Fluid balance and resus

Critical aspects of ICU care

By Wendy J. Stevens, CRNP, BSN, MSN

Critically ill patients are at great risk for volume depletion that may be secondary to internal and external fluid losses. Daily fluid balance assessments may be inaccurate, as total volume losses aren't always recognized or measurable. Hypovolemia can progress from a state of mild dehydration to severe and profound fluid loss that may lead to shock and end organ failure if not treated appropriately. Despite the daily recording of a positive or negative fluid balance, patients' clinical status must be addressed in relation to evaluate the severity of their hypovolemic state. These patients can present with symptoms that may be acute, chronic, or acute on chronic.

According to experts, "A patient may be in shock despite having a normal heart rate and blood pressure."[1] That statement truly emphasizes that a critically ill patient can have normal hemodynamic parameters; however, end organ perfusion may be compromised. Without adequate fluid resuscitation, tissue ischemia worsens, as well as chances for an optimal recovery lessen.

The circulating volume in the body is a small percentage compared to the overall fluid that the body contains. As a result, a 15% to 20% blood loss has less effect on the circulation than if there is a 30% to 40% blood loss, which can result in life-threatening circulatory failure.[2] Fluid resuscitation must not be delayed and treatment goals are set in order to guide management. Daily physical exams, laboratory values, and invasive monitoring evaluate the effectiveness of adequate fluid resus-

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citation. Without restoration of effective circulation, organs and tissues become ischemic and irreversible damage may ensue.

Early recognition

The timing of resuscitation is critical as mortality is directly related to the extent and duration of organ hypoperfusion of those in hypovolemic shock.[2] Identification of the high-risk patient who may be labeled as a "nonresponder" to initial management, may require more aggressive fluid resuscitation in order to avoid irreversible consequences of shock.[3] Patients who present early with signs of hemodynamic compromise must be monitored in the critical care unit. Signs and symptoms of hypovolemia vary depending on the type and duration of fluid loss. For example, lethargy, confusion, anxiety, dry mucous membranes, and tachycardia can present in the early stages of hypovolemia. Late and more ominous signs of volume depletion include decreased responsiveness, tachycardia with a low blood pressure, renal failure along with poor urine output, slow capillary refill, and weak peripheral pulses. Keep in mind, these signs and symptoms may be an early clue to an underlying acute illness.

In order to adequately fluid resuscitate a patient, it's important to know, if possible, what type of fluid has been lost, what types of fluid resuscitation can be used, as well as the amount and timing of fluid resuscitation. Depending on the patient's current condition, a thorough account of the history of the present illness, physical exam, and medical history are important pieces of information to obtain which may help guide treatment. The timing of recovery, as well as expected benefit, depends on when treatment is first initiated.

Physiology of fluid compartments

Having knowledge of the body's fluid composition develops a greater appreciation for how to interpret losses in a dehydrated or hypovolemic patient. All adults differ in body composition and may not require the same amount of fluid because weights, heights, and lean body mass vary. Water makes up the largest component of the body and is present in higher quantities in people who have more muscle mass versus fat and is present in higher quantities in an infant versus an adult. Fat has a low content of water. A person with minimal muscle mass or a person who's obese will have a lower percentage of body water. As a result, a lean person will have a higher content of water where substantial fluid losses may not be well tolerated. Infants have a total body water (TBW) of 80% which puts them at even greater risk, as well as intolerance of fluid losses secondary to a higher TBW (compared with an adult).[4]

The percentage of fluid in each body compartment is consistent in adults regardless of their size; however, the overall TBW content may vary. The body's fluid compartments can be divided into TBW and the components of blood which are listed in "Fluid compartments by gender." Values are rounded and may not equal exact total

Fluid compartments by gender

Fluid	Male (80 kg)	Female (70 kg)
Total body fluid	0.6 liter or 600 mL/kg = 48 liters	0.5 liter or 500 mL/kg = 35 liters
Whole blood	66 mL/kg = 5.3 liters	60 mL/kg = 4.2 liters
Plasma	40 mL/kg = 3.2 liters	36 mL/kg = 2.5 liters
Red blood cells	26 mL/kg = 2 liters	24 mL/kg = 1.6 liters

(Marino, P. 2007)

of whole blood amount). Values depicted are based on an 80 kg male versus a 70 kg female. The 80 kg male typically may have more lean muscle mass than a 70 kg female. Therefore, the TBW is 10% less in a 70 kg female versus an 80 kg male.[2]

