Fluid Choice for Resuscitation and Perioperative Administration

Fluid administration is considered the cornerstone of resuscitative therapy for the treatment of hypotension, hypovolemia, and physical trauma. Isotonic salt (crystalloid) solutions (e.g., lactated Ringer’s solution [LRS]) are the most frequently recommended fluids for this therapy because they are considered to be safe, efficacious, and cost-effective. Seminal studies conducted by Post et al in the late 1940s and early 1950s demonstrated that crystalloid administration reduced mortality in hemorrhaged dogs. Subsequently, Shires et al concluded that the total extracellular fluid volume was decreased independent of blood loss in dogs that were hemorrhaged, thereby suggesting the need for both intravascular and interstitial fluid replacement. These studies, in conjunction with the low incidence of adverse effects associated with the liberal administration of large volumes of isotonic crystalloids to wounded soldiers and surgical candidates during the Korean and Vietnam wars, provided “evidence” that large-volume crystalloid therapy was necessary and well tolerated and could be lifesaving. Subsequently, however, several authors, including Shires, urged careful evaluation of fluid requirements in traumatized patients and moderation in crystalloid administration. Regardless of this caution, the administration of large volumes of crystalloid solutions has become standard for trauma patients, during the perioperative period, and for the treatment of hypotension and hypovolemia.

Fluid therapy is not innocuous and should not be so automatic as to preclude consideration of the volume, electrolyte content, osmotic value, oncotic pressure, acid–base characteristics, viscosity, and oxygen-carrying characteristics of the fluid being administered relative to these same values in the patient. This review discusses each of these issues and provides historical and current perspectives based on a diverse set of references and reviews. Fluid therapy should not be formulaic or “recipe-based” (i.e., focused on mL/kg/min or hr) but rather goal directed; that is, designed to address each of the aforementioned issues while attempting to attain a predetermined goal.

The “Evidence” for Crystalloid Therapy
Fluid therapy with crystalloids has become so routine that it is frequently undertaken without consideration of its adverse effects, including overt fluid overload (edema); electrolyte, acid–base, and rheologic (blood flow–related) disturbances; hypothermia; and potentially delayed recovery from anesthesia and impaired wound healing. When fluid resuscitation is deemed necessary (BOX 1), most veterinary texts and emergency monographs recommend moderate to aggressive fluid therapy regimens (e.g., bolus administration of 10 to 20 mL/kg LRS or normal saline [0.9% NaCl] to effect and replacement of blood loss with LRS at a ratio of 3:1 to 8:1 or a volume of 80 to 90 mL/kg). These regimens are expected to increase or maintain arterial
Fluid Choice for Resuscitation

**WEB EXCLUSIVE**

Blood pressure, cardiac output, tissue perfusion, and oxygenation. However, the administration of isotonic crystalloids only temporarily improves arterial blood pressure, and only when the fluid is administered at rates faster than its rate of redistribution into the interstitial space. Less than 15% of a 1000-mL IV bolus of LRS administered over 1 hour is retained within the vascular compartment 20 minutes after completion of administration.3

Even when isotonic crystalloids are administered at rapid rates (>30 mL/kg/hr), tissue perfusion (microcirculatory [capillary] blood flow) may not change for some organs and may decrease in others (e.g., intestines).3 An increasing amount of data suggests that recipe-based fluid therapy regimens worsen pulmonary gas exchange and limit oxygen diffusion to tissues (by promoting capillary vascular endothelial swelling) in conscious or anesthetized animals.5-7 These studies warn that these regimens should be used sparingly, especially in animals requiring intestinal resection, and are likely to lead to interstitial fluid accumulation and detrimental dilutional (packed cell volume [PCV], proteins, electrolytes, buffers), acid–base (nonrespiratory acidosis), and rheologic effects.8 Excessive crystalloid administration (>30 to 50 mL/kg) increases mortality during uncontrolled hemorrhage and worsens hypothermia, coagulopathy, and immune function.3,4,9,10 Attempts to restore vascular volume by administering large volumes of crystalloids, therefore, must be monitored because this

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**QuickNotes**

Fluid therapy should not be formulaic but rather goal directed.

