

Nutrition, Hydration, Electrolytes, and Acid–Base Balance

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This chapter will review important aspects of nutritional assessment, risks for malnourishment, and interventions in the older adult. Fluid balance will be discussed, as well as specific electrolyte derangements and their consequences. Finally, acid–base balance will be explained, including identification and consequences of both respiratory and metabolic conditions.

NUTRITION

Nutrition, hydration, and electrolyte balance are related and can have a profound impact on a person's functional status, immune competence, and overall well-being. Beyond eating for physiological survival, food has social and cultural significance. This complex view of food often is enhanced in older adults who survived the Great Depression or other hardships during which food was scarce.

The term *malnourished* can refer to individuals who are undernourished or even those who are obese. Either problem can lead to chronic illnesses and contribute to morbidity and mortality.

When assessing nutritional status, one must remember that caloric requirements are a function of basal metabolic rate (BMR) and activity level. BMR is calculated as follows:

Women: $BMR = 655 + (4.35 \times \text{weight in pounds}) + (4.7 \times \text{height in inches}) - (4.7 \times \text{age in years})$

Men: $BMR = 655 + (6.23 \times \text{weight in pounds}) + (12.7 \times \text{height in inches}) - (6.8 \times \text{age in years})$

This calculation accounts for the gradual decline in BMR that occurs with age. Because the activity level for most older adults decreases with time, caloric requirements are reduced accordingly. Older adults who do not reduce their caloric intake or increase their caloric expenditure will notice an increase in body weight. However, extreme dietary restrictions may lead to excessive weight loss, depression, anxiety, postural hypotension, or skin problems. The chronic diseases that affect many older adults (e.g., hypertension; congestive heart failure; renal, liver, and pulmonary diseases) often require dietary interventions for disease management.

Early identification of and intervention with at-risk persons or those with nutritional deficiencies can result in improved health and quality of life for older adults (Council on Practice, 1994; White, 1991).

Nutritional Screening

Nutritional screening is the first step in identifying people who are at risk for poor nutrition and its complications. Multiple screening tools are available, but one of the most widely used was developed as part of the Nutrition Screening Initiative (NSI), which was a collaboration among the American Academy of Family Physicians, the American Dietetic Association, the National Council on Aging, and 35 other aging agencies (Dwyer, 1991). The tool—DETERMINE Your Nutritional Health—can be used as a self-evaluation or completed by a caregiver or healthcare professional. The NSI tool identifies persons who need a comprehensive nutritional assessment by a score of 3 or more (see www.vda.virginia.gov/pdfdocs/Nutritional_Chk1st.pdf).

Nutritional Assessment

A nutrition assessment is more comprehensive than a screening and usually is completed by a registered dietitian or as a collaborative effort among the dietitian, nurses, or other members of the healthcare team. The components of a comprehensive nutrition assessment follow.

Dietary History and Intake

- Food preferences and eating habits
- Cultural or religious food practices
- Meal schedule
- Fluid intake (types)
- Alcohol intake
- Special diets
- Vitamin or supplement use

Social and Cognitive Factors

- Functional limitations
- Control over food choices and preparation
- Financial status
- Cognitive changes affecting appetite and self-feeding
- Psychosocial issues such as depression or isolation

Clinical Evaluation

- Chronic illnesses
- Physical exam
- Oral health
- Chewing and swallowing
- Cognitive or psychological assessment

- Medications
- Lab work (e.g., complete blood count [CBC], electrolytes, blood urea nitrogen [BUN], creatinine, serum proteins, pre-albumin, lipids)

Anthropometric Assessment

- Body mass index (BMI)
- Skinfold measurements
- Waist circumference (fat distribution)
- Weight changes (usual weight)

Physiological Changes Affecting Nutrition

The physiological changes of aging that affect nutritional status include

- Declining sensory function (e.g., vision, smell, taste, hearing)
- Declining gastrointestinal function, inhibiting digestion and excretion
- Delayed gastric emptying, leading to early satiety
- Changes in oral cavity, particularly dentition and taste buds
- Mouth dryness
- Decreased metabolic rate
- Decreased hepatic and renal reserves
- Diminished thirst
- Declining functional status.

Not only does declining sensory function affect appetite and interest in food, but these changes also may affect the older adult's ability to detect foods that are spoiled. Altered smell and taste can increase the risk of foodborne illness.

Changes in body composition occur with the aging process. A decrease in lean body mass begins in the third decade of life. Due to a simultaneous increase in body fat, there often is little change in weight. Loss of lean body mass can be attenuated by exercise, which has been shown to improve functional status by 10–20 years (Cress, Buchner, Questad, Essehman, deLateur, & Schwartz, 1999). At ages 65–70, there is usually a decline in body weight that continues until death (Morley & Thomas, 1999). A retrospective analysis of data from the Systolic Hypertension in Elderly Program (1984–1990) demonstrated that in older adults, weight stability predicted mortality better than high or low baseline body mass index (Somes, Kritchevsky, Shorr, Pahor, & Applegate, 2002).

Psychosocial Factors Affecting Nutrition

Psychosocial aspects play an important role in the desire to eat as well as the acquisition of nutritional foods. Older adults who live alone may have little motivation to prepare and consume a balanced meal. Inadequate resources to buy healthy foods is another problem. Less-expensive foods tend to be those with the least nutritional value. Federal programs only reach about one-third of the population in need. In summary, psychosocial factors that affect nutrition are

- Poverty
- Culture
- Social isolation
- Depression
- Dementia
- Inability to access programs/transportation
- Lack of education/information.

