because of the danger of precipitate formation.

Hypercalcemia (serum calcium greater than 10.5 mg/dL): Hypercalcemia can occur from increased bone resorption caused by malignancies, primary hyperparathyroidism, immobilization, hyperthyroidism, and uncommonly -excessive vitamin D (increased enteral absorption). Symptoms of hypercalcemia are nausea, vomiting, thirst, polyuria, uremia, urinary stones, confusion, coma, fatigue, incontinence, myalgia, decreased reflexes, hypertension, and cardiac arrhythmias.[3]

Treating hypercalcemia: Physicians generally treat hypercalcemia with IV 0.9% sodium chloride and diuretics to stimulate urinary calcium output. In severe cases in which patients do not respond to other treatments, dialysis may be necessary. In nonacute and more chronic conditions, such as hypercalcemia of malignancy, physicians use medications such as glucocorticoids, calcitonin, and bisphosphonates to decrease bone resorption.[3,9] For hypercalcemic patients, calcium can easily be omitted from the PN solution.

Phosphorus

Phosphorus is the main intracellular anion and a structural component of bones and teeth. As phospholipid, it's also a structural component of cell membranes.[3] Phosphorus also functions as a buffer in maintaining normal pH, in storing and transferring energy from metabolism(adenosine triphosphate- ATP), and as part of nucleic acids and nucleotides.[1] It's necessary for many other functions as well, including oxygen release from hemoglobin as part of 2,3-diphosphoglycerate.[3,9] Abnormal serum phosphorus concentrations may indicate the need to adjust the amount of phosphorus in PN.

Hypophosphatemia (serum phosphorus less than 2.5 mg/dL):

Causes of hypophosphatemia include inadequate intake, increased excretion, ECF to ICF transfer, and hypercalcemia.

Inadequate phosphorus intake can occur from malabsorption, the use of phosphorus-binding antacids, or a lack phosphorus in PN formula. Increased phosphorus excretion can occur with hyperparathyroidism and acidosis. ECF to ICF transfer of phosphorus can occur in respiratory alkalosis, the stress response, anabolism, and by the action of insulin. An intracellular shift of phosphorus from the ECF to the ICF is one of the most dangerous complications of refeeding syndrome.[16]

The effects of hypophosphatemia are related to a lack of cellular adenosine triphosphate (ATP) and decreased red blood cell 2,3-diphosphoglycerate. A lack of ATP results in less energy, while decreased 2,3-diphosphoglycerate in red blood cells results in inadequate oxygen delivery to tissues. Symptoms of hypophosphatemia include skeletal muscle weakness, bone

pain and fractures, decreased cardiac output, decreased blood pressure, confusion, convulsions, coma, and impaired function of blood cells and platelets.[3]

For patients at risk of refeeding syndrome, underfeeding calories and cautiously advancing feeding during the first three to five days helps to prevent hypophosphatemia in severely malnourished patients.[16]

Treating hypophosphatemia: Physicians treat hypophosphatemia with IV potassium phosphate or sodium phosphate over a period of four to six hours to minimize the risks of hypocalcemia or calcium-phosphate precipitation. For patients with normal renal function, doses range from 0.08 to 0.32 mM/kg for mild to moderate hypophosphatemia and 0.32 to 0.64 mM/kg for severe hypophosphatemia.[9]

For patients who are hypophosphatemic, phosphorus may be increased in the PN; however, because of the risk of calcium phosphate precipitate formation,[18,19] it should not be higher than approximately 20 mM/L of PN solution (as based on this author's experience), and this amount depends on final volume and other components of the PN solution. Published tables showing solubility curves[18] can help RDs and pharmacists determine safe amounts of phosphorus to add to PN. Some pharmacy software can alert pharmacists to excessive amounts of phosphorus prior to PN compounding.