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**General Diagnostic Criteria for Fluid Resuscitation**

**Owner Observations**
- Weakness
- Lethargy, depression progressing to recumbency/unresponsiveness
- Pallor
- Abdominal distention (e.g., hemoabdomen)
- Loss of appetite
- Decreased body temperature

**Other Historical Considerations/Predispositions**
- Time from trauma/hemorrhage to presentation
- Severity of trauma
- Hydration status
- Volume (mL/kg) of blood lost
- Duration of hypotension
- Initial PCV/TS
- Baseline chemistries
- Concurrent disease
  - Heart failure
  - Pulmonary disease
  - Renal failure
  - Other

**Physical Examination Findings**
- Signs of trauma (skin abrasions, soft tissue swelling, fractures)
- Ongoing hemorrhage
- Moderate to severe pain
- Depressed mentation

**Laboratory Findings**
- High PCV (dehydration, hemoconcentration)
- Normal or reduced PCV (<30%); serial PCV/TS should be evaluated
- Normal or reduced TS (<5.5 g/dL); can precede decreased PCV in acute hemorrhage
- Normal or reduced PaCO₂ (<30 mm Hg; respiratory alkalosis)
- Normal or decreased bicarbonate (<15 mEq/L) and base excess (>−5 mEq/L; nonrespiratory acidosis)
- Increased lactate (>2 mM/L; lactic acidosis)
- Prerenal azotemia
- Anuria (<0.5 mL/kg/hr), oliguria (0.5–1.0 mL/kg/hr), or dilute urine (urine specific gravity <1.010; Na⁺ concentration <10 mEq/L)
- Elevations in creatine kinase due to muscle trauma

PCV = packed cell volume, TS = total solids
practice has been demonstrated to be remarkably inefficient and transient at best, predisposing animals to iatrogenic complications.3,10

In short, evidence supporting the long-term benefits of recipe-based crystalloid fluid regimens during anesthesia and surgery or the repeated administration of 10 to 20 mL/kg or larger boluses of LRS to hypotensive or hypovolemic anesthetized dogs or cats does not exist. The potential for isotonic fluids to produce beneficial effects is further hindered by the cardiovascular, lymphatic, and renal and gastrointestinal losses. Hetastarch anesthesia, for example, decreases urine excretion, promotes accumulation of extravascular (interstitial) fluid, facilitates third-space losses, and inhibits transcapillary refill in normotensive or hypotensive hemorrhaged animals administered a 25-mL/kg/20 min crystalloid bolus.6,7 The administration of isotonic crystalloids during isoflurane anesthesia for the replacement of blood loss increases total tissue water in the intestines, liver, skeletal muscle, and lungs.7

The ability of dogs and cats to tolerate large volumes of crystalloid solutions is more a testament to their compensatory capabilities (cardiovascular, lymphatic, and renal) and the capacity of the extravascular fluid compartments than evidence of the therapeutic value of this therapy. It is highly likely that many animals live in spite of large volumes of colloids rather than because of them.

Properties of Crystalloid and Colloidal Solutions

Solutions exert an osmotic effect on tissue, known as osmotic pressure (measured in mOsm/L), based on the number of particles in solution. The osmotic pressure of blood depends on the serum concentration of diffusable salts (electrolytes), proteins, and other molecules.11 Commercially manufactured crystalloids contain prescribed concentrations of various electrolytes diluted in water (TABLE 1). These relatively small (<50 Da) molecules rapidly diffuse into the interstitial space, providing temporary and minimal intravascular volume expansion. Other than hypertonic saline, most crystalloids used for resuscitation are isotonic and isomotic; that is, they have electrolyte concentrations similar to those of plasma. LRS is hypotonic (273 mOsm/L) compared with plasma (285 to 310 mOsm/L) and therefore promotes extravascular fluid redistribution.

Colloidal solutions contain molecules (albumin, dextrans, hetastarch)12 that are too large (>30,000 Da) to rapidly diffuse out of the blood vessels. As a result, they exert an intravascular colloid osmotic (oncotic) pressure (COP). The COP of most colloidal solutions is generally greater (range: 25 to 80 mm Hg) than that of dog or cat plasma (18 to 23 mm Hg), slowing the flux of fluid into the interstitial space and, when larger doses (>10 mL/kg) are administered, resulting in the temporary movement of fluid from the interstitial space into the vascular compartment (TABLE 2). More concentrated colloidal solutions contain more molecules and generate a greater COP. The COP counteracts intravascular hydrostatic (blood) pressure and helps balance fluid distribution between the intravascular and interstitial fluid compartments, as expressed in Starling’s law of capillary hemodynamics.a Clinically, COP values below 10 to 12 mm Hg in small animal patients are associated with significant loss of intravascular fluid into the interstitial fluid compartment, hypovolemia, and increased morbidity and mortality.