An adult's TBW content is distributed throughout the extracellular (intravascular and interstitial) and intracellular fluid compartments. Both compartments have the same osmolality, which is determined by the sodium (Na^+) salt concentration in the extracellular compartment or the potassium salt concentration within the intracellular compartment. The concentrations of these cations normally stay relatively constant; however, water constantly shifts in order to maintain an equal osmolality between the two compartments.[5] Approximately 60% to 65% of TBW exists in the intracellular compartment because more potassium salts exist intracellularly, drawing more fluid than total sodium salts in the extracellular space; however, the osmolality of both compartments remains the same. The remaining 35% to 40% of TBW exists in the extracellular compartment. The extracellular space is defined as the intravascular and interstitial compartments, of which 11% to 12% of the volume is intravascular and the remaining 75% to 80% is in the interstitial space.[4] Note that the intravascular space is not a large compartment when compared to the other fluid compartments within the body, which is why it's very sensitive to volume depletion.

Inside albumin

Albumin is an important component of the extracellular fluid compartment. It's a large protein molecule that exists in higher concentrations in the interstitial versus intravascular spaces. However, it isn't able to freely cross the capillary membranes, and therefore helps to maintain adequate colloid pressure to hold water in the intravascular space and retain effective circulating volume. Albumin plays an important role in fluid resuscitation; however, it isn't often a first choice secondary to its cost and availability. This is important to take into consideration when deciding which kind of fluid will be most beneficial during initial stages of fluid resuscitation.

Fluid deficits

The type of fluid deficit often defines patients' clinical condition as well as the cause of their hypovolemic state. Patients can present with a fluid deficit secondary to various medical and or environmental conditions. Hypovolemia is best understood when classified as relative secondary to internal fluid shifts as well as insensible losses or absolute secondary to a direct, quantifiable, and usually measurable loss.

It's common that relative and absolute hypovolemic states can coexist in certain clinical conditions as well. A patient who's relatively hypovolemic may have adequate volume; however, it doesn't remain or presently exist in the intravascular space. In other words, it isn't effective circulating volume. Examples include patients with open abdomens, those in septic or distributive shock, and patients who have high temperatures, profuse diaphoresis, large pulmonary secretions, are ventilator dependent without adequately humidified circuits, or have third spaced fluid in the interstitial compartment with massive edema. Relative loss that's due to third spacing may include those with an ileus, intestinal obstruction, compartment syndrome from a fracture, or an intra-abdominal compartment syndrome secondary to a bleed, ascites, or severe acute pancreatitis. Absolute hypovolemia is considered to be measurable fluid loss. Examples include but are not limited to hemorrhage, diarrhea, and high output fistulas (greater than 500 mL/day).[6,7]

Vital signs

Signs and symptoms vary between those who may be mildly dehydrated, profoundly hypovolemic, or in shock and showing signs and symptoms of organ dysfunction. Three ways to evaluate a patient's fluid status are vital signs, end points, and the use of invasive monitoring.

Experts conducted a series of studies to assess the accuracy of vital signs when there is an acute moderate-to-severe blood loss. Moderate blood loss was defined as 450 to 630 mL and severe blood loss was equated to 630 to 1,150 mL, or close to 20% loss of total blood volume.[8] They found that supine tachycardia was present in up to 42% of patients with moderate blood loss and only 5% to

Fluid types and compositions

Fluid	Na+	Cl-	K+	Ca++	Mg++	Buffers	pH	Osmolality
NaCl (0.9%)	154	154					5.6	295
LR	130	109	4	3		Lactate (28 mEqL)	6.4	273
Normosol	140	98	5		3	Acetate (27 mEqL)	7.4	295
D5W							4.4	278
Albumin 5%	130	130					6.4 – 7.4	300
Hetastarch 6%	154	154					5.5	300
Hextend 6% (contains 5 mM/L of glucose	143	125	3	5	0.9	Lactate (28 mEqL)	5.5	300

(Gan 1999, Griffith 1986, Halpern et al. 1997)

24% of patients with severe blood loss. Orthostatics were checked as well in patients that could stand upright. They found that 91% to 100% of patients with severe blood loss had a postural pulse rise of greater or equal to 30 beats per minute (bpm) as well as postural dizziness. A normal increase in heart rate is 10 bpm in healthy individuals.[8] Of those with moderate blood loss, 6% to 48% had an increase in pulse rate. Overall, moderate and severe blood loss presented with tachycardia and hypotension in the supine position close to 50% of the time which demonstrates that evaluation of orthostatic vital signs may provide limited information in moderately hypovolemic patients.[8] Once blood loss exceeds 30%, hypotension is usually apparent; however, it's interesting to note that hemodynamic instability may be a late sign in 50% of hypovolemic patients.[9]

What's the end point?

