Critically ill patients are diverse in terms of illness, but many experience electrolyte abnormalities or fluid imbalances that can compromise their clinical status and adversely affect outcomes. These shifts in electrolytes and fluids—the “critical care shuffle”—can be attributed to an underlying chronic disease state, an acute condition that manifests during the course of the patient’s hospitalization, or the administration of certain medications. Monitoring and carefully managing electrolytes and fluid balance is an integral part of assessing and caring for a critically ill patient. This series provides a general overview of the electrolytes tested and I.V. fluids used in critical care areas, as well as the common causes, signs and symptoms, and available treatments to correct electrolyte abnormalities and fluid imbalances. A later article will describe imbalances in potassium, calcium, and magnesium.

The balancing act
Fluid and electrolyte balance play an important role in homeostasis, and critical care nurses assume a vital role in identifying and treating the physiologic stressors experienced by critically ill patients that disrupt homeostasis.1

Electrolytes, found in body fluids, are electrically charged particles (ions). Cations are positively charged ions; anions are negatively charged ions. Electrolytes play a crucial role in transmitting impulses for proper heart, nerve, and muscle function. The number of positively and negatively charged ions should be equal; when this balance is upset, electrolyte abnormalities can occur. Electrolytes can further be classified as extracellular (EC, outside the cell) or intracellular (IC, inside the cell). Sodium is the most abundant EC electrolyte; potassium is the most abundant IC electrolyte.

Just as too little or too much of any one electrolyte can become a problem in maintaining a critically ill patient’s stability, imbalances in fluid homeostasis can also present unique challenges for both you and your patient.

Fluids are in constant motion in the body. Total body water (TBW) normally accounts for about 60% of an adult’s body weight. Forty percent of TBW is in the IC space, and EC water accounts for 20% of body weight: 14% in the interstitial space,
and 5% in the intravascular space. Transcellular fluid (cerebral spinal fluid and fluid contained in other body spaces such as joint spaces, and the pleural, peritoneal, and pericardial spaces) make up about 1% of total body weight. See How body water is distributed.

To maintain homeostasis, fluids need to be stable in the intravascular, interstitial, and IC spaces. The amount of IC fluid is rather stable in the body; intravascular fluid is the least stable and fluctuates in response to fluid intake and loss. Interstitial fluid is the reserve fluid, replacing fluid in the intravascular and IC spaces as needed.1,3

Almost all pathologies affect the fluid balance within the body, especially in critically ill patients. Fluid movement within the various body spaces depends on osmosis—movement of water through a selectively permeable or semipermeable membrane from a solution that has a lower solute concentration to one with a higher solute concentration—and diffusion, or the free movement of molecules or other particles in solution across a permeable membrane from an area of higher concentration to an area of lower concentration, resulting in an even distribution of the particles in fluid. Fluid balance also is regulated by certain hormones:

• Aldosterone, the principal mineralcorticoid produced by the adrenal cortex, promotes sodium retention by the distal tubules, while increasing urinary losses of potassium. This helps to prevent water and sodium losses through the kidneys.

• Antidiuretic hormone (ADH), also known as vasopressin, is synthesized in the hypothalamus and stored in and released by the posterior pituitary gland. ADH triggers the renal tubules to reabsorb water and return it to the intravascular space.

• Natriuretic peptides, such as atrial natriuretic peptide, released from the heart in response to cardiac chamber stretch and overfilling, increase sodium and water excretion by the renal distal and collecting tubules.2

Because the kidneys are the major organs involved in electrolyte and fluid homeostasis, determine the patient’s renal function before attempting to correct a patient’s electrolyte or fluid imbalance.4 Also, don’t let lab numbers or the numerous equations used for fluid replacement override sound clinical judgment. If the patient’s clinical condition doesn’t support the numbers, perform a follow-up test—an error could have occurred in the lab, blood draw, or be the result of blood sample hemolysis.

Now let’s look more closely at sodium and fluid balance.

**Sodium: Water follows**

Sodium is the key EC cation and plays a major role in serum osmolality. When serum sodium levels change, so does serum osmolality and water movement. Normally, serum osmolality is 280 to 300 mOsm/kg; the value is considered critically abnormal if it’s 240 mOsm/kg or lower, or 320 mOsm/kg or higher.5 Sodium also is a primary contributor to nerve impulse transmission and muscle contraction. If you suspect that your critically ill patient has a sodium imbalance, think of water, and specifically, disorders of water balance. Remember the saying, “where sodium goes, water is sure to follow.” In the simplest of terms, sodium and water balance are tightly linked.