Food Guide Pyramid

The U.S. Department of Agriculture (USDA) updated the Food Guide Pyramid in 2005. The USDA website also provides additional information for seniors as well as the opportunity to create individualized meal plans at www.mypyramid.gov.

Oral, Dental, and Swallowing Conditions

The following oral and swallowing problems may affect a person's ability to consume a well-balanced diet:

- Tooth decay
- Missing teeth or ill-fitting dentures
- Periodontal disease
- Xerostomia (dry mouth)
- Taste disorders
- Oral infections or lesions
- Drugs affecting taste, appetite, nausea, dry mouth, and level of consciousness
- Dysphagia related to aging, central nervous system difficulties, or neuromuscular diseases.

About half of all cancers occur in people ages 65 or older, with an average survival rate of 5 years. Alcohol and tobacco use are the greatest risk factors. Treatment of these cancers may result in pain, swallowing difficulties, and immunosuppression and can affect nutritional status.

Failure to Thrive

Often older adults who are losing weight will be diagnosed as having “failure to thrive.” These individuals have a decreased appetite, poor nutritional status, declining functional status, and often are clinically depressed, putting them at risk for dehydration, falls, and impaired immune function. Survival depends on detection and reversal of the cause when possible or any intervention that improves nutritional status, such as medications that stimulate appetite.

Possible causes of failure to thrive are

- Infection (e.g., HIV/AIDS, tuberculosis)
- Cancer
- Inflammatory disease (e.g., polymyalgia rheumatica, rheumatoid arthritis)
- Endocrine disorders (e.g., diabetes mellitus, thyroid disease)
- Organ failure (e.g., heart failure, end-stage lung disease, renal failure)
- Medications (any)
- Psychosocial problems (e.g., depression, grief, intention)
- Neurological disorders (e.g., Parkinson's disease, stroke)
- Cognitive problems (e.g., Alzheimer's dementia, vascular dementia)
- Neglect and abuse.

Nutrition Interventions

Treatment of under-nutrition is initially aimed at correcting reversible causes when possible. For example, treating depression or periodontal disease may improve intake without other interventions. Avoidance of restrictive diets without exacerbating underlying disease is another strategy to improve intake. For example, low-fat foods may not be palatable to some persons or may be too calorie restrictive for others. Therefore, a choice to liberalize diet while giving cholesterol-lowering medications may be made. Foods also can be made more calorie-dense without increasing the volume of the feeding. Examples are using whole milk instead of low-fat milk; adding protein powder to cereals, soups, sauces, or beverages; adding butter to hot foods;

or adding sugar, corn syrup, or honey to sweet foods. Mixing a powdered breakfast drink, whole milk, and ice cream can make a delicious, relatively inexpensive calorie-dense milkshake. Other interventions include the following:

- Time oral supplements not to interfere with meals.
- Refer patients with dysphagia to a speech therapist.
- Assist clients with eating problems.
- Monitor bowel function and treat constipation.

FLUID BALANCE

Body fluid is located primarily in the intracellular (ICF; 60%) or extracellular (ECF; 40%) compartments. The ECF compartment is composed of the blood volume (one-third) and the interstitial space (two-thirds).

Total body water decreases with age. A young adult's body is approximately 60% water, whereas an older adult has only 40% total body water. This decrease in total body water combined with decreased thirst leading to decreased intake, increased sodium loss, and increased insensible fluid losses (through the bowel, skin, and respiratory systems) greatly increases the risk of dehydration in older adults. Altered cognitive status (e.g., remembering to drink) also may increase risk, as can physical limitations and diuretic use. Alterations in fluid balance in turn affect electrolyte balance (O'Donnell, 1995). The types and causes of dehydration are listed in Table 8-1.

Movement of Fluid

Fluid movement in the body occurs by

- *Filtration*—Fluid moves through a semi-permeable membrane from an area of higher hydrostatic pressure to lower pressure.
- *Diffusion*—Solutes (particles) move across a semi-permeable membrane from an area of higher concentration to lower concentration.
- *Osmosis*—Water moves across a semi-permeable membrane from an area of lower particle concentration to higher concentration.
- *Active transport*—Particles move against a pressure gradient; this requires energy.

Table 8-1. Types and Causes of Dehydration

Type	Description and Causes
Isotonic	Equal loss of sodium and water Gastrointestinal illness
Hypertonic	Most common cause Water loss exceeds sodium loss Fever Limited fluid intake
Hypotonic	Sodium loss exceeds water loss Diuretic use

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 41–44), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Osmotic pressure is created by the particle concentrations on either side of a semi-permeable membrane. Sodium is the major contributor to osmotic pressure. Oncotic pressure is the “pulling” force created by the concentration of particles that cannot pass through a membrane. Proteins in the bloodstream are a major contributor to oncotic pressure.

Fluid Regulation

The kidneys are the main organs involved in regulation of body fluids. In states of dehydration (hypovolemia), aldosterone and antidiuretic hormone (ADH) are secreted by the posterior pituitary, causing an increase in sodium and fluid retention by the kidneys. Excessive volume causes suppression of these hormones, leading to increased urine output.

Dehydration

In addition to age-related physiological changes in the kidneys are many other factors that may lead to dehydration in older adults:

- Infections (e.g., pneumonia, cystitis)
- Disease states (e.g., congestive heart failure, diabetes, chronic obstructive pulmonary disease, depression)
- Environmental conditions
- Decreases in thirst sensation
- Decreases in functional ability
- Restraints
- Limited intake due to fear of incontinence.