Calcium phosphate precipitate formation is more likely with the use of calcium chloride than calcium gluconate. Calcium phosphate precipitation also is more likely with a higher solution pH, a lower amino acid concentration, higher temperature, and if calcium is added before phosphorus during PN compounding.[18,19]

Hyperphosphatemia (serum phosphorus greater than 4.5 mg/dL): Hyperphosphatemia is caused by excessive intake, insufficient renal excretion, ICF to ECF transfer, or hypocalcemia. Insufficient excretion occurs in renal failure, hypoparathyroidism, hyperthyroidism, and hypovolemia.[3] Hyperphosphatemia can also result from the transfer of phosphorus from the ICF to the ECF, which can happen from cell breakdown or injury, such as in rhabdomolysis, hemolysis, burns, and during chemotherapy and radiation therapy. Effects of hyperphosphatemia can include paresthesias, cardiac arrhythmias, and soft-tissue calcification[3].

Treating hyperphosphatemia: Physicians treat hyperphosphatemia using enteral phosphate binders, providing dextrose and insulin to promote intercellular transfer, or hemodialysis.[3,9] Phosphorus can be omitted from or decreased in PN solutions. However, keep in mind that intracellular transfer can occur in a malnourished patient, and even when elevated, serum phosphorus can deplete rapidly when PN feeding has begun.[16]

Test Your Skills With This Practice Question
Note: The graded examination follows the article.

1. Which of the following is an example of a condition in which potassium moves from ECF to ICF?

A. Refeeding syndrome
B. Metabolic acidosis
C. Hypermagnesemia
D. Hypovolemia
Answer: A

Table 6: Considerations for Adjusting Electrolyte Content of Parenteral Nutrition

- Nutritional needs
- Laboratory results, trends over time in serum electrolyte levels, possible spurious results
- Clinical reasons for increased or decreased serum electrolyte levels
- Malnutrition: risk of refeeding syndrome
- Fluid intake and output: other IV fluids, oral intake, gastrointestinal drainage, vomiting, wound drainage, ascites, edema, urine output
- Add electrolyte content judiciously to avoid overcorrection and/or waste of parenteral nutrition (PN), especially when preparing a week's supply (for home PN)
- Frequent (daily) changes in electrolyte levels are rarely necessary, even in hospitalized patients
- Before omission of potassium, magnesium, or phosphorus from PN keep in mind potential extracellular to intracellular transfer
- Consider renal function before increasing water, potassium, magnesium, or phosphorus
- Review medications or dosage changes before and after adjusting electrolyte content of PN
- Discuss potential electrolyte and fluid changes with the patient's physician.
- During times of severe parenteral electrolyte product shortages: consider using oral supplements such as calcium and phosphorus, as tolerated, for patients who have some functional small bowel and/or use premixed PN formulas, premixed electrolyte products, and different brands of amino acid products in order to include specific electrolytes, such as calcium, phosphorus, and magnesium, in the PN

Part 4: Chloride and Acid-Base Balance

Chloride, along with sodium, is necessary for maintaining ECF volume, osmolality, and acid-base balance. Chloride is the main anion of ECF and a component of gastric fluid (hydrochloric acid).[1]

Chloride is lost through gastrointestinal fluid loss, urine, and sweat. Hypo- and hyperchloremia occur along with hypo- and hypernatremia (loss of sodium chloride). Low serum chloride can occur with metabolic alkalosis, and high serum chloride concentrations can occur with metabolic acidosis.[3]

Acid-base balance refers to the balance of hydrogen ions and bicarbonate (HCO_3) in the body. A decrease in hydrogen ions results in alkalosis and elevated pH, while an increase in

hydrogen ions results in acidosis and low pH. A decrease in bicarbonate produces metabolic acidosis, and an increase in bicarbonate produces metabolic alkalosis. Serum bicarbonate, often listed as CO_2 on the basic chemistry panel, actually is a measure of the dissolved carbon dioxide, carbonic acid, and bicarbonate, and 95% of this amount is bicarbonate. So, in a practical sense, serum CO_2 on the chemistry laboratory report is a measure of serum bicarbonate.[3]

Both the kidneys and lungs control acid-base balance. The kidneys control serum levels of hydrogen ions and bicarbonate through excretion and reabsorption, while the lungs control the arterial amounts of carbon dioxide gas by ventilation. Hypoventilation causes high arterial carbon dioxide (Pa CO_2) levels and respiratory acidosis, while hyperventilation causes low arterial carbon dioxide levels and respiratory alkalosis. The ratio of bicarbonate to arterial carbon dioxide determines the pH, as explained by the following equation[3]:

Kidneys retain or excrete HCO_3 and H^+

$H^+ + HCO_3 \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2$

Lungs retain or remove CO_2

To accurately determine acid-base disorders, arterial blood gas measurements are used, which consist of pH, arterial carbon dioxide ($PaCO_2$), oxygenation, base excess (normal is 0 ± 2.5 mEq/L), and calculated bicarbonate. The acid-base disorder can be evaluated through a four-step method:

- Step 1: Assess pH (acidosis, alkalosis, or normal). Normal blood pH is 7.35 to 7.45.
- Step 2: Assess arterial carbon dioxide (respiratory). Normal arterial carbon dioxide ($PaCO_2$) is 35 to 45 mm Hg.
- Step 3: Assess bicarbonate (metabolic). Normal bicarbonate (HCO_3) is 22 to 28 mEq/L.
- Step 4: The primary abnormality usually is determined by the pH, and secondary abnormalities are compensatory changes. A high base excess confirms metabolic alkalosis, and a low base excess confirms metabolic acidosis.

In many situations, arterial blood gases are not ordered, and the clinician can discern the cause of the acid-base abnormality if there are obvious reasons, some of which are discussed below.

Metabolic alkalosis can develop from excessive bicarbonate intake, loss of hydrogen ions, or loss of chloride from body fluids or because of hypovolemia (contraction alkalosis). In metabolic alkalosis, the pH and bicarbonate (CO_2 on the chemistry report or HCO_3 on arterial blood gas measurements) will be high. The lungs compensate by retaining carbon dioxide gas to bring down the pH, which will cause measured arterial carbon dioxide ($PaCO_2$) to increase.

This is called respiratory compensation. It's common to find compensatory respiratory acidosis along with primary metabolic alkalosis.

One example of metabolic alkalosis is the loss of hydrogen and chloride, which occurs from excessive gastric drainage, vomiting, or excess diuretic use. The kidneys will reabsorb bicarbonate to maintain electrolyte balance. Metabolic alkalosis also can develop when a chronically hypercapnic patient with respiratory acidosis is placed on ventilator support. The arterial carbon dioxide improves to correct the respiratory acidosis, yet the patient's kidneys, accustomed to retaining bicarbonate, take several days to respond, resulting in posthypercapnic metabolic alkalosis.[3]

Metabolic acidosis can develop from the excessive production or inadequate excretion of acids, excessive bicarbonate loss, or bicarbonate loss with excess chloride intake. In metabolic acidosis, pH and bicarbonate are low. The lungs compensate by increasing respiration to remove carbon dioxide and the arterial carbon dioxide decreases. It's common to find compensatory respiratory alkalosis along with primary metabolic acidosis.

Some examples of metabolic acidosis include diabetic ketoacidosis, lactic acidosis in sepsis, bicarbonate loss from renal failure (renal tubular acidosis), and bicarbonate loss from bowel fistulae, ileostomy, or pancreatic or biliary drainage. Metabolic acidosis also can occur from bicarbonate loss and increased chloride retention after urinary diversion (ileal conduit) procedures.[3]

A complete discussion of acid-base balance is beyond the scope of this article and is discussed elsewhere.[3,12,20] The purpose here is to introduce the subject as it relates to PN use. The amounts of acetate and chloride in PN can be adjusted in cases of metabolic acid-base disorders to assist the physician's efforts to correct acid-base abnormalities. Bicarbonate is not used in PN formulations because it's not stable; thus, the metabolic precursor acetate is used.[2,21,22]

In cases of metabolic acidosis, more sodium acetate and potassium acetate salts, and less chloride salts, should be used to increase the amount of acetate in the PN formula. In cases of metabolic alkalosis, acetate salts should be minimized and more chloride salts (sodium or potassium) can be used.[21] The amount of cations (sodium and potassium) used will determine the amount of anions (chloride and acetate) that can be contained in the PN formula. Adjusting the amounts of acetate and chloride in PN can help the physician correct acute acid-base problems for hospitalized patients and can help maintain normal acid-base balance for long-term home PN patients, which is important because chronic metabolic acidosis can result in loss of bone minerals.[21,23]