Colloids are generally characterized by their molecular weight. For example, the natural colloid albumin is monodisperse, meaning that all the particles are the same size (69 kDa), and its oncotic effect (protein osmotic effect) depends on the concentration administered: 5% is iso-oncotic (308 mOsm) and produces a COP of approximately 20 mm Hg, and 25% is hyperoncotic (1500 mOsm), producing a COP of approximately 100 mm Hg.13,14 Synthetic colloids (dextrans, hetastarch) are polydisperse, meaning that they contain a range of molecular weights, including some molecules that have a low molecular weight (<30 kDa) and pass rapidly through the capillary membrane, thereby decreasing the oncotic pressure.

The duration of a colloid’s COP is related to the fluid’s rate of extravasation, metabolism, and renal and gastrointestinal losses. The duration of a colloid’s COP is related to the fluid’s rate of extravasation, metabolism, and renal and gastrointestinal losses. Hetastarch (amylopectin-based) derivatives that are highly substituted with hydroxyethyl groups take longer to be metabolized to a size that can be filtered by the kidneys. However, the duration of a colloid solution’s clinically effective volume-expanding effect is determined not only by its

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aFor more information on Starling’s law, see “Limited Fluid Volume Resuscitation” (July 2009), also available on CompendiumVet.com.
molecular weight and degree of substitution, but also by its effective molecular radius, shape, electrical charge, and interaction with the vessel wall, especially the vessel wall endothelium.\textsuperscript{8,15} One of the beneficial effects of some colloids is that they are believed to plug membrane pores; similarly, the negative charge on albumin may restrict its penetration through the negatively charged vessel wall.\textsuperscript{16} These properties are important in decreasing colloid leakage from the blood vessels.

The efficacy of colloids in clinical practice is governed by their physical properties and the permeability of the microvasculature. Microvascular permeability and transcapillary leakage are increased in septic, traumatized, and severely hypoxemic animals, and colloid administration in these patients results in a duration of vascular expansion shorter than that in less critically ill patients.

Other essential components of Starling’s law of capillary hemodynamics include capillary wall integrity and the fluid filtration coefficient. This law must be considered in the context of evidence demonstrating the importance of the endothelial glycocalyx and its influence on transvascular fluid flux during health and disease (e.g., shock, sepsis).\textsuperscript{5}

### Clinical Considerations

Concern for the following principles is important when developing fluid-based replacement or resuscitation strategies. Any fluid therapy strategy should be well thought out before administration.

### Acid–Base Disturbances

Acid–base disturbances are common in traumatized, hypovolemic, hypothermic, hypotensive, or anesthetized dogs and cats. Increases

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### Table 1: Characteristics of Fluids Commonly Administered for Resuscitation