The following interventions can prevent dehydration:

- Encourage patient fluid intake of 1,000–3,000 ml daily (e.g., filling a pitcher each day and making sure it is empty at the end of the day).
- Monitor patient lab values for changes.
 - Increased BUN/creatinine
 - Increased serum sodium
 - Increased serum osmolarity
 - Increased hematocrit.
- Monitor patient urine output.
- Monitor patient for constipation or diarrhea.
- Weigh patient daily.
- Teach patient to drink despite not feeling thirsty, particularly if taking diuretics.
- Advise patient to avoid alcoholic, carbonated, and caffeinated beverages, which can increase diuresis.

Fluid Imbalances

Table 8–2 summarizes three types of fluid imbalances.

ELECTROLYTES

Electrolyte imbalance can lead to serious consequences in older adults. Dehydration is the most common precipitant of electrolyte disturbances. Because the causes of dehydration are numerous (see above), electrolyte imbalances are common in older adults.

Electrolytes are inorganic substances (e.g., acids, bases, salts) that break up into ions in solution. Ions may be positively charged (*cations*) or negatively charged (*anions*). Blood testing measures the concentration of various electrolytes in the ECF. Because many electrolytes (e.g., potassium,

Table 8-2. Types of Fluid Imbalances

	Hypovolemia	Hypervolemia	Hypoproteinemia
Definition	Extracellular fluid deficit	Extracellular fluid excess	Loss of oncotic pressure leads to hypovolemia
Causes	Hemorrhage Overdiuresis Vomiting/diarrhea Third-spacing (ascites, burns)	Congestive heart failure Renal failure Liver disease Overzealous IV fluids Sodium overload	Decreased protein intake Increased protein loss Liver/kidney disease Burns Infection Hemorrhage
Clinical findings	Dry mucous membranes Sudden weight loss Oliguria Tachycardia Orthostatic hypotension	Sudden weight gain Pitting edema Tachycardia Tachypnea Elevated blood pressure Elevated jugular venous pressure	Weight loss Impaired healing Edema Immune compromise
Interventions	Correct underlying conditions IV volume replacement Isotonic fluid (0.9% NS, Lactated Ringers) Whole blood, PC, plasma	Correct underlying conditions Semi-Fowler's position Administer diuretics Limit sodium Assess for signs and symptoms of pulmonary edema: crackles in lungs, cough, increased respiratory effort	Complete nutritional assessment High-protein diet IV replacement Whole blood Albumin Plasma

Note. NS = normal saline; PC = packed red blood cells.

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 41–55), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

magnesium) are most abundant in the ICF compartment, the amount in the ECF compartment is but a small portion of the amount in the whole body.

This section discusses the most common electrolytes: sodium (Na^+), potassium (K^+), chloride (Cl^-), phosphorus (PO_4^-), calcium (Ca^{++}), and magnesium (Mg^+).

Sodium (Na^+)

Sodium balance is an index of body water excess or deficit. Hyponatremia (low sodium; see Table 8-3) may result from a loss of sodium in excess of water (primary salt depletion) or from an excess of water, which dilutes the sodium level (dilutional hyponatremia). Most hyponatremia occurs in older adults because of the kidney's inability to excrete free water. With age, the renin-angiotension-aldosterone response is less vigorous, leading to less-efficient resorption of sodium. Congestive heart failure and liver failure can further add to this problem. Older adults often have hyponatremia resulting from inappropriate secretion of ADH (SIADH), which causes water retention and dilutes the sodium.

Table 8–3. Hyponatremia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
<i>Loss of sodium</i>	Anorexia	Review medications
• Vomiting	Nausea/abdominal cramps	Monitor laboratory data
• Diarrhea	Vomiting	<i>Sodium deficit</i>
• Burns	Lethargy	• Daily weights
• Hemorrhage	Confusion	• Intake and output
• Adrenal insufficiency	Muscle twitching	• Encourage high-sodium foods
• Diuretics	Seizures	• Skin care
<i>Gain of water</i>	Coma	• Isotonic IV fluid replacement
Increased fluid intake		• 3% NaCl solution
• Excessive D5W (5% dextrose in water)	Serum Na <135 mEq/L	<i>Water excess</i>
• Psychogenic polydipsia	Serum osmolality <285 mOsm/kg	Daily weights
• Hypotonic/isotonic tube feedings with excessive H ₂ O		Intake and output
Decreased renal function		Water restriction
Chronic heart failure/liver failure		Possible medications
<i>Impaired renal H₂O excretion</i>		• Demeclocycline
• Antidepressants (e.g., selected serotonin reuptake inhibitors [SSRIs], tricyclic antidepressants [TCAs])		• Lithium
• Carbamazepine (Tegretol)		• Furosemide with increased Na–K intake
• Thioridazine (Mellaril)		Safety precautions
<i>Diseases associated with SIADH</i>		
Certain cancers		
• Oat cell of lung		
• Duodenal		
• Pancreas		
HIV/AIDS		
Head trauma		
Stroke		
Tuberculosis		

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 59–88), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Hypernatremia (see Table 8–4) results from excess ingestion or administration of sodium or, more commonly, from a water deficit due to diarrhea or decreased intake. Key points about sodium include the following:

- It is the most abundant electrolyte in ECF contributing to osmotic pressure.
- It cannot permeate to the cell membrane.
- It is absorbed from the gastrointestinal tract and excreted in urine.
- Chloride loss follows sodium loss.
- Aldosterone maintains sodium balance in the body by promoting renal tubular resorption.
- ADH reduces sodium concentration by stimulating water retention.

