Fluid therapy is a cornerstone of treatment for numerous clinical conditions in veterinary and human medicine. We use fluids so commonly that it is easy to forget that they are drugs and should be considered with the same care and tailored to an individual patient’s needs the way we would any medication. Here we review the physiology of fluid therapy, composition of common crystalloid fluids used in veterinary medicine, and review fluids choice and additives for select electrolyte disturbances.

**Physiology**

The body is approximately 60% water, with some variation by age, size, body condition, and species. Of that 60% total body water (TBW), fluid is further subdivided into compartments within the body as outlined in Figure 1:

![Figure 1](image)

**Figure 1:** Approximately 2/3 of total body water (TBW) is intracellular (ICF) and 1/3 extracellular (ECF), with the ECF being further subdivided into ¾ interstitial space (IS) and ¼ intravascular space (IV). For example, a 10 kg dog has approximately 6L of TBW, 4L of which is ICF, 2L is ECF. Of that 2L in the ECF, 1.5L is in the IS and 0.5L in the IV space.

Note that this description of the IV space is referring only to fluid volume; for a 10kg dog, the expected blood volume is 90 ml/kg (900 mL), of which 500mL is the liquid plasma phase described in this diagram and the remaining volume is normal red cell mass (whose volume would be part of the ICF in this diagram as it is contained within the red cells).

Understanding the forces that dictate movement of fluid between these compartments is crucial as these forces determine how conditions affecting total body water are detected on PE and bloodwork, and also where our fluid therapy ends up once administered.

The movement of water between the ICF and ECF is controlled by the relative osmolality of the two compartments. Osmolality refers to the number of particles, here generally the electrolytes potassium
in the cell and sodium outside of the cell, dissolved in the fluid. Therefore, if sodium concentration in
the ECF increases, fluid will move out of the cell until the concentration of particles in the ICF is
concentrated enough that it now equals that in the ECF (and vice versa.)

The movement of water within the ECF between the vascular and interstitial space is dictated by
starling’s forces. The classic starling’s forces describe that increasing hydrostatic pressure within the
vessel will force water out, while increasing oncotic pressure (in health, generally dictated by albumin
concentration) will help hold fluid in the vascular space. A more modern understanding of starling’s
forces also includes the glycocalyx, a layer on the surface of the endothelium that can significantly affect
vascular permeability and water flux. Finally, fluid leaves the interstitial space primarily through
lymphatic drainage. Therefore, conditions that increase hydrostatic pressure, decrease oncotic pressure,
increase vascular permeability, and/or decrease lymphatic drainage will all contribute to excessive fluid
in the interstitial space, aka edema.

We generally infuse fluids into the intravascular space, and they redistribute to the other body
compartments from there, depending on their composition. Fluid composition is often characterized
relative to normal plasma osmolality; that is, hypertonic fluids contain more particles (sodium) than
normal plasma, hypotonic fluids contain less, and isotonic fluids contain an amount of particles
approximately equal to normal plasma osmolality (about 300 mOsm). This is important to understand
because if the patient’s blood does NOT have normal osmolality, the “isotonic” fluid you are infusing
could actually cause significant fluid shifts. The impact of infusing different fluid types on osmolality and
body compartment volume in a patient with starting with normal blood osmolality is outlined in Figure
2.
Figure 2. Normal (initial) body water distribution is represented by the solid box; change in fluid volumes and osmolality after fluid infusion is represented by the dotted-line shaded box.

Top: Infusion of isotonic fluid. There is no change in ICF, but ECF volume is expanded. The IS and IV space expand in proportion to their distribution ratio (3/4 into IS, ¼ into IV space). Osmolality is unchanged.

Middle: Infusion of hypertonic fluid. Increased sodium concentration infused into the IV space will initially greatly increase IV volume as it pulls fluid from the IS space. Increased IS osmolality will subsequently draw fluid from the ICF compartment. In approximately 30 min, the sodium redistributes across the entire ECF in proportion to the ECF distribution ratio (3/4 IS, ¼ IV). The end result is increased overall osmolality, mildly increased ECF volume, and decreased ICF volume.