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Osmolarity (mOsm/L)</th>
<th>Na\textsuperscript{+}</th>
<th>Cl\textsuperscript{−}</th>
<th>K\textsuperscript{+}</th>
<th>Mg\textsuperscript{2+}</th>
<th>Ca\textsuperscript{2+}</th>
<th>Buffer</th>
<th>pH</th>
<th>SIDe</th>
<th>Colloid Oncotic Pressure (mm Hg)</th>
<th>Viscosity (cP)</th>
<th>P\textsubscript{50} (mm Hg)</th>
<th>Concentration (g/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Crystalloids</strong></td>
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<tr>
<td>Normal saline (0.9% NaCl)</td>
<td>308</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td>5.0</td>
<td>0</td>
<td>0</td>
<td>≈1</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>LRS</td>
<td>273</td>
<td>130</td>
<td>109</td>
<td>4</td>
<td>3</td>
<td>L-28</td>
<td>6.7</td>
<td>27</td>
<td>0</td>
<td>0.85</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normosol-R</td>
<td>294</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>3</td>
<td>A-27; L-23</td>
<td>6.6</td>
<td>45</td>
<td>0</td>
<td>≈1</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Plasmalyte-R</td>
<td>295</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>3</td>
<td>A-27; L-23</td>
<td>7.4</td>
<td>50</td>
<td>0</td>
<td>≈1</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hypertonic saline (7.5% NaCl)</td>
<td>2566</td>
<td>1283</td>
<td>1283</td>
<td></td>
<td></td>
<td></td>
<td>5.0</td>
<td>0</td>
<td>0</td>
<td>≈1</td>
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<tr>
<td><strong>Colloids</strong></td>
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<tr>
<td>Dextran 70</td>
<td>309</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td>3–7</td>
<td>0</td>
<td>61.7 ± 0.5</td>
<td>3.68</td>
<td>65.8</td>
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<tr>
<td>5% Albumin</td>
<td>308</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td>19</td>
<td>1.2–1.5</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>6% Hetastarch/saline</td>
<td>308</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td>5.5</td>
<td>0</td>
<td>32.7 ± 0.2</td>
<td>4.3</td>
<td>60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6% Hetastarch/LRS (Hextend)</td>
<td>304</td>
<td>143</td>
<td>124</td>
<td>3</td>
<td>0.9</td>
<td>L-28</td>
<td>5.9</td>
<td>26</td>
<td>43</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6% Hetastarch/saline (Voluven)</td>
<td>308</td>
<td>154</td>
<td>154</td>
<td></td>
<td></td>
<td></td>
<td>5.5</td>
<td>42</td>
<td>2.41</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood</td>
<td>300–305</td>
<td>144–153</td>
<td>110–120</td>
<td>3.8–5.3</td>
<td></td>
<td></td>
<td>7.4</td>
<td>18–23</td>
<td>3.5</td>
<td>27</td>
<td>120–140</td>
<td></td>
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</tr>
</tbody>
</table>

\textsuperscript{A} = acetate, \textsuperscript{L} = lactate, \textsuperscript{LRS} = lactated Ringer’s solution, \textsuperscript{P\textsubscript{50}} = \textsubscript{P}_o\textsubscript{2} at which hemoglobin is half saturated with oxygen, \textsuperscript{SIDe} = effective strong ion difference
in arterial blood CO₂ (increased Pa₉O₂; respiratory acidosis) caused by hypoventilation can exacerbate acidemia and anesthetic drug effects. Alternatively, arterial blood CO₂ may be decreased (respiratory alkalosis) in conscious animals that are frightened, stressed, or in pain, even to the point of compensating for metabolic acidosis. Lactic acidosis is a common cause of nonrespiratory acidosis in hypoxic, hypotensive, hypovolemic animals and, when combined with central nervous system trauma or anesthesia and respiratory acidosis, decreases cardiac contractile force, sensitizes the heart to cardiac arrhythmias, reduces vascular tone and catecholamine responsiveness, and impairs immune cell function. The causes and consequences of these compensatory or additive acid-base effects warrant a thorough acid-base evaluation and a comprehensive approach to the assessment of acid–base disorders in these patients.

All clinically relevant acid–base abnormalities can be explained by changes in strong ions (highly dissociated in water: Na⁺, K⁺, Ca²⁺, Mg²⁺, Cl⁻, lactate⁻), weak acids (not totally dissociated in water: albumin, phosphate), and CO₂. Methods for evaluating acid–base disorders that are based on determination of strong ions are especially helpful in deciphering and treating complex acid–base abnormalities. The charge generated by strong ions produces an apparent strong ion difference (SIDa) that is counterbalanced by negative charges primarily generated by albumin and phosphate, called the effective strong ion difference (SIDe) or buffer base. The difference between the SIDa and the SIDe is termed the strong ion gap (SIG) and should be close to zero in healthy animals with a blood pH of 7.40 and a Pa₉O₂ of 40 mm Hg. Increases or decreases in SIG indicate an acid–base abnormality. Metabolic acidosis caused by the production of large amounts of unmeasured anions (lactic acidosis) occurs during global tissue hypoperfusion, increasing the SIG. The SIG has been correlated with outcome and shown to be a more sensitive determinant of mortality than the lactate, bicarbonate, base deficit, or anion gap value.