Table 8-4. Hyponatremia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Decreased water intake	Thirst (earliest)	Encourage fluid intake
Diminished functional capacity	Dry mucous membranes	Decrease Na ⁺ intake
Dementia	Tachycardia	Administer hypotonic IV fluids
Altered thirst sensation	Oliguria	
High Na ⁺ IV fluids	Confusion	
Vomiting	Lethargy	
Watery diarrhea	Delirium	
Excessive sweating	Stupor	
Fever	Coma	
Excess sodium ingestion	Serum Na >145 mEq/L	
Diabetes insipidus		

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 59–88), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Potassium (K⁺)

Potassium is most abundant in the ICF, where 98% of total body potassium is located. Only 2% of the body's potassium is in the ECF. The high concentration of intracellular potassium is maintained by the Na⁺-K⁺ pump, which controls potassium flux across the cell membrane based on the body's needs. The kidneys excrete 80% of the potassium lost each day, with the other 20% being lost through the bowels (15%) and skin (5%; Metheny, 2000). Imbalances in potassium can cause life-threatening cardiac arrhythmias, including ventricular tachycardia, ventricular fibrillation, and asystole.

Common causes of hypokalemia can be divided into renal, gastrointestinal, sweat losses, and intracellular shifts. Inadequate dietary intake of potassium rarely causes deficiency unless there are concomitant causes of increased loss (e.g., diuretics, diarrhea). Clinical manifestations of hypokalemia are usually not apparent until the serum potassium falls below 3.0 mEq/L. However, patients who are taking digitalis may be more susceptible to arrhythmias at only minor reductions. Symptoms of hypokalemia may include fatigue, cardiac arrhythmias, electrocardiogram (ECG) changes, skeletal or respiratory muscle weakness, muscle cramps, adynamic ileus, impaired insulin release, and sensitivity (see Table 8-5).

Hyperkalemia is uncommon in people with normal renal function but may occur if over-replacement exceeds the kidney's ability to excrete potassium. Also, potassium supplementation given in combination with drugs that interfere with potassium elimination (e.g., angiotensin-converting enzyme inhibitors [ACEI], nonsteroidal anti-inflammatory drugs [NSAIDs], potassium-sparing diuretics) can lead to severe hyperkalemia. Other causes are due to shifts in potassium out of the cells in states of acidosis and decreased aldosterone production, which causes potassium retention. Clinical manifestations of high potassium are cardiac arrhythmias, ECG changes, muscle weakness or paralysis, nausea, diarrhea, and intestinal colic (see Table 8-6).

Table 8-5. Hypokalemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
<i>Renal losses</i> <ul style="list-style-type: none"> • Potassium-wasting diuretics • Excess aldosterone • High glucocorticoid levels • Licorice ingestion (contains enzyme that acts like aldosterone) • Osmotic diuresis • Hypomagnesemia 	<i>Skeletal muscle</i> <ul style="list-style-type: none"> • Weakness • Fatigue • Diminished reflexes • Pain or cramps • Paralysis 	Identify patients at risk (especially those taking digitalis) Give oral supplements with food to decrease gastrointestinal side effects Educate about dietary sources of potassium (e.g., dried fruit, bananas, orange juice)
<i>Gastrointestinal losses</i> <ul style="list-style-type: none"> • Vomiting • Gastric suction • Diarrhea • Ileostomy • Villous adenoma 	<i>Cardiovascular</i> <ul style="list-style-type: none"> • Problems with blood pressure regulation • Postural hypotension • Increased digitalis sensitivity • Arrhythmias 	
<i>Intracellular shifts</i> <ul style="list-style-type: none"> • Alkalosis • Hyperinsulinemia • Beta adrenergic agonists (albuterol) • Hypothermia 	<i>Gastrointestinal</i> <ul style="list-style-type: none"> • Decreased bowel sounds <i>Respiratory</i> <ul style="list-style-type: none"> • Shortness of breath with shallow respirations 	Salt substitutes contain 50–60 mEq per teaspoon and may be dangerous for persons on potassium-sparing diuretics or other medications that cause potassium retention (e.g., angiotensin-converting enzyme inhibitors [ACEIs], angiotensin II receptor antagonists [ARBs], nonsteroidal anti-inflammatory drugs [NSAIDs])
<i>Poor dietary intake</i> <ul style="list-style-type: none"> • Anorexia nervosa • Alcoholism • Sweat losses in persons acclimated to heat 	<i>Central nervous system</i> <ul style="list-style-type: none"> • Confusion <i>Renal</i> <ul style="list-style-type: none"> • Impaired concentrating ability causing polyuria • Serum $K^+ < 3.5$ mEq/L • Alkalosis common 	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 91–108), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Key points about potassium include the following:

- It is the most abundant electrolyte (cation) in the ICF.
- Balance is maintained by the $Na^+ - K^+$ pump.
- Aldosterone is the most important hormone regulating potassium homeostasis.
- Low magnesium levels can lead to hypokalemia (must be corrected together).
- High glucocorticoid levels (Cushing's syndrome or exogenous administration) cause potassium depletion.
- Catecholamines promote movement of potassium into the cells.