Respiratory acidosis can occur in pulmonary disease, respiratory failure, or with excessive carbon dioxide production from overfeeding. pH will be low, and arterial carbon dioxide ($PaCO_2$) will be elevated. The kidneys will retain bicarbonate to compensate, causing bicarbonate and serum carbon dioxide to increase (compensatory metabolic alkalosis). Respiratory alkalosis can occur with hyperventilation, causing low arterial carbon dioxide and

high pH. The kidneys will excrete bicarbonate to compensate, causing the bicarbonate and serum carbon dioxide to decrease (compensatory metabolic acidosis).

While it is appropriate to adjust PN acetate and chloride for metabolic acid-base disorders, it is not appropriate to adjust PN acetate and chloride for acid-base disorders of respiratory origin or for compensatory metabolic responses.

Spurious Lab Results

Infrequently, blood samples become contaminated with PN or other IV infusions, resulting in incorrect reports showing severe and abrupt elevations in glucose, high potassium and/or low sodium concentrations. This can occur if the correct blood sampling technique is not followed—for example, if PN or IV fluids are not stopped, a small aliquot of blood is not discarded, or there is an insufficient time lapse after stopping IV infusions prior to collecting the blood sample.[24]

False hyperkalemia also can occur with traumatic or excessive tissue manipulation with blood draws and by increased red cell permeability and breakdown as the blood sample ages because of the release of intracellular potassium into the serum sample.[12,25]

Incorrect or spurious lab test results can cause waste because of repeat lab draws and testing. More important, there is a risk to the patient if medical decisions are made based on the erroneous results. If lab results suddenly and significantly deviate from prior results, repeat the blood work, with attention toward correcting the sampling technique.

Conclusion

Fluid and electrolyte management in PN is complex. Knowledge of the basic requirements and clinical situations in which a patient's fluid and electrolyte needs increase or decrease is helpful for understanding how to make appropriate PN adjustments.

More clinical information, not just lab information, is needed to properly evaluate and treat sodium, water, and other electrolyte problems. In general, increases and decreases in serum electrolyte concentrations result from abnormalities in fluid and electrolyte intakes, losses, and intracellular and extracellular transfer.

Although there are no standard guidelines for adjusting electrolytes and fluid in PN, with knowledge and experience, RDs can become proficient at making appropriate formula adjustments for their patients who use PN.

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Resources

- Dietitians in Nutrition Support dietetic practice group of the Academy of Nutrition and Dietetics

- Parenteral nutrition electrolyte/mineral product shortage considerations. American Society for Parenteral and Enteral Nutrition website.

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Examination

1. Which of the following are the electrolyte components used PN?
A. Sodium, potassium, phosphorus, and strontium
B. Sodium, chloride, potassium, magnesium, calcium, phosphorus, and acetate
C. Sodium, acetate, and chloride only

2. Which of the following are the three main reasons that a patient's serum potassium, magnesium, and phosphorus concentrations increase and decrease?
A. Changes in body temperature, body position, and IV fluid content
B. Only from changes in the PN content of potassium, magnesium, and phosphorus
C. Intake, excretion, and intracellular and extracellular transfer

Questions 3 and 4 refer to the following case: A patient began PN in the hospital and has been home for two weeks with a resolving bowel obstruction. The patient tells you that her gastric drainage output has decreased and she is taking the PN as ordered. She also complains of ankle edema and swelling in her feet and hands, and is concerned about her weight gain.

Electrolytes, BUN, and creatinine are normal except Na = 132.
Weight: 135 lbs (61 kg), up from 124 lbs (56 kg) in the hospital
PN contains 3-L volume, 200 mEq Na, 145 mEq Cl, 55 mEq acetate, 70 mEq K, 24 mEq Mg, 10 mEq Ca, and 18 mM P per day.