Traditionally, measurements of adequate fluid resuscitation include normalization of the heart rate (HR), blood pressure (BP), and urine output (UOP) in both medical and surgical patient populations. Research has shown that despite compensation in shock, ongoing hypoperfusion at the tissue level exists. Three main indicators have been studied to show tissue hypoperfusion at the cellular level and these are lactate levels, mixed venous oxygen saturations (SvO_2), and base deficit (greater than -4), which is derived from an arterial blood gas. In two studies of trauma patients, researchers found that despite normalization of vital signs and urine output, close to 80% of patients still had evidence of inadequate tissue perfusion as defined by an elevated lactate level (greater than 4) or decreased SvO_2 (less than 60%).[10,11] Two other populations that have been studied, assessing end points, have been noncardiac surgical patients and those that are septic.

Lactate and base deficit

The relationship between serum lactate levels and hypovolemic shock, as well as its correlation with death in critically ill patients, has been extensively evaluated in several publications from 40 years ago.[12] Base deficit has also been evaluated as a measurement of global tissue acidosis since the 1980's.[9] Elevated lactate levels are associated with a metabolic acidosis in most cases of hypoperfusion and signify an ongoing oxygen debt at the tissue and cellular level. Both lactate levels and base deficit can be elevated together; however, base deficit can be elevated alone in end stage renal failure and in this case, does not indicate a lack of tissue perfusion or an acute process. When comparing elevated lactate levels versus an increased base deficit, an elevated lactate in a hemodynamically unstable patient is associated with a significantly higher mortality rate if not corrected quickly.[2]

Gastric pH

In the last several years, there has also been research done on gastric mucosal pH as a regional end point of resuscitation as it reflects adequacy of perfusion to the gut. However, as of yet, this has not become a frequent measurement that is used in intensive care units (ICUs) today. It's considered an adequate regional indicator of resuscitation because blood flow in hypoperfused states isn't equally distributed, and gastric pH may be the only indicator that perfusion to the splanchnic bed or gut mucosa is still being affected despite a normal lactate and base deficit.[13] As a result, clinicians have found that

Signs and symptoms associated with electrolyte imbalances

Electrolyte	Signs and symptoms	Hemodynamic effects
High glucose	• Polyuria, polydipsia, severe dehydration, altered mental status, metabolic acidosis	• Tachycardia secondary to dehydration from high glucose (osmotic diuresis), hypotension
Low glucose	• Decreased mental status, seizure	• Dependent on other electrolyte abnormalities
High and low sodium	• Excessive thirst, lethargy, confusion, seizures, coma	• Dependent on other electrolyte abnormalities
High and low potassium	• < 2.5 mEq/liter = Diffuse muscle weakness • 2.5-3.5 = usually asymptomatic, often due to diuresis, magnesium depletion • > 5.5 may be associated with rhabdomyolysis, acidosis, blood transfusions (old blood > 14 days)	• K+ (< 2.5 - 3.0) - Prolongation of QT interval but not specific to low potassium levels, (usually other electrolyte deficiencies present), flattened T waves • K+ (> 6.0) – beginning ECG changes, widened QRS, loss of P waves, peaked T waves • K+ 8.0 – Atrioventricular blocks
Low magnesium	• Associated with: secretory diarrhea (not vomiting), hypophosphatemia, altered mentation, seizures, tremors, slurred speech, metabolic acidosis	• Tachyarrhythmia, torsade de pointes

(Kassirer, Hricik, & Cohen 1989, Marino 2007, Martin 1969, Williams, & Rosa 1988)

despite treatments to improve the traditional end points of HR, BP, UOP, organ tissue oxygenation deficits persist as evidenced by changes in gastric pH.[1]

Evaluate electrolytes

Metabolic derangements and acid/base disorders as well as their associated signs and symptoms play a large role when evaluating and treating hypovolemic patients. Laboratory values that are important indicators of volume status are sodium, creatinine, hemoglobin, and lactate. These values may be elevated or decreased depending on their history or disorder that has initially caused their hypovolemic state. (See "Signs and symptoms associated with electrolyte imbalances.") A patient's electrolyte status will determine what type of fluids will be most beneficial during resuscitation. A high sodium level and an elevated creatinine are often initial clues pointing toward dehydration. However, a person's baseline sodium level may be low in those with heart failure or cirrhosis where their effective circulating volume is higher than normal and they may not be hypovolemic.