Blood and Plasma Viscosity

Tissue oxygenation is critical to patient survival and depends on blood flow and the intrinsic oxygen-carrying capacity of blood. It is primarily determined by the cardiac output, hemoglobin concentration, hemoglobin saturation, and arterial partial pressure of oxygen. In addition, blood viscosity is a key, yet underemphasized, determinant of blood pressure, microcirculatory blood flow distribution, and tissue perfusion. Traumatized, hemorrhaged, or anemic animals depend on blood viscosity for maintenance of microvascular perfusion. Even when blood pressure and cardiac output (macrocirculatory parameters) are within normal limits, severely decreased plasma viscosity can cause maldistribution of blood flow to vital organs in traumatized, septic, and anesthetized animals, resulting in inadequate tissue oxygenation.

**QuickNotes**

The efficacy of colloids in clinical practice is governed by their physical properties and the permeability of the microvasculature.
As shown in the following equation, blood viscosity (η) is a determinant of resistance to fluid blood flow:

\[
\text{Resistance} = \eta \times \frac{L}{r^4}
\]

\( L \) = length; \( r \) = radius

Blood viscosity depends on the PCV squared. Microcirculatory viscosity is linearly related to the PCV and becomes increasingly dependent on plasma viscosity as vessels become smaller.\(^{25}\) As vessels become smaller, the PCV decreases because of central streaming of red blood cells (the Fahraeus-Lindquist effect). This effect gradually decreases the blood viscosity in the capillaries to values that are half those in the arteries. The reduced viscosity helps to maintain tissue blood flow and oxygen delivery by reducing vascular resistance (blood flow = pressure/resistance).

Vascular tone also decreases in smaller vessels because of the reduction in hemoglobin concentration. Hemoglobin is a nitric oxide scavenger, and nitric oxide is a vasodilator; therefore, in healthy animals, the reduced viscosity in smaller vessels prevents small vessel vasoconstriction and maldistribution of blood flow. However, marked decreases in PCV due to trauma, hemorrhage, or hemodilution negate these benefits. Rapid decreases in PCV to values <15% of normal reduce blood viscosity and oxygen-carrying capacity so dramatically that vessel wall shear stress, the principal factor responsible for producing and releasing nitric oxide release in small vessels, decreases,\(^{26}\) compromising blood flow and oxygen delivery to vital organs. The ideal viscosities of blood (= 5 to 7 cP [centipoise]) and plasma (= 2.0 to 2.2 cP) for maintaining optimal blood flow and oxygen delivery to tissue in healthy or moderately ill animals are achieved at PCV values between 30% and 35%.\(^{27}\) (cP is the unit of dynamic viscosity in units of g·cm\(^{-1}\)·s\(^{-1}\)).

Capillary (microcirculatory) perfusion can be noninvasively assessed by determining the number of capillaries that have flowing red blood cells (functional capillary density [FCD]). FCD is a linear function of capillary pressure and depends on viscosity, small vessel tone, and arterial blood pressure.\(^{25}\)

**QuickNotes**

The question that continues to confront and perplex clinicians is not whether fluids should be administered but which fluid, how much, and how fast.

**Treatment Recommendations**

The question that continues to confront and perplex clinicians is not whether fluids should be administered but which fluid, how much, and how fast; therefore, fluid resuscitation recommendations vary depending on each patient. Not even blood is an ideal resuscitative fluid for the treatment of trauma, hypovolemia, or hypotension in every animal. Issues associated with availability, collection, storage, appropriate donors, crossmatching, oxygen-carrying capability, the potential for transfusion reactions, effects on inflammation and immune function, and the volume to be administered confound the use of blood and underscore the need for careful selection of new resuscitative fluid alternatives.

**Box 2** presents a revised approach to short-term fluid administration that takes the above principles into consideration and presents an alternative to recipe-based protocols that emphasize excessive fluid loading with crystalloid fluids.\(^{28–30}\) This approach incorporates diagnostic criteria and many of the principles of “fast-track surgery,” a multimodal approach directed toward enhancing recovery and reducing morbidity by emphasizing the importance of anesthesia, analgesia, reduction of surgical stress, fluid management, acid–base balance, nutrition, ambulation, and an organized team.\(^{31}\)

