Numerous conditions—metabolic, infective, traumatic, and iatrogenic—can cause fluid depletion. In such cases, initiating intravenous (IV) fluid replacement is commonplace. In fact, IV fluid replacement therapy is one of the most common invasive procedures hospitalized patients undergo, and it’s performed in certain outpatient and home care settings as well.

Fluid loss can put patients at substantial risk for fluid and electrolyte imbalances, which can lead to shock and multiple organ failure. Although IV therapy for fluid depletion is practiced widely, if not administered scrupulously, it actually can exacerbate the fluid and electrolyte imbalance. It’s essential, therefore, that the practitioner responsible for initiating and maintaining fluid replacement therapy understand the basic mechanisms supporting fluid balance, the consequences of fluid loss, and the rationale for fluid replacement.

In this article, I’ll review the basics of fluid balance and the etiology of fluid loss. I’ll discuss how to assess fluid depletion, outline the principles of fluid replacement therapy, and explain the context in which various types of solutions are administered. I will not, however, cover the treatment of diabetes mellitus and diabetes insipidus, which follow different principles that are beyond the scope of this article.

**FLUID MECHANICS**

Body water represents approximately 60% of a person’s total weight. For a 70 kg man, this amounts to about 42 L. Intracellular fluid accounts for two thirds of it (roughly 28 L) and extracellular fluid for the other third (about 14 L).

Extracellular fluid includes both the interstitial fluid (around 11.2 L) and plasma (the remaining 2.8 L). In addition to plasma, intravascular fluid contains the fluid volume of its other major constituent, the red blood cells.

The fluid component of red blood cells represents about 2 L of intracellular fluid. A person’s total blood fluid volume, therefore, equals about 5 L—plasma plus the fluid component of red blood cells.

The body loses fluid through such normal physiologic functions as breathing and urination. But when certain diseases or environmental conditions substantially increase fluid loss, the body may be unable to maintain homeostasis, and fluid replacement may be necessary.

**NORMAL FLUID LOSS**

Normal fluid loss includes both insensible and sensible losses. Each day the skin loses approximately 300 mL and the lungs lose approximately 700 mL of water from evaporation. This insensible water loss remains mostly stable at roughly 1 L/day and is responsible for negligible electrolyte loss. Hyperventilation intensifies losses from the lungs, and fever increases losses from both skin and lungs. The amounts of fluid lost through these two routes increases by 10% for every degree of fever above 37° C.\(^1\)

Sensible water losses occur mainly through urination, but also through perspiration and defecation. The average adult voids between 1 and 1.5 L urine daily. Urine sodium and potassium generally range in amounts from 40 to 80 mEq/L. Perspiration contains

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sodium in amounts ranging from 34 mEq/L to 50 mEq/L. Normal sweating, however, accounts for a daily fluid loss of only 100 mL. Profuse sweating, as occurs with strenuous exercise, can raise this amount to 1,500 mL, making both fluid and salt loss significant. In the absence of diarrhea, fluid lost in stool ranges from 100 to 200 mL in adults.

In total, therefore, basic adult fluid losses equal approximately 2.5 L daily. These losses should be taken into account when considering fluid replacement.

**ABNORMAL FLUID LOSS**

Excessive fluid loss can mean either dehydration (the loss of simple water or of more water than salt) or hypovolemia (the loss of isotonic saltwater). These two types of water loss affect the body in different ways.

Simple water losses may be brought on by water deprivation or by an increase in body or environmental temperature. Hyperglycemia, diabetes insipidus, and nasogastric overfeeding (especially if not balanced with adequate water intake) are other possible causes of simple water loss.

The loss of simple water increases the solute content of the blood, which results in intravascular hypertonicity. This causes an osmotic shift of water from within the cells into the plasma. There is, thus, a loss of fluid from both the extracellular and intracellular compartments.

Saltwater may be lost with gastrointestinal (GI) losses, sweating, hypoalectadonism, diuretic use, and third spacing (the leakage of saltwater fluid into a third space that’s outside of the extracellular and intracellular fluid compart-
ments, where it’s unavailable to support the circulation). Third spacing can occur with ascites, bowel obstruction, pancreatitis, and tissue injury (from trauma, infection, or burns) that causes vasodilation.