**How body water is distributed**

The graphic shows the approximate size of body compartments in a 154.3-pound (70 kg) adult.
Hyponatremia
In hyponatremia, water moves from an area of low intravascular sodium concentration to an area of high sodium concentration. The result is excess fluid volume in the IC compartment and a fluid volume deficit in the EC compartment.

Signs and symptoms of hyponatremia vary depending on the speed of onset, magnitude of sodium deficit, and cause. When serum sodium is less than 135 mEq/L (hyponatremia), signs and symptoms generally reflect hypoosmolality and movement of water into muscle, neural, and gastrointestinal (GI) tissue resulting in muscle cramps, muscle weakness, decreased deep tendon reflexes, headache, mental status changes, dizziness, nausea, vomiting, abdominal cramps, and diarrhea. Serum and urine osmolality will be decreased. In severe hyponatremia (serum sodium of 115 mEq/L or less), you may notice muscle twitching, focal weakness, papilledema, and signs of increasing intracranial pressure, such as lethargy, confusion, hemiparesis, and seizures. Without treatment, increased intracranial pressure can be fatal.

Hyponatremia is often caused by or associated with treatments and diagnoses common to patients in CCUs. For instance, administering hypotonic I.V. solutions and some medications (such as thiazide diuretics, nonsteroidal anti-inflammatory drugs, and selective serotonin reuptake inhibitors) may contribute to the development of hyponatremia. Hyponatremia also is associated with heart, liver, and kidney failure, which are common among critically ill patients.

When managing a patient with hyponatremia, the goal is to identify and treat the underlying cause of the sodium imbalance. Treatment will also depend on whether the patient has a fluid balance abnormality. Depending on the underlying cause, you may administer diuretics, restrict water intake, or administer hypertonic saline (for example, 3% sodium chloride solution, although this fluid should only be used in severe cases of symptomatic hyponatremia because it can rapidly induce fluid volume overload). We’ll discuss the use of crystalloid solutions later.

Hypernatremia
In hypernatremia, sodium pulls water from the IC space into the intravascular space. The dehydrated cells shrink or shrivel, and the EC space becomes fluid overloaded. Signs and symptoms of hypernatremia include thirst (a compensatory mechanism that’s troublesome in critically ill patients, who often are fluid-restricted or physically unable to drink); signs and symptoms related to hyperosmolality and movement of water out of neural tissue, including irritability, restlessness, headache, disorientation, and decreased deep tendon reflexes; signs and symptoms related to decreased IC fluid including dry skin and mucous membranes, decreased skin turgor, and decreased salivation and lacrimation.

The patient’s urine specific gravity and osmolality increase, and central venous pressure decreases. In severe hypernatremia (serum sodium of more than 160 mEq/L), the patient may develop seizure activity and coma.

Hypernatremia in critically ill patients can be caused by vomiting, diarrhea, open wounds, hyperventilation, fever, hypertonic enteral tube feedings without water supplementation, nasogastric suctioning, GI drains, excessive administration of sodium-containing fluids such as 3% sodium chloride solution and sodium bicarbonate, and medical conditions such as diabetes insipidus and Cushing syndrome.

As with treating hyponatremia, you’ll implement measures to restore fluid balance in addition to correcting the underlying cause of the sodium imbalance. Treatment measures will depend on whether the patient is hypovolemic, isovolemic (also called euvoletic), or hypervolemic. Most critically ill patients with hypernatremia are also hypovolemic, so you’d administer isotonic or hypotonic sodium chloride solution as prescribed. Crystalloid solutions are discussed later.
Fluid balance: Dry or wet?

Fluid imbalances are common in critically ill patients. Fluid and electrolyte balance are closely related in the sense that electrolyte imbalances can be avoided when you pay close attention to the patient’s nutritional status and the use of I.V. fluids. Causes of fluid volume deficit in the critically ill patient can include GI loss, infection, renal loss, and third-space fluid shifts. In contrast, causes of fluid volume excess are overadministration of fluids, heart failure, renal failure, and certain medications.

Dry: Dehydration

Dehydration, or fluid volume deficit, is characterized by a decrease in EC fluid and occurs when fluid intake is less than the body’s fluid needs, when patients experience excessive loss of body fluids, or when third-spacing sequesters EC fluid where it can’t contribute to cardiac output. Isotonic fluid deficit is the most common type of fluid volume deficit, in which there are proportionate losses in sodium and other electrolytes and water. It’s almost always caused by a loss of body fluids and is often accompanied by a decrease in fluid intake. Without compensatory mechanisms, perfusion to vital organs may be decreased, so fluid replacement is vital.