Calcium (Ca^{++})

Ninety-nine percent of the body's calcium is located in bones and teeth. The 1% of calcium that is circulating is partly ionized (47%) and partly bound to protein (53%). Calcium is important for the following body functions:

- Transmission of nerve impulses
- Skeletal and cardiac muscle contraction and relaxation

Table 8–6. Hyperkalemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Pseudohyperkalemia from fist clenching during blood draw or specimen hemolysis	<i>Cardiovascular</i>	Identify patients at risk (especially those with renal disease)
<i>Decreased renal excretion</i>	• EKG changes = narrow, peaked T waves, shortened QT interval, prolonged PR interval	Avoid salt substitutes, potassium supplements, or potassium-sparing diuretics in patients with renal disease
• Chronic kidney disease (CKD)	• Ventricular arrhythmias	Advise patients to avoid high-potassium foods (e.g., coffee, tea, cocoa, oranges, bananas, dried beans, dried fruits, whole grain breads, meat, eggs)
• Potassium-sparing diuretics	• Cardiac arrest	Administer the following treatments as ordered:
• Trimethoprim (antibiotic)	<i>Neuromuscular</i>	Sodium polystyrene (enema, oral, NG)
• Nonsteroidal anti-inflammatory drugs (NSAIDs) (with CKD)	• Muscle weakness	IV glucose and insulin
• Angiotensin-converting enzyme inhibitors (ACEIs) (inhibit aldosterone secretion)	• Parasthesias	Calcium gluconate
• Adrenal insufficiency (Addison's disease)	• Paralysis	Sodium bicarbonate
• Excessive oral or parenteral intake	<i>Gastrointestinal</i>	
<i>Intracellular shifts</i>	• Diarrhea	
• Acidosis	• Intestinal colic	
• Burns	• Serum K ⁺ > 5.0 mEq/L	
• Crush injuries	• Acidosis common	
• Catabolic states		
• Chemolysis of malignant cells		
• Beta blockers		

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 91–108), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

- Cardiac conduction and automaticity
- Blood clotting
- Hormone secretion.

Total calcium levels reflect both the ionized and non-ionized calcium in the blood. As long as pH and albumin levels are normal, total calcium is a reliable marker of active (ionized) calcium levels. However, when the albumin is abnormal, the total calcium must be mathematically “corrected” (see formula below). Changes in the blood pH also affect calcium levels. Alkalosis (increased pH) will increase the amount of calcium that is bound to protein, and acidosis will decrease protein binding. Ionized calcium, which is the physiologically active form, also can be directly measured (normal: 4.6–5.1 mg/dL).

Corrected Calcium

$$\text{Normal albumin (4)} - \text{Patient albumin} \times 0.8 + \text{Ca}$$

Regulation of calcium is controlled primarily via the action of parathyroid hormone (PTH), calcitonin, and calcitriol, which is an active metabolite of vitamin D. Table 8–7 summarizes the effect that each has on calcium regulation. Calcium enters the body through intestinal absorption. About 30%–50% of ingested calcium is absorbed under the influence of vitamin D.

Hypocalcemia (see Table 8–8) may occur due to low albumin levels, but this generally does not affect the ionized or active calcium level. However, a patient with an alkalemic blood pH may have a low ionized calcium (more bound to protein) and show signs of hypocalcemia, despite the total serum calcium level being in the normal range. Abnormally low calcium levels may also be seen with the following:

- Parathyroid or thyroidectomy
- Radical neck surgery for cancer
- Acute pancreatitis
- Elevated serum phosphate (hyperphosphatemia)
- Low magnesium level (hypomagnesemia), which inhibits PTH secretion
- Vitamin D deficiency/inadequate sunlight
- Malabsorption syndromes
- Citrate from rapid blood transfusions
- Alcoholism
- Renal failure.

Ninety percent of hypercalcemia (Table 8–9) is attributed to primary hyperparathyroidism or malignancy. The remainder of cases is usually due to the following:

- Thiazide diuretics
- Immobilization
- Lithium use
- Vitamin D or A overdose
- Renal transplantation (due to parathyroid hyperplasia).

Phosphorus (PO₄⁻)

Phosphorus is regulated by PTH and has an inverse relationship with calcium. Phosphorus is an abundant intracellular anion and is found in all tissues of the body. Forty-five percent of

Table 8–7. Effects of Parathyroid Hormone, Calcitonin, and Calcitriol on Calcium Regulation

Parathyroid Hormone (PTH)	Calcitonin	Calcitriol (1,25-dihydroxyvitamin D3)
Promotes transfer from bone to plasma	Antagonizes PTH	Promotes intestinal absorption
Increases intestinal absorption	Released when calcium levels are high	Enhances bone resorption
Increases renal reabsorption	Decreases calcium release from bone	Stimulates renal reabsorption

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 112–127), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Table 8-8. Hypocalcemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Primary hypoparathyroidism	<i>Neuromuscular</i>	Identify patients at risk
Surgical removal of parathyroid tissue	<ul style="list-style-type: none"> • Circumoral or peripheral numbness or tingling 	Monitor airway
Acute pancreatitis	<ul style="list-style-type: none"> • Muscle cramps 	Take safety precautions with confusion
Malabsorption	<ul style="list-style-type: none"> • Carpopedal spasm 	Take seizure precautions when severe
Alkalemic states	<ul style="list-style-type: none"> • Tetany or neuromuscular irritability 	Educate patients about reducing risk of osteoporosis:
Hyperphosphatemia	<ul style="list-style-type: none"> • Laryngeal stridor 	<ul style="list-style-type: none"> • Adequate calcium and vitamin D intake
Hypomagnesemia	<ul style="list-style-type: none"> • Hyperactive deep tendon reflexes 	<ul style="list-style-type: none"> • Regular weight-bearing exercise
Excessive transfusion of citrated blood	<ul style="list-style-type: none"> • Chvostek's sign • Trousseau's sign 	<ul style="list-style-type: none"> • Smoking cessation
Sepsis	<i>Cardiac</i>	Calcium chloride should be diluted and given through a central vein if possible due to risk for venous sclerosis or soft-tissue damage with extravasation
Hypoalbuminemia	<ul style="list-style-type: none"> • Decreased ventricular contractility • Prolonged QT interval • Arrhythmias 	
	<i>Central nervous system</i>	
	<ul style="list-style-type: none"> • Altered mental status • Depression/psychosis 	
	Total serum Ca ⁺⁺ <8.9 mg/dL	
	Ionized Ca ⁺⁺ <4.6 mg/dL	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 112–127), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

phosphorous circulating in the blood is either complexed or bound to protein, and the other 55% is ionized or in the active form. Phosphate is found in most foods, including red meat, fish, chicken, legumes, eggs, and milk products. It is efficiently absorbed in the jejunum in the absence of malabsorption disorders or antacids that block absorption. The kidneys are the primary route for phosphorus excretion, thus playing an important role in regulation.