If water and salt are lost in isotonic amounts, there is no osmotic shift in fluids between the extracellular and intracellular compartments. In such cases, volume depletion occurs only in the extracellular fluid compartment.

**ASSESSING FLUID DEPLETION**

The degree of fluid depletion may be estimated by weight loss, with one pound representing 500 mL of fluid. Using this guide, weight losses of 2% (or 2.4 lb in a 120-lb person) would be considered mild fluid depletion, whereas 5% (or 6 lb in a 120-lb person) would be considered moderate and 8% (or 9.6 lb or more in a 120-lb person) would be considered severe.

Alternatively, water deficit may be calculated as follows: water loss in liters = total body water x [(serum sodium/140) – 1], where total body water in liters is said to be 60% of body weight in kilograms for lean men and 50% of body weight in kilograms for lean women. Fluid loss estimates must factor in history and physical signs (though these are minimal when depletion is mild) and replacement must be guided by continual physical assessment and laboratory findings.

History should include medications, fluid intake and output, illnesses, and any signs or symptoms of fluid loss (such as thirst, fatigue, weakness, malaise, or decreased urinary output). Keep in mind etiologies associated with simple water loss (fluid deprivation, an increase in environmental or body temperature, hyperglycemia, diabetes insipidus, or hyperalimentation) and with saltwater loss (GI tract loss, profuse sweating, hypoalectadonism, diuretic use, or third spacing).

A drop in intracellular fluid, as occurs with simple water loss, typically causes thirst and such central nervous system disturbances as restlessness, weakness, listlessness, muscular twitching, irritability, disorientation, delusions, and hallucinations. The patient may have flushed skin, fever, dry and sticky mucous membranes, oliguria, or orthostatic hypotension. Laboratory values show an elevated serum sodium level (above 145 mEq/L), increased serum osmolality (above 295 mOsm/kg), reduced urinary sodium (10 to 20 mEq/L or less), and—except in the case of diabetes insipidus—a normal urine specific gravity above 1.015. If fluid depletion is due to diabetes insipidus, urine specific gravity may be as low as 1.005.

A drop in extracellular fluid, as occurs with both simple and saltwater losses, causes thirst, fatigue, muscle cramps, weakness, and postural dizziness. Body temperature may be low and the skin is dry. Assess skin turgor by pulling up on the skin covering the sternum: Tenting that remains for several seconds indicates fluid deficit. The mucous membranes are dry and eyeball tension may be reduced (to the touch). The tongue is shrunken and coated with multiple furrows (in addition to the normal midline furrow). Typically, a diminished appetite progresses to nausea and vomiting.

If salt is lost in proportion to water, the serum sodium level may be normal, though there would be a relative increase in urea, albumen,
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red blood cells, hemoglobin, and Hct secondary to hemoconcentration. If more water than salt is lost, laboratory findings are similar to those that accompany simple water (intracellular fluid) loss.

With a 20% fluid loss or less, cardiac output remains adequate. With moderate fluid deficit cardiac output decreases. The neck veins are flat when the patient is supine (unless the patient has congestive heart failure). Capillary refill is delayed (greater than three seconds) and urinary output reduced (25 to 30 mL/hour or less). An early and accurate sign of fluid volume deficit is the presence of postural hypotension with systolic pressure dropping 15 mm Hg or more when the patient moves from a supine to sitting position. Another indication is the narrowing of pulse pressure. A plasma fluid volume deficit of 30% or more is accompanied by hypotension; cold, clammy, mottled skin; delayed capillary refill (of three to five seconds or more); and stupor or coma, as shock ensues and cardiac and cerebral perfusion is reduced.7

**INTRAVENOUS THERAPY FOR NORMAL FLUID LOSS**

If normal fluid losses are to be replaced intravenously (if oral replacement isn’t possible or is contraindicated), sodium chloride—and often potassium—may be added to the replacement fluid as indicated by urinary output and serum levels of these electrolytes. Normally, sodium and potassium excretion in urine varies because the healthy kidney can conserve or waste as needed—though random specimens tend to contain at least 40 mEq/L of both.9

Customarily, when added to replacement fluid, salt is replenished in hypotonic amounts starting at 0.45%, or 77 mEq/L. Potassium additions start at 20 mEq/L and can be increased to 40 mEq/L, which is usually sufficient to replace normal loss.