Bottom: Infusion of hypotonic fluid. The fluid will redistribute across all compartments according to the distribution ratios of TBW (2/3 ICF, 1/3 ECF, etc.). This will result in a decrease of osmolality of all compartments and an increase in volume of all compartments, with the majority of volume ending up in the ICF.
<table>
<thead>
<tr>
<th>Fluid</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>Mg</th>
<th>Buffer</th>
<th>Osmolality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9%NaCl</td>
<td>154</td>
<td>154</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>n/a</td>
<td>308</td>
</tr>
<tr>
<td>LRS</td>
<td>130</td>
<td>109</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>Lactate</td>
<td>275</td>
</tr>
<tr>
<td>Norm-R</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>Acetate, gluconate</td>
<td>295</td>
</tr>
<tr>
<td>Plasmalyte</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>Acetate, gluconate</td>
<td>295</td>
</tr>
<tr>
<td>Plasma</td>
<td>145</td>
<td>105</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>Bicarb, proteins, etc.</td>
<td>300</td>
</tr>
</tbody>
</table>

Table 1: Composition of common isotonic crystalloid fluids relative to plasma. All electrolytes/buffers in mEq/L, osmolality in mOsm/L

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>Mg</th>
<th>Buffer</th>
<th>Osmolality</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.5%NaCl</td>
<td>1283</td>
<td>1283</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>n/a</td>
<td>2566</td>
</tr>
<tr>
<td>3% NaCl</td>
<td>513</td>
<td>513</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>n/a</td>
<td>1026</td>
</tr>
<tr>
<td>8.4%NaHCO3</td>
<td>1000</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>bicarb</td>
<td>2000</td>
</tr>
<tr>
<td>Plasma</td>
<td>145</td>
<td>105</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>Bicarb, proteins, etc.</td>
<td>300</td>
</tr>
</tbody>
</table>

Table 2: Composition of common hypertonic crystalloid fluids relative to plasma. All electrolytes/buffers in mEq/L, osmolality in mOsm/L

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>Mg</th>
<th>Buffer</th>
<th>Osmolality (in BAG)</th>
<th>Osmolality (in PATIENT)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D5W (5% dextrose in water)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>50g/dL dextrose</td>
<td>278</td>
<td>0</td>
</tr>
<tr>
<td>0.45% NaCl</td>
<td>77</td>
<td>77</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>154</td>
<td>154</td>
</tr>
<tr>
<td>0.45% NaCl + 2.5% dextrose</td>
<td>77</td>
<td>77</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>25g/dL dextrose</td>
<td>293</td>
<td>154</td>
</tr>
<tr>
<td>Plyte 56</td>
<td>40</td>
<td>40</td>
<td>13</td>
<td>0</td>
<td>3</td>
<td>acetate</td>
<td>110</td>
<td>110</td>
</tr>
<tr>
<td>Plasma</td>
<td>145</td>
<td>105</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>Bicarb, proteins, etc.</td>
<td>300</td>
<td>300</td>
</tr>
</tbody>
</table>
Table 3: Composition of common hypotonic crystalloid fluids relative to plasma. All electrolytes/buffers in mEq/L, osmolality in mOsm/L. Final column assumes normal metabolism of dextrose (i.e. non-diabetic).

**Indications and doses for crystalloid fluids:**

**Isotonic fluids**

Isotonic fluids are very good at filling the interstitial space and moderately good at filling the intravascular space. As such, they are commonly used in the resuscitation of intravascular volume (shock boluses) and replacement of dehydration deficits. The compositions of commonly used isotonic fluids are outlined in Table 1.

Shock doses: Dog = 90 ml/kg. Cat = 60 ml/kg.

Fluids for IV resuscitation are commonly administered in quarter shock doses titrated to effect. Some animals with significant hypovolemia or ongoing losses may require volumes in excess of these doses, and others with underlying cardiovascular or renal disease may not be able to handle high volume bolus therapy. Therefore fluid resuscitation from shock must be titrated in each individual based on response of the shock state to fluid therapy.

Correction of dehydration is generally performed by adding the estimated dehydration deficit on to the patient’s daily maintenance needs; if ongoing losses are significant, replacement of these will also need to be factored into a fluid therapy plan. Dehydration deficit estimate is based on PE findings, with <5% being undetectable and >10-12% resulting in hypovolemic shock. Animals between 5-10% will have signs of dehydration on PE (dry MM, prolonged skin tent/sticky interstitium, sunken eyes, thirst) and possibly blood work (elevated PCV/TP, prerenal azotemia, concentrated urine with decreased urine output) without signs of hypovolemic shock (pallor, prolonged CRT, tachycardia, poor pulses, cold extremities, decreased mentation).

Many ways to calculate maintenance fluid needs have been described, including the following:

1) 30(BW kgs) +70ml/day
2) 60(BW kgs) +140ml/day
3) 40-60ml/kg/day
4) 2-4ml/kg/hr
5) Dogs: 132(BW kgs^0.75), cats: 70(BW kgs^0.75)

These calculations are based on healthy dogs in experimental settings with variable activity levels; the author usually uses either equation 3 or 5 depending on patient size. Very small animals need more fluid on a surface area basis than very large ones, so linear equations such as #3 tend to be less accurate at extremes of weight. Once a fluid therapy plan is generated, it is important to keep in mind that it is an ESTIMATE, and the patient likely needs more or less fluids than you have guessed. Reassessment of the hydration status of the patient throughout therapy is critical to prevent volume under- or overloading. Monitoring of fluid therapy is discussed below.