Phosphorus serves many functions. One is the formation of adenosine triphosphate (ATP), the major source for cellular energy facilitating muscle contraction, transmission of nerve impulses, and transport of electrolytes. Phosphorus also is important for the following:

- Intracellular messages
- Muscle function
- Red blood cell function
- Metabolism of protein, carbohydrate, and fat.

Low serum phosphorus levels may reflect a true body deficit or may be due to shifting of phosphorus into the cells. Measurement of urinary phosphorus excretion can help differentiate these two states. In cases of a total body deficit, urinary excretion will drop to less than 50–100 mg/day (Pemberton & Pemberton, 1994).

Table 8–9. Hypercalcemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Hyperparathyroidism	<i>Neuromuscular</i>	Identify patients at risk
Malignant disease	• Muscle weakness	Increase mobilization
Drugs	• Decreased deep tendon reflexes	Encourage oral fluids
• Thiazide diuretics	• Muscle hypotonicity	Consider restriction of high-calcium foods
• Lithium	<i>Gastrointestinal</i>	Take safety precautions with confusion
• Excessive calcium	• Nausea, vomiting	Monitor for digoxin intoxication if on medication
• Excessive vitamin D or A	• Anorexia	Note medications that might cause hypercalcemia
• Excessive calcium-containing antacids	• Constipation	Administer bisphosphonates as directed
• Theophylline	<i>Central nervous system</i>	Administer phosphates if low
• Prolonged immobilization	• Confusion	
• Renal disease	• Lethargy	
	• Depression	
	• Psychosis	
	• Stupor, coma	
	Total serum Ca ⁺⁺ >10.3 mg/dL	
	Ionized Ca ⁺⁺ >5.1 mg/dL	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 112–127), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

The causes of hypophosphatemia are listed in Table 8–10, but nutritional recovery syndrome deserves further explanation. Debilitated older adults are at particular risk, as are persons with anorexia nervosa and alcoholics. Malnourished patients are in a catabolic state, causing depletion of intracellular phosphorus stores. Despite this, serum levels remain normal. Administration of a large glucose load, usually as total parenteral nutrition (TPN), causes the pancreas to release insulin, moving glucose and phosphorus into the cells. If replacement phosphates are insufficient, this situation leads to severe phosphate depletion.

Hyperphosphatemia (Table 8–11) may be the result of decreased renal phosphate excretion, increased intake or absorption, or a shift of phosphorus out of the cells into the ECF. Renal excretion of phosphates is dependent on the glomerular filtration rate and will decrease in acute and chronic renal failure. Cases of intoxication from phosphosoda enemas have been documented (Fass, Do, & Hixson, 1993; Korzets, Dicker, Chaimoff, & Zevin, 1992). Shifts in phosphorus to the ECF may be seen in any condition that causes muscle or tissue breakdown, such as sepsis, burns, or rhabdomyolysis. Tumor lysis that results from administration of chemotherapy can cause large shifts in phosphorus.

Magnesium (Mg⁺⁺)

The majority of magnesium is located in bones (two-thirds) and inside the cells (one-third). Only 1% is in the ECF space, and only 0.3% is in the serum. Of the serum magnesium, two-thirds is ionized (active form) and one-third is bound to proteins. This distribution (very small amount in the serum) makes testing magnesium levels problematic. A serum test represents only a very small portion of the body's total stores of magnesium. Elevated levels are good predictors

Table 8–10. Hypophosphatemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Glucose/insulin administration	<i>Neuromuscular</i>	Identify patients at risk
Re-feeding after starvation	• Muscle pain/tenderness	• Malnourished on
Hyperalimentation	• Muscle weakness	triphosphopyridine
Respiratory alkalosis	• Parasthesias	nucleotide
Alcohol withdrawal	<i>Cardiac</i>	• Alcoholics
Phosphate-binding antacids	• Decreased contractility	• Diabetic ketoacidosis
	<i>Central nervous system</i>	Monitor for signs of
	• Altered mental status	hypocalcemia while replacing
	• Seizures	phosphorus
	• Respiratory failure	
	Serum PO ₄ <2.5 mg/dL	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 146–153), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Table 8–11. Hyperphosphatemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Renal failure	<i>Signs of hypocalcemia</i>	Identify patients at risk
Chemotherapy	• Tetany	Observe for signs of
Overdose of supplementation	• Fingertip and circumoral	hypocalcemia
Excessive Fleet's phosphosoda	parasthesias	Use phosphate-containing
Large vitamin D intake	• Muscle pain/spasm	enemas and laxatives
	<i>Long-term precipitation of</i>	cautiously
	<i>phosphate</i>	
	• Skin	
	• Cornea	
	• Kidney	
	• Heart	
	• Arteries	
	Serum PO ₄ >4.5 mg/dL	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 146–153), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

of magnesium excess, but a normal level does not guarantee the lack of a total body deficit. Tables 8–12 and 8–13 outline the causes, assessments, and interventions of low and elevated magnesium levels.