Choice of fluid for resuscitation

Three types of fluids frequently used today for active resuscitation are red blood cells, acellular colloid fluids with large molecules that keep fluid intravascularly (hetastarch, dextran 40, albumin), and crystalloid fluids with added electrolytes (NS, LR, Normosol). (See "Fluid types and compositions.")

Depending on the composition of certain fluids, varied amounts of fluid remain in the intravascular space for a certain length of time. For example, because more sodium exists in the extracellular compartment (intravascular and interstitial), fluids higher in sodium such as NaCl and LR will preferentially enter the interstitial space as sodium floats freely between the interstitium and blood vessels and water follows. Therefore, only 25% of a liter of NaCl will remain in the intravascular space. The 1,100 mL that actually enters into the extracellular fluid space is due to water shifts secondary to a slightly higher sodium content administered as compared to what exists in our plasma (154 mEq verses 140 mEq) and 100 mL is actually pulled away from the intracellular compartment to be added to the interstitium. Crystalloids mainly expand the interstitial space, not the intravascular space, yet are used initially to resuscitate a patient with hypovolemia.

Colloid vs. crystalloid

Both crystalloid and colloid solutions are considered effective for the resuscitation of a hypovolemic patient as neither fluid provides a survival benefit that is superi-

Advantages and disadvantages

Fluid	Advantages	Disadvantages	Price (per liter)
Normal saline	• Interstitial fluid replacement • Not viscous	• Hyperchloremic metabolic acidosis • Large volumes lower pH • 25% in intravascular space	$1.46
Lactated ringers	• Interstitial fluid replacement • Buffer, maintains stable pH • Not viscous	• Not to be given with blood transfusions—calcium in LR would inhibit anticoagulant effect of citrate in PRBCs • 25% in intravascular space	$1.48
Albumin (5% and 25%) **Give with furosemide (Lasix) for optimal diuresis	• 70% intravascular fluid replacement • Rare reactions • Volume expander • Buffer, antioxidant properties, inhibits platelet aggregation • Not viscous • Optimal for third spacing (pulmonary or peripheral edema), severe hypoalbuminemia/ malnutrition/cirrhosis/ARF	• 25% albumin not good for initial resuscitation—small volumes administered • Expensive	$30.63
Hetastarch 6% **Limit use to 1,500 mL in 24 hrs	• Effective volume expander • Not viscous	• Inhibits Factor VII and von Willebrand Factor (vWF), impairs platelet adhesiveness, coagulation effects are dose dependent (> 1,500 mL/24 hrs)	$27.63
Dextran **Limit use to < 20 mL/kg	• Effective volume expander • Dextran 70 lasts 12 hours • Dextran 40 lasts 6 hours • Not viscous	• Dose-related bleeding tendency, impairs platelet aggregation, decreases levels of Factor VIII and vWF, enhances fibrinolysis	$14.96
Hypertonic saline (3%, 7.5%)	• Possible benefit in head trauma (currently in use) • Less volume —expands intravascular volume with fluid shifts to decrease cerebral edema	• Cell dehydration	$5.00 or $13.00 per 500 mL, depends on brand)

(Griffel & Kaufman 1992, Imm & Carlson 1993, Jacob et al. 2005, Vincent et al. 2004, Chiara et al. 2003, Cooper et al. 2004)

or to the other.[14-16]

Experts have conducted large studies on patients using colloids versus crystalloids in fluid resuscitation. Nineteen trials consisting of 1,315 participants (trauma, burns, and surgical patients) were done to compare differences in mortality between colloid and crystalloid resuscitation. The overall mortality difference was low (4%), or four deaths for every 100 patients resuscitated. The greatest difference noted between the use of colloids and crystalloids in that study was cost.[17] A large trial known as the SAFE (Saline versus Albumin Fluid Evaluation) study was conducted between the years of 2001 and 2003 including mainly trauma and septic patients. Again, there was no difference found in 28 day outcomes which was defined as the number of days spent in the ICU, hospital length of stay, duration of mechanical ventilation, and duration of renal-replacement therapy.[16]

Regardless of mortality, different clinical scenarios may warrant the use of colloid versus crystalloid. For example, dehydrated patients secondary to vomiting, diarrhea, or diabetic ketoacidosis may require crystalloids first if there is no obvious blood loss. Another patient, who is malnourished and has chronic liver disease, may benefit from a colloid such as albumin, as they have low stores of this protein which normally helps keep fluid the intravascular space. Patients with