Calories also are added to replacement fluid therapy. Each 1-L solution containing 5% dextrose supplies 50 g of carbohydrate and approximately 170 calories. While even 2 or 3 L provides only a portion of the patient’s daily caloric intake, it’s useful in preventing ketosis, which occurs with starvation. A patient who isn’t eating and is receiving parenteral therapy requires supplemental protein, vitamins, and other electrolytes after one week.5 A discussion of total parenteral nutrition, however, is beyond the scope of this article.

For normal fluid loss, the ideal replacement generally consists of 5% dextrose in water with 0.45% sodium chloride and 20 mEq/L potassium delivered in amounts of 2 to 3 L/day until normal oral intake of fluids and food is resumed.9 Serum electrolytes are monitored and supplemental sodium and potassium are adjusted accordingly. For patients who require less electrolyte replacement, hypotonic salt solutions also come in 0.33% solutions, which contain 56 mEq/L sodium chloride.

Hypertonic salt solutions have no place in maintenance fluid therapy. Even isotonic saline isn’t used unless the patient’s serum sodium level falls below 130 mEq/L. Hypertonic 3% saline solution may be used if the serum sodium level falls below 115 mEq/L.8

Close serial electrolyte monitoring helps prevent overhydration and hyponatremia. Potassium in amounts greater than 40 mEq/L is irritating to small veins and, therefore, can’t be infused peripherally.

**INTRAVENOUS THERAPY FOR ABNORMAL FLUID LOSS**

For abnormal water losses that exceed the loss of salt, treatment consists of simple water administration. Often, this water replacement must be administered intravenously, as patients in such cases tend to vomit. Usually, a 5% glucose solution is used. It’s administered gradually over 48 hours. Convulsions can occur if the brain expands too quickly.

Replace no more than half the patient’s water deficit within the first 24 hours to lower the serum sodium concentration by about 0.5 mEq/L/hr.4 Of course, frequent assessment of the patient and lab values should guide all fluid replacement.

When replacing abnormal saltwater losses, a balanced salt solution, such as normal saline, can be used to replace half the deficit; after that, the patient should be reassessed. Evaluate electrolytes, intake, and output, and consider ongoing weight and fluid losses until vital signs and urinary output are normal. Serial measurement of urine sodium may be useful. If urine sodium is below 25 mEq/L in a patient with no renal disease, kidneys are sensing persistent volume depletion and more fluids should be given.9

GI disorders cause a loss of electrolyte rich fluids. Losses from the upper GI tract, above the pylorus, are isotonic and contain sodium, chloride, potassium, and hydrogen. Losses from below the pylorus are isotonic and contain sodium, potassium, and bicarbonate.10 Upper GI tract losses usually occur from vomiting or nasogastric suctioning. Diarrhea is the most common cause of lower GI content loss; others include intestinal resection and fistulas.10

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Fluid lost through vomiting and nasogastric suctioning contains 70 mEq/L of sodium chloride, 10 to 20 mEq/L of potassium, and hydrogen chloride ions.\(^3\) Losses of this type can result in alkalosis. Since 0.45% normal saline solution contains 77 mEq/L sodium chloride, an appropriate replacement fluid would contain 5% dextrose and 0.45% sodium chloride with 20 mEq/L potassium. Fluid replacement containing sodium chloride and potassium allows for renal excretion of bicarbonate and a reversal of the alkalosis. For every 1 mL fluid lost in the previous 24 hours, 1 mL of replacement fluid is administered.\(^3\)

In addition to a loss of bicarbonate, diarrhea commonly causes a potassium loss of 20 to 40 mEq/L and in severe cases, such as that associated with colitis, up to 90 mEq/L. The usual replacement fluid for this loss is lactated Ringer’s solution. Containing 130 mEq/L of sodium, 109 mEq/L chloride, 28 mEq/L lactate, 4 mEq/L potassium, and 3 mEq/L calcium, its components are similar to those of extracellular fluid. Since lactate is metabolized in the liver to bicarbonate, lactated Ringer’s can be helpful when the patient has mild acidosis.\(^3\) Electrolytes should be monitored and replaced in accordance with serum levels and established urinary output. As with upper GI tract fluid losses, for every 1 mL fluid lost from the lower GI tract over the previous 24 hours, 1 mL fluid is replaced.\(^3\)