3. How will you adjust the electrolytes in this patient's PN?
A. Reduce potassium and chloride
B. Reduce sodium and chloride
C. Increase acetate
D. Increase phosphorus

4. How many liters of PN fluid will you suggest with the next order?
A. 1.2
B. 2
C. 3.5
D. No change

Questions 5, 6, and 7 refer to the following case: A patient has an inoperable bowel obstruction due to metastatic colon cancer, and a percutaneous endoscopic gastrostomy (PEG) tube has been placed for palliative gastric drainage. He is now on home PN. He complains of thirst and has been drinking a lot of fluids, all of which drain from the G-port, and he has not been measuring it. He has been losing weight during the past four weeks, and you have determined that he needs more calories. You receive a message from the home nurse that the patient has had severe weight loss in the past week and is complaining of weakness.

Chemistry Labs: BUN: 45, Cr: 0.8, K: 3, Na: 126, Cl: 70, CO_2: 47
Height: 5'11"; weight: 174 lbs (79 kg); was 189 lbs (86 kg) last week
Current 3 L volume PN that is infused on a 14 hour nocturnal cycle. The PN contains 2,100 kcal, 120 g amino acids, 110 mEq Na, 90 mEq K, 115 mEq Cl, 55 mEq acetate, 20 mM P, 8 mEq Ca, 18 mEq Mg, and standard trace elements and multivitamins.

5. What factors, other than calorie expenditure, are contributing to this patient's weight loss?
A. Excessive fluid intake
B. Fluid loss/ECF volume expansion
C. Fluid loss/ECF volume depletion
D. Insufficient phosphorus in the PN

6. What acid-base problem exists?
A. Metabolic alkalosis
B. Metabolic acidosis
C. No acid-base problem
D. Respiratory alkalosis

7. What changes will you make to this patient's PN?
A. Increase sodium, potassium, and acetate; decrease volume and chloride
B. Increase volume and sodium; decrease potassium
C. Increase volume, sodium, potassium, and chloride; decrease acetate
D. Increase potassium; decrease volume and sodium

Questions 8, 9, and 10 refer to the following case: You are developing a PN feeding plan for a patient who has a high-output ileostomy. The patient is losing 2.5 L/day from the ostomy. He states that his urine looks dark, and he is urinating less often and in smaller amounts than usual.

Labs: Na: 128, Cl: 91, BUN: 55, Cr: 1.2, CO_2: 18, K: 3.8, Mg: 1.7, P: 4.5
Weight: 147 lbs (67 kg), down from 152 lbs (69 kg) one week ago

8. Which of the following fluid and electrolyte disorders does the patient have?
A. Hypervolemic hyponatremia
B. Normal serum sodium
C. Euvolemic hyponatremia
D. Hypovolemic hyponatremia

9. This patient is losing an excessive amount of which nutrients and electrolytes?
A. Sodium, chloride, bicarbonate, and water
B. Phosphorus and water
C. Glucose and hydrogen
D. None of the above

10. What changes to the PN will be helpful to improve hydration status and acid-base balance?
A. Decrease water, chloride, and sodium
B. Increase sodium, chloride, acetate, and water
C. Add bicarbonate and water
D. Decrease chloride

Question 11 refers to the following case: A severely malnourished patient is admitted to the hospital with poor intake and weight loss due to an obstructing duodenal mass. A workup is in progress to determine whether the patient will have surgery, a duodenal stent, or a jejunal feeding tube placement. The patient has received some IV hydration, and you have been consulted to provide PN recommendations so that he can be nourished during this time.

Height: 5'10"; weight: 114 lbs (52 kg)
PN is started at a volume of 1,500 mL, providing 1,100 total kcals, which consist of 148 g of dextrose, 150 mL of 20% lipid, and 75 g of protein per day. Serum electrolyte levels are within the normal range, except the phosphorus level is 4.8 with a normal creatinine of 1 and normal urine output. The PN has been ordered with no phosphorus content.

11. What is your recommendation regarding this patient's PN?
A. Continue PN plan as ordered until enteral feeding commences.
B. Add 12 mM P/day to the PN prescription.
C. Add 60 mM P/day to the PN prescription.
D. Increase the volume of the PN to 3.6 L/day.