Resuscitative and perioperative fluids should be balanced (SIDa = 24 mEq/L), have a COP between 20 and 30 mm Hg, maintain or increase plasma viscosity, and, when appropriate, carry oxygen.\(^{19,24,26,32}\) Hypotonic fluids cause water to accumulate in the interstitial fluid compartment and predispose patients to endothelial edema. Conventional wisdom suggests that resuscitative fluids should contain an electrolyte concentration similar to that of plasma and be isotonic (290 to 310 mOsm/L) and iso-osmotic (18 to 23 mm Hg) with plasma, particularly when administered to increase intravascular volume. Although it has been proposed that 3 mL of isotonic crystalloid is sufficient to replace 1 mL of lost blood (3:1), the ratio used in practice is highly variable and can be as high as 8:1, which often results in fluid overload.\(^{35}\) Administration of large volumes of crystalloids under pressure can increase blood loss in a hemorrhaging patient and worsen acidosis, coagulopathy, and the systemic inflammatory response.\(^{5}\) Furthermore, the administration of crystalloids to produce “normal” or “supernormal” hemodynamic variables (e.g., mean arterial pressure,
**Fluid Choice for Resuscitation**

**Revised Fluid Resuscitation Treatment Protocol**

### Initial Treatment
- Maintain vascular volume and tissue perfusion during anesthesia
  - Crystalloids for maintaining hydration: 1–2 mL/kg/hr
  - Crystalloids mixed with colloids to maintain vascular volume and systolic arterial blood pressure between 100 and 120 mm Hg
    - Lactated Ringer’s solution mixed with Hextend (50:50 ratio): 3–10 mL/kg/hr for up to 4 hr
- Restore blood volume and tissue perfusion (i.e., fluid therapy)
  - Colloids to maintain systolic arterial blood pressure between 100 and 120 mm Hg
    - Hextend: 5–20 mL/kg in dogs, 5–10 mL/kg in cats; 5–10 mL/kg/day for both species
    - Voluven: 10–20 mL/kg in dogs and cats; 50–60 mL/kg/day
    - 23% saline mixed with Hextend (1:2 ratio): 3–5 mL/kg to effect
    - Administer Hextend or Voluven 1:1 or 1:2 for each mL of blood loss until maximum dose or PCV <20%
    - Fresh blood: 5–20 mL/kg to effect
- Maintain hemoglobin concentration >7 g/dL (PCV >20%)
  - mL donor blood = Recipient blood volume × Desired PCV − Actual PCV/PCV of donor blood
- Control hemorrhage
  - Compression
  - Hemostasis
  - Surgery
  - Hemostatic agents (desmopres-sin 1 μg/kg SC with serial buccal mucosal bleeding time evaluation)

### Alternative/Optional Treatments/Therapy
- Catecholamines (only after or with fluid replacement)
  - Dopamine: 1–10 μg/kg/min in dogs; 3–10 μg/kg/min in cats; to effect or MAP >60 mm Hg if fluid resuscitation does not restore arterial blood pressure
  - Dobutamine: 1–10 μg/kg/min in dogs; 1–5 μg/kg/min in cats; to effect or MAP >60 mm Hg
  - Norepinephrine (0.01–1 μg/kg/min) to a MAP >60 mm Hg and vasopressin (0.0005–0.001 μg/kg/min)
- Treat clinically relevant cardiac arrhythmias (rapid or multifocal tachycardia)
  - Lidocaine: 50 μg/kg/min with potassium 0.5 mEq/kg/hr IV, when appropriate
  - Maintain body temperature >98.2°C (>36.8°C)
- If refractory to above (e.g., sepsis)
  - Norepinephrine (0.01–1 μg/kg/min) to a MAP >60 mm Hg and vasopressin (0.0005–0.001 μg/kg/min)
- Treat clinically relevant cardiac arrhythmias (rapid or multifocal tachycardia)
  - Lidocaine: 50 μg/kg/min with potassium 0.5 mEq/kg/hr IV, when appropriate
  - Maintain body temperature >98.2°C (>36.8°C)