**WHEN THE PATIENT IS IN SHOCK**

Hypovolemic shock is most often due to bleeding or third spacing, though any of the causes of volume depletion can produce a similar outcome. The type of fluid selected for replacement usually depends on the fluid lost. Patients who are bleeding require blood, which most often is infused rapidly under pressure until the patient’s Hct is 35%. In most other cases, the fluid of choice is isotonic saline or lactated Ringer’s solution. The rate of replacement is approximately 1 to 2 L in the first hour.\(^3\) Patients who don’t respond to the initial fluid resuscitative efforts should have invasive hemodynamic monitoring.

The risks of congestive heart failure and pulmonary edema can be minimized through the avoidance of an excessively elevated pulmonary capillary wedge pressure.\(^11\)

The rate of infusion should be rapid as long as the patient’s systolic blood pressure and cardiac filling pressure remain low. Colloid solutions such as albumin, plasma, or dextran, may be used instead of crystalloids, especially if plasma oncotic pressure is low (total serum protein is less than 5.4 g/dL) or if resuscitation is expected to require substantial amounts of fluid (with replacement representing more than 5% of body weight).\(^11\)

Colloids remain in the plasma and don’t cross over into the interstitial space, as do crystalloids. An improvement in mental status, urinary output, and vital signs provides evidence that tissue perfusion is satisfactory.

The ability to recognize the clinical manifestations of fluid depletion is crucial to clinicians managing a wide variety of disease states. A basic understanding of potential causes and factors influencing fluid balance will guide the practitioner in providing optimal care and achieving the best possible outcomes for patients requiring IV fluid replacement therapy.

**REFERENCES**


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Intravenous fluid therapy involves the intravenous administration of crystalloid solutions and, less commonly, colloidal solutions. The type, amount, and infusion rates of fluids are determined based on the indication for fluid therapy and specific patient needs. Crystalloid solutions are used to resuscitate patients who are hypovolemic or dehydrated, correct free water deficits, replace ongoing fluid losses, and meet the fluid requirements of patients who cannot take fluids enterally. The use of colloidal solutions is controversial and should be reserved for special situations (e.g., severe cases of low oncotic pressure). Intravenous fluid therapy has long been the mainstay of treatment of kidney disease, including acute kidney injury and uremic crisis associated with chronic kidney disease. Careful management of fluid dose is critical, as animals with kidney disease may have marked derangements in their ability to regulate fluid homeostasis and acid-base status. Fluid overload (FO) is characterized by hypervolemia, edema, or both. In clinical practice it is usually suspected when a patient shows evidence of pulmonary edema, peripheral edema, or body cavity effusion. FO may be a consequence of spontaneous disease, or may be a complication of intravenous fluid therapy. Intravenous fluid therapy is one of the most common interventions in acutely ill patients. Each day, over 20% of patients in intensive care units (ICUs) receive intravenous fluid resuscitation, and more than 30% receive fluid resuscitation during their first day in the ICU. Virtually all hospitalized patients receive intravenous fluid to maintain hydration and as diluents for drug administration. In this narrative Review, we provide an overview of current theories of human physiology relating to intravenous fluid therapy. In relation to critically ill adults, we discuss the mechanisms by which hypovolaemia and fluid overload affect the kidney and the balance of risks and harms of different strategies for fluid management, including the type and amount of fluid. Intravenous fluids (IVFs) are the most common drugs administered in the intensive care unit. Despite the ubiquitous use, IVFs are not benign and carry significant risks associated with under- or overadministration. Hypovolemia is associated with decreased organ perfusion, ischemia, and multi-organ failure. There are protocols for intravenous fluid therapy [22], but their uniform application implies giving the same volume to different patients and in different circumstances. To address the uniqueness of each patient and situation, rather than infusing a predefined amount of fluid, the goal should be to tailor fluid infusion based on a needs analysis [23,24].