Questions 12 and 13 refer to the following case: An 80-year-old man is admitted to the hospital from a nursing home with a history of decreased oral intake, altered mental status, and a urinary tract infection.

Labs: Na: 152, Cr: 1.5, BUN: 54, Cl: 122
Height: 5'10"; weight: 143 lbs (65 kg)

12. What is the patient's free-water deficit?
A. 4.2 L
B. 3.3 L
C. 1.5 L
D. No deficit

13. How much water should be repleted in the first 24 hours in addition to this patient's maintenance fluid requirements?
A. 0.5 L
B. 1.6 L
C. 3.3 L
D. 6.6 L

Questions 14 and 15 refer to the following case: A nurse asks you about the adequacy of the sodium content of a patient's PN because the patient has hyponatremia.

Labs: Na: 131, Cl: 101, glucose: 380, BUN: 23, Cr: 1
Weight: stable but patient is complaining of hunger and thirst

14. What is the estimated "corrected" serum sodium concentration?
A. 128
B. 130.5
C. 135
D. 145

15. Does this patient require more sodium in the PN?
A. Yes
B. No

Questions 16, 17, and 18 refer to the following case: A patient is in the surgical ICU following an abdominal aortic aneurysm repair with a postoperative ileus. You have been consulted to evaluate for PN. The patient's lab work is unexpectedly abnormal.

Chemistry Labs: Na: 131, Cl: 112, K: 5.4, CO_2: 14, Mg: 2, P: 5.4, Ca: 7.1, albumin: 2.9, BUN: 56, Cr: 2
Arterial blood gas measurements: pH: 7.2, $PaCO_2$: 30, HCO_3: 16, base excess: -5.8

16. What type of acid-base problem do you find?
A. Metabolic acidosis with respiratory compensation
B. Respiratory acidosis with metabolic compensation
C. Respiratory alkalosis with metabolic compensation
D. Metabolic alkalosis

17. What changes will you make to the PN content?
A. Increase acetate; decrease chloride
B. Increase chloride; decrease acetate
C. Add potassium acetate
D. Increase magnesium content

18. What other electrolyte changes are necessary?
A. Increase sodium and chloride
B. Decrease potassium and phosphorus
C. Decrease sodium and magnesium
D. Increase potassium and sodium

Questions 19 and 20 refer to the following case: A patient is in the cardiac ICU with congestive heart failure and generalized edema. Patient's weight is 187 lbs (85 kg), up from 167 lbs (76 kg) three days ago, and the total fluid intake has been higher than output since admission. The patient has been receiving furosemide (Lasix), and the physicians have ordered 20 mEq KCl given intravenously three to four times daily for the past three days.

Labs: Na: 129, Cl: 113, K: 3, Cl: 98, CO_2: 21, Mg: 1.9, P: 3.1, BUN: 23, Cr: 1.1, Ca: 7.2, albumin: 2.2

19. What type of hyponatremia does this patient have?
A. Hypertonic hypervolemic
B. Hypotonic hypervolemic
C. Hypotonic hypovolemic
D. Isotonic hypervolemic

20. What changes do you suggest for the next bag of PN?
A. Concentrate the formula for less fluid and omit phosphorus.
B. Increase sodium chloride by 20 mEq/day.
C. Decrease potassium chloride by 40 mEq/day.
D. Concentrate the formula for less fluid and increase potassium by 40 mEq per day.

21. You notice that your patient on long-term PN has no added calcium in their PN formula. A colleague tells you that there is no need to add it because the patient's serum calcium concentration is normal. Your plan is to do which of the following?
A. Continue the same PN with no calcium.
B. Provide 10 mEq calcium per day in the PN.
C. Remove phosphorus from the PN
D. None of the above

22. A severely malnourished patient who weighs 60 kg is going to to begin PN with a volume of 1.8 L You have recommended a low calorie level due to the risk of refeeding syndrome. How much potassium will you specify to be in the patient's PN?
A. 15 mEq/L
B. 40 mEq/day
C. 120 mEq/L
D. 95 mEq/day