### Supportive Treatment
- Improve tissue oxygenation if hypoxic
  - Oxygen cage (FiO₂ >40% of air)
  - Oxygen insufflation via nasal prongs or cannula or face mask (>50 mL/kg/min)
- Administer pain therapy
  - Hydromorphone: 0.05–0.15 mg/kg IV, IM q4–6h
  - Fentanyl: 3–5 μg/kg IV loading dose followed by 3–10 μg/kg/hr
  - Lidocaine: 50 μg/kg/min
  - Ketamine: 1–30 μg/kg/min
- Correct acid–base and electrolyte disorders
  - Sodium bicarbonate is generally of minimal or no value in animals with lactic acidosis
  - If pH <7.1 (HCO₃⁻ <10 mEq/L), administer sodium bicarbonate 0.5–1 mEq/L as needed
- Treat clinically relevant cardiac arrhythmias (rapid or multifocal tachycardia)
  - Lidocaine: 50 μg/kg/min with potassium 0.5 mEq/kg/hr IV, when appropriate
  - Maintain body temperature >98.2°C (>36.8°C)

### Patient Monitoring
- Heart rate and rhythm: 80–120 bpm
- Systolic arterial blood pressure: >90 mm Hg
- Mucous membrane color: pink
- Capillary refill time: <2 sec
- Level of consciousness and attitude: conscious and alert
- Pupillary diameter: normal
- Urine production: >0.5 mL/kg/hr initially
- Body temperature: >98.2°C (>36.8°C)
- Arterial/venous acid–base values
  - pH: 7.35–7.45
  - Blood gases: PaO₂ >80 mm Hg; PaCO₂ 30–60 mm Hg
  - HCO₃⁻: 18–23 mEq/L; BE: ±2 mEq/L
- Mixed venous oxygen saturation: >75%
- Sublingual PaCO₂: 30–60 mm Hg
- Transcutaneous PaCO₂: <80 mm Hg
- Electrolytes: sodium, potassium, chloride, calcium
- Lactate: <2 mM/L

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**References**

3. Urine output also depends on the rate of fluid administration in conjunction with assessment of ongoing fluid losses.
4. BE = base excess, HCO₃⁻ = bicarbonate, PaO₂ = fraction of inspired oxygen, MAP = mean arterial pressure.
Fluid Choice for Resuscitation

WEB EXCLUSIVE

central venous pressure) does not guarantee improved tissue perfusion or oxygenation. Therefore, it is important to administer crystalloid fluids judiciously and monitor the patient frequently to avoid complications.

Fluids that minimize or decrease the SIG provide a greater survival advantage by minimizing or eliminating the deleterious effects of acidemia. The SIDa is forced toward the SIDa of the infused fluid. For example, some hetastarch solutions (e.g., Hextend) increase the SIDa, thereby lowering the SIG and helping prevent acidosis after hemorrhagic shock. Fluids that decrease the SIDe and increase the SIG, such as normal saline (SIDa = 0), pre-dispose animals to hyperchloremic (nonrespiratory) acidosis. Furthermore, popular balanced and buffered crystalloid solutions (LRS, Normosol-R) require an intermediary metabolic step to achieve their effective SIDa, which is still less than that of plasma.

**TABLE 1**

<table>
<thead>
<tr>
<th>Milestones/Recovery Time Frames</th>
<th>Prognosis</th>
<th>Unfavorable criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved attitude and awareness</td>
<td>Favorable criteria</td>
<td></td>
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<tr>
<td>Strong, regular peripheral pulse</td>
<td>—Improved consciousness and awareness</td>
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<tr>
<td>Normal heart rate and rhythm</td>
<td>—Strong regular pulse (normal arterial blood pressure)</td>
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<tr>
<td>Pink mucous membranes and normal capillary refill time</td>
<td>—Normal heart rate and rhythm</td>
<td></td>
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<tr>
<td>Normal body temperature</td>
<td>—Normal breathing, mucous membrane color, and capillary refill time</td>
<td></td>
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<tr>
<td>Normal eating, drinking, and urine production</td>
<td>—Normal body temperature</td>
<td></td>
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<tr>
<td>Normal acid–base status</td>
<td>—Normal urine production</td>
<td></td>
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<tr>
<td></td>
<td>—Normal acid–base (lactate) and electrolyte values</td>
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<tr>
<td></td>
<td></td>
<td>—Unresponsiveness to fluid resuscitation or vasopressors</td>
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<td></td>
<td></td>
<td>—Loss of consciousness</td>
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<td></td>
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<td>—Poor mucous membrane color and prolonged capillary refill time</td>
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<td></td>
<td></td>
<td>—Hypotension</td>
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<td></td>
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<td>—Tachypnea, labored breathing or apnea</td>
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<td></td>
<td></td>
<td>—Hypothermia (&lt;96°F [35.6°C])</td>
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<td>—Unresponsive and ongoing acidemia</td>
</tr>
</tbody>
</table>

**QuickNotes**

It is important to administer crystalloid fluids judiciously and monitor the patient frequently to avoid complications.