Magnesium stores decrease about 15% between ages 30 and 80. The balance of magnesium depends on dietary intake and renal excretion. Magnesium is absorbed in the jejunum and ileum and is found in green vegetables, seafood, nuts, and grains. The kidneys are very efficient

Table 8-12. Hypomagnesemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Chronic alcoholism	<i>Neuromuscular</i>	Identify patients at risk
Re-feeding after starvation	• Parasthesias	Take safety precautions for confusion and seizures
Triphosphopyridine nucleotide without magnesium supplementation	• Muscle cramps/twitching	Monitor swallowing (can cause dysphagia)
Diarrhea/laxative abuse	• Chvostek's sign	Encourage increased dietary intake
Nasogastric suction/vomiting	• Trousseau's sign	Ensure complete detailed orders for magnesium replacement as various concentrations exist
Malabsorption	<i>Cardiac</i>	Monitor deep tendon reflexes (knee jerks) during magnesium administration and hold infusion if absent
Drugs increasing renal wasting	• Increased digoxin sensitivity	
• Loop and thiazide diuretics	• Hypertension	
• Aminoglycosides	• Arrhythmias	
• Amphotericin B	• Coronary artery spasm	
• Cisplatin	<i>Central nervous system</i>	
• Cyclosporine	• Altered mental status	
Drugs causing intracellular shifts	• Depression/psychosis	
• Glucose	• Seizures	
• Insulin	<i>Metabolic</i>	
• Catecholamines	• Low potassium	
Uncontrolled diabetes mellitus	• Low calcium	
Citrated blood products	• Low phosphorus	
	• Insulin resistance	
	Serum Mg ⁺⁺ <1.3 mEq/L	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 131–142), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Table 8-13. Hypermagnesemia: Causes, Assessments, and Interventions

Causes	Assessments	Interventions
Renal failure	Peripheral vasodilation/flushing	Identify patients at risk
Overdose of supplementation	Nausea/vomiting	Observe for assessment signs
Adrenal insufficiency	Hypotension	Avoid magnesium-containing medications in patients with renal insufficiency
Excessive magnesium-containing antacids or laxatives	Bradycardia	
	Decreased deep tendon reflexes	
	Respiratory depression	
	Coma	
	Cardiac arrest	
	Serum Mg ⁺⁺ >2.1 mEq/L	

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 131–142), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

at conserving magnesium or excreting excess amounts as needed. Magnesium is closely coupled with calcium and phosphorus, as well as potassium.

Several factors increase the risk of elevated magnesium levels in older adults, including

- Age-related decline in renal function
- Increased consumption of magnesium-containing antacids or mineral supplements
- Possible increased absorption due to altered gastrointestinal mucosa (Clark & Brown, 1992).

Magnesium plays a role in

- ATP production and utilization
- Neuromuscular control
- Neuronal control
- Cardiovascular tone
- More than 300 enzymatic reactions.

ACID-BASE BALANCE

Disturbances of acid–base balance can be classified as either acidosis or alkalosis, with the primary disorder being either respiratory or metabolic. This section reviews acid–base regulation and the method for classifying these derangements.

Regulation of Acid–Base Balance

The body maintains the internal pH within a very narrow range, between 7.35 and 7.45. This balance is maintained via various buffering systems. A buffer is able to rapidly take up or release a hydrogen (H^+) ion to change the pH of the blood. An increase in hydrogen ions reduces the pH (*acidosis*) and a decrease increases the pH (*alkalosis*). Most buffering is provided by the kidneys (*metabolic*) and the lungs (*respiratory*). In addition, several less important buffering systems are at work in the ECF and ICF:

- Organic and inorganic phosphates
- Plasma proteins
- Red blood cells
- Hemoglobin.

The lungs eliminate “acid” by blowing off CO_2 or can compensate for a metabolic alkalosis by retaining more CO_2 . The kidneys either eliminate or retain bicarbonate ions (base), depending on the blood pH. The carbonic anhydrase equation describes the transport:

In the lungs carbonic acid dissociates into CO_2 (exhaled) and water:



In the kidneys, carbonic acid can dissociate into bicarbonate ions (either reabsorbed or eliminated) and hydrogen ions:



Lungs

With normal lung function, the respiratory center in the brain responds to the arterial pressure of carbon dioxide ($PaCO_2$) to increase the rate and depth of breathing (ventilation). In persons

with chronic CO_2 elevations, the drive to breathe is stimulated by a fall in the arterial pressure of oxygen (PaO_2).

The lungs provide rapid compensation for acid–base disturbances, usually responding within minutes to hours. Alterations in the rate and depth of ventilation influence the amount of CO_2 that is eliminated. For example, in the case of diabetic ketoacidosis (metabolic acidosis), the rate and depth of ventilation are increased, leading to elimination of maximal CO_2 to increase the pH.

Kidneys

The kidneys compensate by eliminating or retaining bicarbonate ions (HCO_3) and hydrogen ions (H^+). This compensation is slower than compensation by the lungs and takes hours to days. Therefore, the kidneys will not compensate for acute respiratory disturbances, but will compensate for chronic conditions. For example, a patient with chronic obstructive pulmonary disease will develop a respiratory acidosis due to chronic CO_2 retention. To compensate, the kidneys will eliminate H^+ ions and retain HCO_3 to maintain the blood pH within the normal range.

Electrolytes

Conditions of alkalosis are generally associated with hypokalemia. The release of H^+ into the ECF causes the movement of potassium from the ECF into the ICF (to maintain electroneutrality), resulting in low levels of potassium in the ECF. This alteration is most pronounced with metabolic alkalosis and less so with respiratory alkalosis.