Choosing the right fluid

Colloid fluid (infusion of 1 liter each)	Oncotic pressure (mm Hg)	Change in plasma volume	Duration of effect in intravascular space
10% destran 40	40	– 1.5	6 hours
6% hetastarch	30	1.0 – 1.3	10 hours
5% albumin	20	0.1 – 1.3	16 hours
25% albumin	70	4.0 - 5.0	16 hours

(Halliwell 1988, Gan 1999, Moore 1965, Shires 1964, & Weil 2004)

poor cardiac function can benefit not only from blood products if they are ischemic, but also from crystalloids in order to promote flow and enhance cardiac output if volume is indicated.

When trying to augment cardiac output and blood pressure, colloids have an advantage over crystalloid solutions as a larger percentage enters the intravascular space and remains there for a longer period of time.[2] This is because colloids provide the greatest effect on intravascular volume expansion and improve flow secondary to their low viscosity which is equal to that of water.[2] For example, up to 80% of a dextran 40 colloid solution enters and remains in the intravascular space. Three to 4 liters of crystalloid fluid would have to be given in order to equal the amount of 1 liter of colloid that remains in the intravascular space. Both crystalloids (NaCl, LR) and colloids (hetastarch, albumin 5%, 10% dextran 40) have equal viscosities and provide equal flow which in turn augments cardiac output and blood pressure.[2]

Possible complications

Fluid resuscitation as well as blood transfusions can be beneficial to a hypovolemic patient; however, risks and complications must always be taken into consideration with any treatment given. However, in the acute phase of hypovolemia or shock, circulation is a priority and side effects from excess fluid are to be expected if the clinical status of a patient worsens. Adverse effects commonly seen in critically ill patients that may require immediate treatment are cerebral edema (which may lead to increased intracranial pressure), acute lung injury, acute respiratory distress syndrome, transfusion related lung injury, abdominal compartment syndrome, and third spacing or edema.[3] Large volumes may also dilute clotting factors and enhance bleeding (secondary coagulopathy), disrupt thrombus formation, and induce hypothermia (See "Advantages and disadvantages.")

Third spacing

Third spacing is usually apparent within the first few hours to days and can take weeks to resolve depending on the patient's clinical course. As mentioned before, close to 70% of crystalloid infusions enter the interstitial space and don't stay within the vasculature. There's an associated hypoalbuminemia secondary to dilution from large volume resuscitation which further exacerbates third spacing or peripheral edema.

Critically ill patients with large amounts of edema are at risk secondary to many compounding factors in addition to their reason for being in the ICU. For example, hypoalbuminemic patients retain excess fluid and are prone to infection as edematous tissues easily break down into pressure ulcers secondary to poor circulation to the dependent areas of the body (scalp, back, sacrum, heels). It's also common to see third spacing in sepsis as well as septic and anaphylactic shock secondary to the capillary leak phenomenon due to cytokine activation and endothelial destruction. The presence of third spaced edema, in conjunction with a positive fluid balance seen in hypotensive patients with acute renal failure, clearly indicates that the clinical appearance of patients doesn't always match their hemodynamic status.

Treatment of third spacing is effectively done with a colloid that has an oncotic pressure greater than or equal to the normal plasma oncotic pressure of 25 mm Hg.[2] As shown in "Choosing the right fluid," 25% albumin delivers the highest colloid oncotic pressure; however, it does not adequately replace volume for dehydrated or hypovolemic patients because it is administered in amounts of 50 to 100 mL. It therefore isn't an initial choice in the acute phases of fluid resuscitation. However, it's useful in relatively hypovolemic patients who are very edematous, as it assists in shifting excess fluid from the interstitial compartment back into the intravascular space.

Inside the ICU

A fluid balance is not always able to be precisely calculated, which is why the patient's clinical exam as well as hemodynamic status evaluated together may be a more accurate reflection of their volume status. Vital signs are not as reliable as end points of resuscitation alone as they lack detection of ischemia at the cellular level. Endpoints indicating ongoing tissue hypoperfusion include lactate, base deficit, and gastric mucosal pH. If abnormal, effective circulation must be restored and maintained to achieve homeostasis and end organ perfusion.

Whether resuscitating with a crystalloid, colloid, or blood products, it's important to accomplish stability rapidly. Further delays in fluid resuscitation in the face of impeding organ failure can only compromise a patient's outcome and chance for an optimal recovery. M

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