**Hypertonic fluids**
Hypertonic saline (HTS) should not be administered at a bolus concentration above 7.5% in a peripheral vessel due to the significant risk of phlebitis from hyperosmolality. HTS is a good “stopgap” measure in shocky patients; each 1mL given provides a quick bump of approximately 4mL in IV volume for about 20 min, after which time the sodium redistributes across the entire ECF compartment. This also raises ECF osmolality and draws fluid from the ICF compartment. This can be particularly helpful in the early resuscitation of traumatic brain injury patents as it both helps to (transiently) improve blood pressure and decrease brain swelling. For hypotensive patients, HTS should be chased with additional fluids as needed as it will not expand the vascular space for very long. Common bolus doses are 3-7mL/kg, with the lower end of the dose recommended for cats.

Hypertonic saline is rarely needed for the correction of hyponatremia as most causes are due to retention of water rather than lack of salt (see Sodium section below).

The compositions of commonly used hypertonic fluids are outlined in Table 2.

Hypotonic fluids

For every 1000mL of water infused into the IV space, only about 83 mL (1/12) will remain in the IV space; the majority (2/3) will end up in the IC space. Therefore, hypotonic fluids are never used for bolus IV volume resuscitation, only to replace free water losses, which are generally diagnosed by finding hypernatremia on blood work. Doses will vary depending on deficit and ongoing losses (see Sodium section below). The compositions of commonly used hypotonic fluids are outlined in Table 3.

Monitoring fluid therapy

Every initial fluid plan is a guess, albeit an educated one. We “test” this hypothesis by administering fluids to the patient and gauging their response. Therefore, the more aggressive we are in administering volumes and rates of IV fluids, the more aggressive we must be with monitoring that response so as to not overshoot. Common parameters to monitor to assess adequacy of fluid therapy include repeat physical exam assessment, repeat bloodwork to look for normalization of previous values indicating hemoconcentration, weight gain by the patient, resolution of thirst, increased production of progressively dilute urine, and calculation of “ins and outs” if ongoing losses are being quantified in some manner (urinary catheter, drain production, etc.). Very small patients may also receive a significant amount of volume via flushes and IV medications, so these volumes should be recorded when considering “ins.” Clinical signs of fluid overload often present as development of interstitial edema. An early place to look for edema is the conjunctiva; other common sites include the distal limbs, under the jaw, and gravity-dependent areas of the body. Respiratory distress/cough due to fluid overload and pulmonary edema is possible, but is usually a late finding unless the patient has significant cardiac disease. Monitoring trends in resting respiratory rate is safer and more sensitive for early detection of pulmonary edema than waiting for overt crackles on auscultation. Cavitary effusions with transudates or modified transudates may also be seen, and serous to serosanguinous discharge from wounds, incisions, or the respiratory system may be noted. Mild to moderate elevations in blood pressure and heart rate can be seen with fluid overload, but are affected by many variables and are therefore not reliable indicators to rule it in or out.

Using fluids/additives for correction of select electrolyte abnormalities

Sodium

It is important to remember that the vast majority of “sodium” abnormalities seen on bloodwork are actually free water abnormalities. When the absolute amount of sodium in the patient changes, then generally water should follow and maintain the same sodium concentration (i.e. “where salt goes, water...
follows). For example, if the kidney retains salt and a corresponding amount of water with it, this will increase overall fluid volume in the body, but the fluid reabsorbed is isotonic so the overall sodium concentration in the blood will not change. However, if we hold the absolute amount of sodium in the body static but change the water content in which it is dissolved, then we see the sodium value as measured on bloodwork go up or down. Therefore, when you see a dysnatremia on bloodwork, generally think water, not salt.

Hypernatremia can result from lack of intake of free water or excess intake of salt (by the patient or iatrogenic administration of hypertonic fluids), but more commonly results from loss of hypotonic fluid from the GI tract or kidneys. The free water deficit is commonly calculated using the following equation:

\[
\text{Water deficit} = 0.6 \times \text{Body weight (kg)} \times [(\text{Plasma Na}/\text{Normal Na}) - 1]
\]

This calculated deficit is replaced over the same amount of time it took to acquire the sodium abnormality. For example, if the patient had bloodwork at 8am with a Na of 140 and at 8pm the Na is 160, it is safe to drop the Na back to 140 within a 12 hour timespan. More commonly, the timespan is not known and the change must be assumed to be chronic; at that point, the goal should be to drop the Na by no more than 0.5mEq/hr to avoid cerebral edema.