Administering large amounts of crystalloid fluids during profound hemorrhage may cause hemodilution and reduces blood and plasma viscosity. Too low a blood or plasma viscosity can be detrimental to tissue perfusion and oxygenation. Administering fluids that help to maintain or increase plasma viscosity (colloids) helps to decrease small vessel resistance and improve microcirculatory blood flow, thereby improving microvascular perfusion and long-term survival. Systemic vascular resistance is also increased, which may help to maintain arterial blood pressure, thereby further supporting microcirculatory perfusion.

The oxygen-carrying capability of the resuscitative fluid may be critical in animals that have suffered significant blood loss. Blood is an ideal oxygen-carrying colloid. The arterial oxygen content of blood primarily depends on the hemoglobin concentration ([Hb]), which can be indirectly assessed by the PCV ([Hb] = \( \frac{1}{3} \) PCV). Oxyglobin, a hemoglobin-based oxygen-carrying solution in a balanced electrolyte solution (300 mOsm/kg, pH = 7.8), has a COP of 42 mm Hg, a viscosity of approximately 2.4 cP, and a P\(_{50}\) (P\(_o2\) at which hemoglobin is half saturated with oxygen) of 37 mm Hg (blood P\(_{50}\) = 27 mm Hg), qualifying it as an oxygen-carrying plasma expander. Oxyglobin contains small molecules that facilitate perfusion and oxygenation of smaller vessels that might otherwise restrict the passage of RBCs.\(^b\)

\(^b\)At the time of publication, according to the manufacturer, the supply of Oxyglobin was limited to remaining stock. This product may no longer be available in the United States.
Fluids with colloidal value are the best choice for supporting or maintaining vascular volume but are not without problems (e.g., hypervolemia, hemodilution, coagulopathy, renal failure). Commercially available colloids (dextran, hetastarch) are polydisperse and produce COPs that help to maintain intravascular volume and promote transcapillary refill (Table 2). The relationship between COP and hydrostatic pressure becomes critical in animals suffering from shock or hypo-oncotic states and in hypotensive surgical patients that have lost significant quantities of blood (>15 to 20 mL/kg).

First-generation hetastarch-based colloids in saline or LRS (e.g., Hextend) have a relatively long duration of effect (6 to 12 hr) and are considered to be safe and effective if administered doses do not exceed 20 to 40 mL/kg/day and if blood clotting mechanisms and renal function are not impaired. Large doses (>30 mL/kg/day) of commercially available colloids may negatively influence coagulation and renal function. More recent hetastarch-based colloids (Voluven) produce fewer adverse effects on coagulation and renal function but have a shorter duration of effect (4 to 6 hr), even when administered in relatively large doses (50 to 60 mL/kg/day).

All colloids should be administered at lower doses than crystalloids because they are retained in the intravascular space for longer periods of time than crystalloids. “Hypotensive” fluid resuscitation strategies should be considered in animals with uncontrolled hemorrhage only if adequate monitoring capabilities are available. Low-volume fluid resuscitation strategies that include colloids, small amounts (3 mL/kg) of hypertonic saline (7.0% to 7.5% sodium chloride), or a combination of both can be administered to restore mean arterial blood pressure to 60 mm Hg until hemorrhage is controlled. The administration of small volumes of 7.5% hypertonic saline rapidly improves hemodynamic variables, increases the effective circulating volume (hyperosmolar effect), and helps restore tissue perfusion by dilating precapillary resistance vessels. In addition, hypertonic saline has been shown to decrease mortality by producing antiinflammatory effects, limiting and correcting endothelial and gut edema, restoring gastrointestinal barrier function and motility, and limiting and controlling increases in intracranial pressure following traumatic brain injury. Combining hypertonic saline with Hextend or blood is known to be more beneficial than administration of either product alone and helps offset potential undesirable effects.

For a review of these strategies, see “Limited Fluid Volume Resuscitation” (July 2009), also available on CompendiumVet.com.

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**References**

17. Adrogue HJ, Madias NE. Management of life-threatening acid-
Fluid Choice for Resuscitation