Clinical manifestations of fluid volume deficit can develop rapidly. If you suspect that your patient is at risk for developing hypovolemia, assess for acute weight loss, increased thirst, decreased skin turgor, dry mucous membranes, oliguria, high urine specific gravity, weak and rapid pulse, flattened neck veins, increased temperature, decreased central venous pressure, muscle weakness, postural hypotension, and cool, clammy pale skin related to peripheral vasoconstriction. Being familiar with these clinical findings lets you quickly identify and intervene for patients and avoid potential medical emergencies.

Treatment of fluid volume deficit includes prompt identification and treatment of the underlying cause and fluid replacement.

A critically ill patient can be dehydrated while appearing fluid overloaded.

Most critically ill patients will need I.V. fluid replacement. Generally, replace fluid based on the type of fluid lost: blood transfusions for blood loss, isotonic solutions for patients experiencing diarrhea.

Other I.V. fluids that are available for the treatment of hypovolemia are colloids, which are high-molecular-weight substances that under normal conditions don’t pass through the semipermeable membrane in the vascular space. Colloid solutions are hypertonic, increasing capillary oncotic pressure and pulling fluid from the interstitial space into the vascular compartment. Medications may be indicated to treat the causes of dehydration, such as antidiarrheals, antiemetics, and antipyretics.

Third-spacing is another way a critically ill patient can be dehydrated while appearing fluid overloaded. Fluid that accumulates in these spaces is physiologically useless because it’s not available for use as reserve fluid or function. Causes of third-spacing include:

- Injury or inflammation such as burns, sepsis, crush injuries, massive trauma, cancer, intestinal obstruction, and abdominal surgery. These all increase capillary permeability and let fluid, electrolytes, and proteins leak from the intravascular space.
- High vascular hydrostatic pressure from renal failure or heart failure, which pushes abnormal volumes of fluid from blood vessels.
- Malnutrition and liver failure from cirrhosis, chronic alcohol abuse, or starvation. These conditions prevent the liver from producing albumin, thus lowering capillary oncotic pressure.

Treating third-spacing can be challenging. Osmotic diuretics can be used to mobilize some of the fluid back into the intravascular space for elimination by the renal system. However, this is usually only a temporary measure because of the nature of the disease processes that caused the third-spacing. Large third space fluid collections can be physically removed (by paracentesis for ascites or thoracentesis for pleural effusion), and then the vascular space can be rehydrated with I.V. fluids.
Wet: Overhydration
Fluid overload or fluid volume excess is a clinical sign of fluid retention or intake that exceeds the body’s fluid needs. Hypervolemia refers to increased fluid in the intravascular and interstitial spaces. A critically ill patient with a severe fluid overload or poor cardiac function is at risk for acute pulmonary edema,12

Signs and symptoms of fluid volume excess include acute weight gain, full and bounding pulses, distended neck veins, pulmonary crackles, peripheral edema, and elevated central venous pressure. Volume-overloaded patients can develop flash (or acute) pulmonary edema, a life-threatening complication that requires swift intervention to prevent respiratory arrest.

Pharmacotherapy for the overhydrated critically ill patient consists of diuretics such as furosemide, bumetanide, or torsemide. These diuretics can also cause electrolyte losses, so frequently monitor the patient’s serum electrolyte levels and ECG.

Because of dependent edema, patients are at risk for developing skin breakdown, such as in the sacral area. Turn and reposition the patient at least every 2 hours to decrease the risk of skin breakdown.

Nutritional interventions include limiting sodium and fluid intake. For example, at discharge, the patient and family may need to be taught how to read food labels in order to limit dietary sodium intake. The patient also may need long-term fluid management.

To determine whether therapy has been effective, monitor the patient’s intake and output and daily weight. Patients should be weighed at the same time every day; a rapid weight gain is the cardinal sign of fluid retention and overload.

Staying in balance
As you’ve learned, the levels of sodium and body water can shift up or down. Assessing the volume status of the critically ill patient is notoriously more challenging to detect than electrolyte abnormalities, which can be measured directly.10 The next article in this series will focus on managing the remaining electrolytes that are part of the “critical care shuffle.”

REFERENCES

RESOURCES

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