Another helpful tool is the Adrogué-Madias equation, which calculates the expected change in Na given an infusion of 1L of a fluid with a known sodium concentration:

\[
\text{Expected change in serum Na} = (\text{Na + K in 1L of fluid} - \text{patient Na}) / (0.6 \times \text{body weight [kg]} + 1)
\]

For example, let’s say you had a 10kg patient with a sodium of 175, and you wanted to give it 120mL/kg/day of Plasmalyte (Na=140, K=5):

\[
[(140+5) - 175] / (0.6 \times 10 +1) = -30/7 = -4.2. \text{ So administration of 1L of plyte to this patient would drop the Na by 4.2. At 120mL/kg/d, this patient will be receiving 50 mL/hr, so it should take him 20 hours to decrease by 4.2. 4.2/20 = 0.21, well below the 0.5 mEq/hr margin of safety.}
\]

That being said, you can do all the math in the world and the patient’s sodium often will do what it wants. Therefore, frequent monitoring (at least q4-6h initially) of the patient’s response to any change in fluid type or rate of infusion is key in correction of sodium disorders.

As previously stated, hyponatremia implies an excess of free water in the body and unless iatrogenic or spurious, almost always is secondary to a low effective circulating volume or a disorder of ADH. Both will result in renal retention of water; with low effective circulating volume, although the body is attempting to retain both water and salt, for various reasons, water is “winning” as far as amount retained. Occasionally excessive intake (water toxicity) is also a cause. Treatment is usually aimed at resolution of the underlying disease process, but if hyponatremia is severe enough to cause neurological signs, judicious use of higher sodium fluids can be considered depending on the patient’s volume status. The Adrogué-Madias equation can similarly be used to calculate corrective fluid types and volumes for hyponatremia.

**Chloride**

Similar to Na, Cl also changes with free water status and so should change concurrently with sodium concentration. When Cl is changing independently of sodium, this indicates acid/base disturbance in the body, with an elevated Cl associated with acidemia and a low Cl with alkalemia. Hypochloremia is most commonly associated with loss of upper GI fluid in dogs. Hyperchloremia can be associated with many acid/base abnormalities, usually secondary to renal retention of Cl in lieu of bicarbonate. An important
and commonly overlooked cause of hyperchloremia is iatrogenic; fluids such as NaCl contain a very high concentration of Cl relative to Na and are therefore acidifying. Colloids are commonly administered in 0.9% NaCl, and addition of high levels of KCl supplementation to fluids also increases Cl content. Hyperchloremia is becoming a “hot topic” in human medicine and may be associated with worsening renal function, hemodynamic status, lung function, and may worsen inflammation. Therefore, clinicians should be cognizant about reducing iatrogenic Cl supplementation in hyperchloremic patients.

**Potassium**

Potassium is commonly supplemented in IV fluids to correct hypokalemia and prevent “washout” of normal potassium levels with IVF. The problem is that many clinicians choose K supplementation by an absolute amount per bag, i.e. 40 mEq/L. This does not take into account the RATE at which the fluids are administered and hence the actual dose given is not being calculated. A safer and more effective way to dose K is in mEq/kg/hr, using guidelines like the one below:

<table>
<thead>
<tr>
<th>Serum K concentration (mmol/L)</th>
<th>Rate of K supplementation (mEq/kg/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2</td>
<td>0.5</td>
</tr>
<tr>
<td>2-2.4</td>
<td>0.4</td>
</tr>
<tr>
<td>2.5-2.9</td>
<td>0.3</td>
</tr>
<tr>
<td>3.0-3.4</td>
<td>0.2</td>
</tr>
<tr>
<td>3.5-5</td>
<td>0.1</td>
</tr>
<tr>
<td>&gt; 5</td>
<td>None, recheck q4-8h as therapy is initiated</td>
</tr>
</tbody>
</table>

Generally, doses in excess of 0.5 mEq/kg/hr or “Kmax” are discouraged due to cardiovascular risk with high-dose KCl supplementation. Severe cases may require higher levels of supplementation, but continuous ECG monitoring should be used and nursing staff should be exceedingly careful not to flush lines containing high concentrations of K. High concentrations of KCl are caustic to peripheral veins, so infusions containing >80mEq/L should be administered via central line to avoid phlebitis over time, and K should not exceed 200mEq/L by any route. Clinicians should be cognizant that they are also infusing Cl (see above) and consider use of oral K supplementation or KPhos in patients who also require phosphorus supplementation (e.g. DKA).

For all fluid additives (KCl, but also dextrose, pain medications metoclopramide, etc.), it is imperative that not only the concentration of the additive in the bag be considered, but also the rate of administration, as this will change the total dose delivered. If a patient will require frequent changes in fluid rate, consider running 2 fluid lines: the first, a bag of fluids run at the basic maintenance rate containing the additives desired, the second a bag with no additives that can be adjusted to compensate for dehydration or ongoing losses. Alternatively, syringe pumps can be used to “piggyback” additives onto fluid lines, or in-line burettes if frequent additive changes may be needed to reduce changing between multiple bags.