In contrast, acidosis leads to hyperkalemia. In this scenario, H^+ ions move into the ICF to raise the plasma pH, in exchange for a potassium ion moving into the ECF. This movement of potassium leads to a relative hyperkalemia. Again, this shift is much more pronounced with metabolic acidosis than respiratory acidosis.

Arterial Blood Gas Interpretation

Arterial blood gas samples are used to determine acid–base balance. Table 8–14 lists the components and significance of the information reported from an arterial blood gas sample.

CO_2 reported on the chemistry panel (venous blood) reflects primarily the bicarbonate level and is therefore a marker of metabolic status. This can be confusing, as the PaCO_2 in the arterial blood reflects respiratory acid.

Table 8–14. Arterial Blood Gas Interpretation for Acid–Base Balance

Test	Normal Value	Significance of Change
pH	7.35–7.45	Low = acidosis High = alkalosis
PaCO_2	35–45 mmHg	Low = respiratory alkalosis High = respiratory acidosis
HCO_3	21–28 mmol/L	Low = metabolic acidosis High = metabolic alkalosis
PaO_2	35–45 mmHg	Low = impaired gas exchange
O_2 saturation	95%–100%	Low = impaired gas exchange

The four major acid–base derangements and their causes are listed in Table 8–15.

To determine the acid–base disturbance for a given blood gas, one must follow these steps (see Table 8–16):

- Identify if the derangement is an acidosis (low pH) or alkalosis (high pH).
- Examine the CO_2 to determine if this value explains the abnormal pH. If yes, then the problem is respiratory in origin.
- Examine the HCO_3 to determine if this value explains the abnormal pH. If yes, then the problem is metabolic in origin.

Examples include those found in Tables 8–17, 8–18, 8–19, and 8–20.

Table 8–15. Acid–Base Derangements and Causes

<p><i>Metabolic Acidosis</i></p> <ul style="list-style-type: none"> • High anion gap • Diabetic ketoacidosis • Lactic acidosis • Toxic ingestion (e.g., aspirin, methanol) • Renal failure • Normal or low anion gap • Diarrhea • Excess chloride 	<p><i>Metabolic Alkalosis</i></p> <ul style="list-style-type: none"> • Vomiting • Gastric suction • Excessive alkali ingestion • Diuretics • Hypokalemia • Hypoaldosteronism
<p><i>Respiratory Acidosis</i></p> <ul style="list-style-type: none"> • Respiratory depression or hypoventilation • Chronic lung disease 	<p><i>Respiratory Alkalosis</i></p> <ul style="list-style-type: none"> • Hyperventilation due to any cause • Anxiety • Hyperthermia • Thyrotoxicosis • Excessive mechanical ventilation • Pregnancy • Sepsis • Early salicylate intoxication

Adapted from *Fluid and electrolyte balance: Nursing considerations* (pp. 158–172), by N. M. Metheny, 2000, Philadelphia: Lippincott Williams & Wilkins.

Table 8–16. Classification of Acid–Base Imbalances

	pH	CO_2	HCO_3
Metabolic acidosis	↓	Normal	↓
Respiratory acidosis	↓	↑	Normal
Metabolic alkalosis	↑	Normal	↑
Respiratory alkalosis	↑	↓	Normal

Table 8-17. Case 1: Metabolic Acidosis

pH	7.31
CO ₂	30
HCO ₃	18

Table 8-18. Case 2: Respiratory Acidosis

pH	7.32
CO ₂	49
HCO ₃	26

Table 8-19. Case 3: Metabolic Alkalosis

pH	7.47
CO ₂	44
HCO ₃	32

Table 8-20. Case 4: Respiratory Alkalosis

pH	7.48
CO ₂	30
HCO ₃	26

Compensation

As discussed above, the lungs and kidneys will attempt to compensate for acid–base imbalances caused by the other system. The lungs act rapidly to compensate, and the kidneys take longer. Successful compensation will bring the pH back to normal range (7.35–7.45).

The steps for interpreting compensated blood gases are the same, except the pH (Step 1) will be within normal range. In this case, consider 7.40 to be the “normal” level for pH. Any value higher represents a primary alkalosis, and a pH below 7.40 represents a primary acidosis. Then follow Steps 2 and 3 to determine if the primary derangement is a respiratory or metabolic disorder. Table 8–21 illustrates. Because the pH is in normal range, this blood gas would be identified as *compensated*.

It also is possible to have a mixed acid–base disorder. For example, a patient who has lactic acidosis and COPD might have the disorder found in Table 8–22.

Table 8-21. Case 5: Compensated Respiratory Acidosis

pH	7.36
CO ₂	52
HCO ₃	33

Table 8-22. Case 6: Mixed Respiratory and Metabolic Acidosis

pH	7.12
CO ₂	55
HCO ₃	14

SUMMARY

This chapter reviewed the basic tenets of nutrition, fluid, and electrolyte and acid-base balance. Adequate nutritional screening and assessment are needed to prevent the complications of under- or over-nutrition. Derangements of fluid, electrolytes, and acid-base balance are common in older adults and can have lethal consequences. Nurses should remain vigilant and seek to prevent imbalances, but when these do occur, it is vital that the nurse promptly identify and seek treatment for the disorder.

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INTERNET RESOURCES

- Food pyramid and meal planning: www.mypyramid.gov
- Nutrition Needs for the Older Adult: http://fcs.tamu.edu/food_and_nutrition/
- Nutrition Screening Tool: http://www.healthyearkansas.com/healthy_aging/pdf/nutrition_screening.pdf